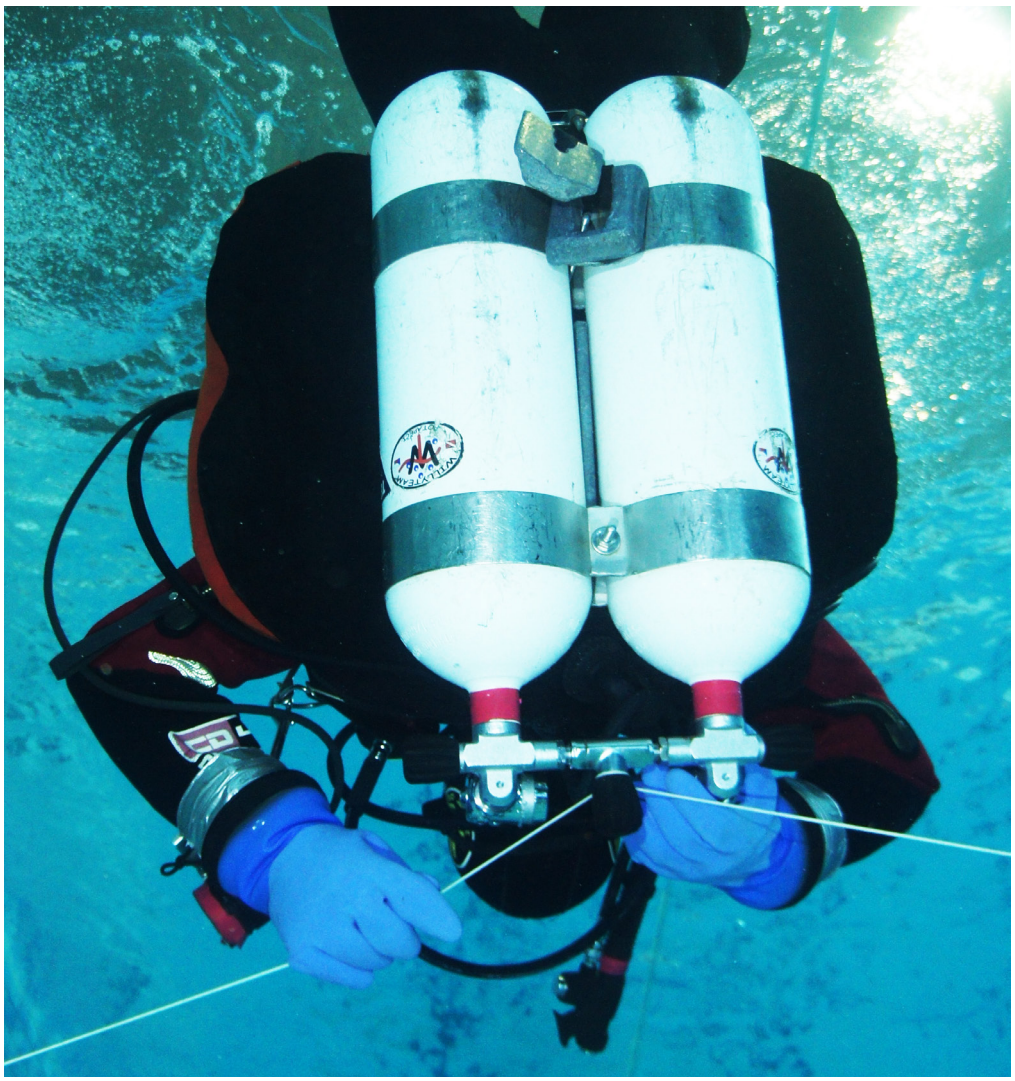
Diving and Hyperbaric Medicine

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

SPUMS *Volume 56 No. 3 September 2026* EUBS



Scenario reconstruction in accident analysis

Twelve original articles

Two technical reports

Three reviews

One short communication

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

Editor

Simon Mitchell editor@dhmjournal.com

European (Deputy) Editor

Lesley Blogg euroeditor@dhmjournal.com

Editorial Manager

Nicky Telles editorialassist@dhmjournal.com

Submissions: https://www.manuscriptmanager.net/dhm

Editorial Board

David Cooper, Australia
David Doolette, USA
Christopher Edge, United Kingdom
Ingrid Eftedal, Norway
Peter Germonpré, Belgium
Jacek Kot, Poland
Claus-Martin Muth, Germany
Neal Pollock, Canada
Monica Rocco, Italy
Chris Sames, New Zealand
Martin Sayer, United Kingdom
Erika Schagatay, Sweden
Robert van Hulst, The Netherlands

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

SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY

OFFICE HOLDERS

President
Stephan Roehr president@spums.org.au
Past President
Neil Banham pastpresident@spums.org.au
Secretary
Douglas Falconer secretary@spums.org.au
Assistant Secretary
Ian Gawthrope secretary@spums.org.au
Treasurer
Louisa Kippin treasurer@spums.org.au
Education Officer
Xavier Vrijdag education@spums.org.au
Chairman ANZHMG
Robert Webb anzhmg@spums.org.au
Committee Members
Bridget Devaney bridget.devaney@spums.org.au
Elizabeth Elliott elizabeth.elliott@spums.org.au
Catherine Meehan catherine.meehan@spums.org.au
Soon Teoh soon.teoh@spums.org.au
San Clarke san.clarke@spums.org.au
Webmaster
Joseph Davey webmaster@spums.org.au

ADMINISTRATION and MEMBERSHIP

Membership
Send an email to: admin@spums.org.au
For further information on SPUMS and to register to become a member, go to the Society's website: **www.spums.org.au**
The official address for SPUMS is:
c/o Australian and New Zealand College of Anaesthetists,
630 St Kilda Road, Melbourne,
Victoria 3004, Australia
SPUMS is incorporated in Victoria A0020660B

EUROPEAN UNDERWATER AND BAROMEDICAL SOCIETY

OFFICE HOLDERS

President
Bengusu Mirasoglu bengusu.mirasoglu@eubs.org
Vice President
Anders Kjellberg anders.kjellberg@eubs.org
Immediate Past President
Jean-Eric Blatteau jean-eric.blatteau@eubs.org
Past President
Ole Hyldegaard ole.hyldegaard@eubs.org
Honorary Secretary
Peter Germonpré peter.germonpre@eubs.org
Member-at-Large 2025
Benoît Desgraz benoit.desgraz@eubs.org
Member-at-Large 2024
Pieter Bothma pieter.bothma@eubs.org
Member-at-Large 2023
Michal Hajek michal.hajek@eubs.org
Member-at-Large 2022
Anne Räisänen-Sokolowski anne.raisanen-sokolowski@eubs.org
Liaison Officer
Phil Bryson phil.bryson@eubs.org
Webmaster
Peter Germonpré webmaster@eubs.org

ADMINISTRATION and MEMBERSHIP

Membership Secretary and Treasurer
Kathleen Pye secretary@eubs.org
For further information on EUBS and to complete a membership application, go to the Society's website: **www.eubs.org**
The official address for EUBS is:
c/o Mrs Kathleen Pye, Membership Secretary and Treasurer
35 Sutherland Crescent, Abernethy,
Perth, Perthshire PH2 9GA, United Kingdom
EUBS is a UK Registered Charity No. 264970

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

CONTENTS

Diving and Hyperbaric Medicine Volume 56 No. 3 September 2026

217 The Editor's offering

Original articles

218 Impact of neoprene wetsuits on lung volumes and work of breathing: implications for military diver safety and performance
Antoinette Houtkooper, Thijs T Wingelaar, Pieter-Jan AM van Ooij, Joost G van den Aardweg

226 Occupational health challenges in commercial diving: a qualitative study of psychological risk, training gaps, and organisational responsibilities
Holly Blake, Basharat Hussain, Lianne Hufton, Elisha Cousins, Graham Johnson, Andrew Tabner

237 A calculation method for inert gas tension based on a multi-tissue model
Shuai Di, Chumeng Liu, Jinghua Gu

245 A feasibility study of combined hyperbaric oxygen treatment and a lifestyle medicine program for multimorbid conditions
Elizabeth Elliott, Sam Manger, John Mercer, John Stevens

257 No difference in core temperature or thermal sensation in male and female scuba divers using wetsuits or semi-drysuits in 22.6°C sea water
Fabian N Möller, Kavi Dodge, Tucker Orman, Igor Glavičić, Željko Dujić, Nikola Foretić, Andrew T Lovering

265 Microbiological monitoring of scuba cylinders under routine filling and seasonal diving conditions
Recep Özkan, Osman Turkmen, Elif Okumuş, Sukran Özhan, Hasan Sivrikaya, Ese Başbulut, Kübra Ozgok Kangal

271 The effect of drysuit diving in warm water on body temperature and post immersion orthostatic hypotension
Joshua T Murphey, Hayden W Hess, Morgan L Worley, Glenda Anderson, Jacqueline Schwob, Brian A Monaco, David Hostler

280 Maintained critical flicker fusion frequency after acute hyperbaric hyperoxia at 140 kPa
Jochen D Schipke, Thomas Muth, Anne-Kathrin Brebeck, Dominik von Hertlein, Fabian N Möller

286 Early nitrogen wash-out for inside attendants during hyperbaric oxygen therapy: a novel oxygen distribution regimen
Mårten Silvanius, Oscar Plogmark, Måns Forsberg, Oskar Frånberg

294 The effect of hyperoxic environment on lung function and myopisation in an extended closed-circuit rebreather diving expedition
Sofia A Sokolowski-Wojnicki, Mikolaj J Wojnicki, Anne K Räisänen-Sokolowski

303 Scuba-related injuries presenting to United States emergency departments: paediatric and adult patterns
Andy Tom, Sam Lindberg, Sergio Navarro, Daniel Stephens

309 Prevalence of laryngopharyngeal reflux symptoms in divers
Bastien A Valencia-Sanchez, Natalie Weiss, Evelyn Echevarria Cruz, Raana Zakeri, Frauke Tillmans, Amy Rutt

Technical reports

317 Forensic analysis of an unwitnessed fatal diving accident using in-water investigative reconstruction
Veronika Rybárová, František Novomeský, Radek Prygl, Jiří Sejba

Technical reports continued.

323 Evaluation of external ventricular drain performance under hyperbaric conditions: a bench study
Rahulkumar Ramchandani, Zhiliang C Lin, Bridget Devaney, Rosalind L Jeffree, Theo Tsouras

Review articles

329 Diving-related ear, nose, and throat disorders: a structured review using a phase-of-dive framework
Anna Onderkova

342 Mental health and neurodevelopmental factors relevant to recreational fitness-to-dive assessment: a structured review and clinical considerations
Calina Ouliaris, Bram Querido, Thijs T Wingelaar, Neil D Banham

350 Ultrasound protocols used to detect vascular gas emboli in divers: a systematic review
Benjamin L Turner, Thijs T Wingelaar, Feiko JM de Jong, PieterJan AM van Ooij, Rigo Hoencamp

Short communication

361 Evaluating the FitMáx questionnaire for fitness-to-dive assessments in a military population
Pieter D Dijk, Paul Clarijs, Thijs T Wingelaar

World as it is

365 Adaptations to breath-hold diving: from traditional divers to elite athletes
Jochen D Schipke, Ulrich Limper, Kay Tetzlaff

Case reports

371 Bilateral sudden sensorineural hearing loss following indirect lightning exposure successfully treated with hyperbaric oxygen therapy: a case report
Recep Özkan, Osman Türkmen, Kübra Yapici, Zülküf Kaya

376 A case of severe multi-system decompression sickness requiring critical care, aggressive fluid resuscitation and hyperbaric oxygen therapy
Alice Palmer, Craig Holdstock, Doug Watts, Rosanna J Stokes, Jack Whiteley

381 Decompression sickness or ciguatera intoxication: a case report
Robert A van Hulst

384 In-water recompression for severe decompression illness in competitive freediving: a case report
Jake Johnston, Juan Valdivia, Leigh Baker, Kelly Heape

Erratum

388 Measuring whole-body inert gas uptake and washout during submersion
Oscar Plogmark, Kristian Soltesz, Carl Hjelte, Oskar Frånberg

389 SPUMS notices and news

395 EUBS notices and news

397 Events, courses & meetings

398 Diving and Hyperbaric Medicine: Instructions for authors
(Short version – updated January 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

The September 2026 issue of DHM is the largest ever with 23 citable articles. This reflects a sustained increase in submissions to the journal over 2025 and 2026. Although the journal's content is typically relatively balanced, this issue is very 'diving-centric' with 19 of the 22 articles having their primary focus on diving matters. There are too many diverse topics to comment on them all. Suffice it to say that we are reporting a lot of very strong original work in September 2026.

I have just returned from a very successful EUBS meeting in Geneva and would like to offer the organising team (Rodrigue Pignel, Alice Varichon, Justine Bernard,

Thomas Schaller) and the team my sincere thanks and congratulations for hosting the meeting. The venue was terrific with an enormous screen in the main auditorium adding to the pleasure of watching many well-prepared presentations. There was a very collegial social vibe at this event attended by over 400 delegates and accompanying persons.

I presented an editor's update at the meeting. Highlights for the journal included a record year for submissions in 2025 which will be exceeded in 2026. More submissions are not necessarily better; this would only be true if the fraction of high-quality submissions were proportionately higher. We are currently receiving moderate numbers of case reports which I am taking a conservative line on. A significant proportion of these are cases of successful treatment of accepted indications for HBO which there seems little point in publishing. Moreover, if I were to publish such cases it sets an unwanted precedent. There is some room for editorial discretion, but in general I am rejecting such cases. We continue to receive submissions from many countries with Australia (not surprisingly) being the leading SPUMS oriented contributor and Türkiye (followed closely by the Netherlands) being the leading EUBS contributors.

Another highlight was maintenance of our JCI impact factor above 1 (1.3 for 2025 after 1.1 in 2024). The impact factor is a capricious metric for small journals and can vary substantially after single highly cited papers move in and out of the citation window. It follows that I counsel society members not to over-interpret the importance of impact factors. With that said, it is fair to say that an impact factor of 1.3 is an excellent result for a small, extremely narrowly-focused societal journal. No matter how high the quality of our publications, they are never going to attract high numbers of citations because we are a niche field that comparatively few people participate in. It follows that the scope for increasing the impact factor beyond where it sits now is small.

The increase in submissions and publication numbers brings challenges in relation to workload for our editorial team. Our entire editorial process is conducted 'in house' and workload is directly proportional to submissions and publication numbers. With that said, the most substantial editorial challenge is obtaining timely high-quality peer review. Reviewer fatigue seemed a substantial problem in 2024 and 2025 and continues into 2026. The number of non-responses or refusals is palpably higher than earlier in my editorship. One of the problems is a small number of expert reviewers in this niche field, coupled with our policy of trying hard to match reviewer expertise to manuscript content. In a niche field such as diving and hyperbaric medicine it can be very challenging to find expert reviewers for some of our articles, because it is not uncommon for some groups to be the only researchers worldwide actively working on a particular topic. Similarly, certain reviewers with specific areas of expertise tend to be overloaded with review work. In part this flows from the rapid expansion of a medical publishing 'industry' which, in my humble opinion, exploits the goodwill of academics in requesting them to perform 'free' reviews that the industry then uses in for-profit publication activity. I suspect our colleagues have started to resent receiving so many review requests. Despite all this, average initial review times are approximately confluent with our stated goal of four weeks, but it is acknowledged there can be marked variability. Unfortunately, this is a natural consequence of asking busy professional colleagues to give up their time in (sometimes onerous) review processes for no reward other than professional satisfaction. We remain extremely grateful to our reviewers for their efforts in supporting the journal.

While at Geneva I attended the EUBS excom meeting. As with the SPUMS excom, the EUBS group is uniformly supportive of the journal, and the editorial team is grateful to both excoms and the Journal Governance Committee for their support. In relation to the editorial team, Lesley Blogg remains the European (Deputy Editor) and Nicky Telles continues her indispensable high-quality work as the Editorial Manager. The SPUMS Treasurer has changed this year, and is now Dr Louisa Kippin from Brisbane Australia. She is effectively the de facto journal accountant and deserves massive credit for managing related finances (with similar recognition for Dr Stephan Roehr who was treasurer but assumed SPUMS presidency in May). Finally, we are proud to add Associate Professor David Cooper, intensivist and hyperbaric physician from Hobart, Tasmania, to the Editorial Board. A grateful welcome to the team David.

Professor Simon Mitchell
Editor, Diving and Hyperbaric Medicine

Cover photo: Inversion of a diver with cave guideline entanglement on the scuba manifold isolation valve in an accident reconstruction. See Rybarova et al. In this issue

Original articles

Impact of neoprene wetsuits on lung volumes and work of breathing: implications for military diver safety and performance

Antoinette Houtkooper[1], Thijs T Wingelaar[1,2], Pieter-Jan AM van Ooij[1,3], Joost G van den Aardweg[3]

[1] *Royal Netherlands Navy Diving and Submarine Medical Centre, Den Helder, the Netherlands*
[2] *Amsterdam UMC, location AMC, Department of Anaesthesiology, Amsterdam, the Netherlands*
[3] *Amsterdam UMC, location AMC, Department of Pulmonology, Amsterdam, the Netherlands*

Corresponding author: *Antoinette Houtkooper, Royal Netherlands Navy Diving Medical Center, Rijkszee en Marinehaven, PO Box 10.000, 1780 CA Den Helder, the Netherlands*
a.houtkooper@mindef.nl

Keywords
Airway resistance; Chest wall compression; Diving physiology; Forced oscillation technique; Lung volume

Abstract
(Houtkooper A, Wingelaar TT, van Ooij PJAM, van den Aardweg JG. Impact of neoprene wetsuits on lung volumes and work of breathing: implications for military diver safety and performance. Diving and Hyperbaric Medicine. 2026 30 September;56(3):218–225. doi: 10.28920/dhm56.3.218-225. PMID: 42743560.)
Introduction: Neoprene wetsuits may impose mechanical constraints on the chest wall, potentially altering respiratory function. This study investigated the impact of neoprene wetsuits on lung volumes, airway mechanics, and work of breathing (WOB) in healthy male divers.
Methods: A randomised crossover trial was conducted with 31 male divers at the Royal Netherlands Navy Diving Medical Centre. Participants underwent pulmonary function testing, including spirometry, body plethysmography, the forced oscillation technique (FOT), and diffusion capacity measurements, both with and without a hoodless standardised 5 mm neoprene full body wetsuit with a neoprene neck seal. Primary outcomes included changes in forced vital capacity (FVC), functional residual capacity (FRC), airway resistance (R_{aw}), reactance (X_{rs}), and WOB.
Results: Wearing a neoprene wetsuit led to statistically significant reductions in FVC (2.8%, $P < 0.05$), forced expiration in one second (2.9%, $P < 0.05$), FRC (4.0%, $P < 0.05$), and expiratory reserve volume (10.9%, $P < 0.05$), alongside increases in inspiratory capacity and tidal volume. R_{aw} increased significantly ($P < 0.05$), while the FOT revealed altered airway mechanics, evidenced by increased X_{rs} at multiple frequencies ($P < 0.05$). Diffusion capacity remained unchanged, suggesting preserved alveolar-capillary function.
Conclusions: Neoprene wetsuits induce mechanically restrictive effects on the chest wall, reducing static and dynamic lung volumes and increasing WOB. While these changes may not be clinically relevant at rest, their impact needs to be determined during strenuous or prolonged dives, particularly when combined with other equipment that limits thorax excursions. Future research should explore the effects of the military 5 mm wetsuit under immersed conditions to better understand their operational impact on diver performance and safety.

Introduction

Scuba diving is a physically demanding activity that exposes individuals to hydrostatic pressure, thermal stress, and equipment-related constraints, which requires psychological and physiological adaptation.[1] Neoprene wetsuits provide thermal insulation and buoyancy but also impose mechanical constraints on the chest wall.[2] Previous research has demonstrated that wetsuits can alter respiratory mechanics, reducing dynamic lung volumes (e.g., forced vital capacity [FVC] and forced expiratory volume in one second [FEV_1]).[3,4]

While the observed changes in these studies are likely irrelevant to recreational divers,[5] minor changes in respiratory function can have significant consequences for performance, safety, and operational efficiency among military, technical or commercial divers. Additionally, the broader impact of wetsuits on pulmonary function, including static lung volumes, airway resistance, and diffusion capacity, remains poorly understood.[3,4]

Although a decrease in static lung volumes may seem obvious due to the external compressive forces on the chest wall, it is essential to emphasise that a neoprene wetsuit insulates the whole body, and therefore may also restrict

abdominal breathing manoeuvres.[6] A lower functional residual capacity (FRC) is particularly concerning for divers, as it increases the work of breathing (WOB) and reduces the lung's buffering capacity during ventilation, especially while performing physical tasks, potentially leading to earlier onset of dyspnoea, and greater susceptibility to alveolar collapse during exertion or at depth.[7–9] Buffering refers to the ability of the lung to maintain the acid-base balance in the body, primarily by regulating carbon dioxide levels. This buffering role is critical for maintaining stable alveolar oxygen and carbon dioxide levels and for preventing atelectasis.[10]

The forced oscillation technique (FOT) is a non-invasive method for assessing respiratory mechanics. It offers a unique opportunity to evaluate airway resistance, the opposition of airflow in the respiratory system, and reactance, which refers to the elastic and inertial properties of the respiratory system that oppose changes in airflow, under restrictive conditions.[11] However, its application in diving research is not well-established, leaving a gap in our understanding of how wetsuits influence these aspects of respiratory physiology. This gap is due to a lack of comprehensive data on how chest wall restriction from wetsuits alters airway mechanics and the work of breathing at rest and under simulated diving conditions.

To comprehensively address these challenges, a multimodal approach, incorporating techniques such as body plethysmography, diffusion, and FOT is required. The latter may be able to detect restriction of small airways, reduced lung compliance, and increased work of breathing, as observed in studies of restrictive lung conditions.[11] Such changes could contribute to dyspnoea and respiratory fatigue, particularly during prolonged and strenuous dives.

Drawing on existing literature, we anticipated that wearing a wetsuit would reduce dynamic lung volumes, including FVC and FEV_1.[3,4] Additionally, we expected a reduction in static lung volumes, including FRC and expiratory reserve volume (ERV), due to the restrictive effect of the wetsuit on the chest wall as fulfilled by hydrostatic pressure in naked subjects.[12] We further hypothesised that the wetsuit would increase airway resistance (R_{rs}) and decrease reactance (X_{rs}) due to mechanical compression of the chest wall and altered respiratory mechanics, as described above.[12] We expected that diffusion capacity would remain unaffected.

Methods

A prospective, randomised crossover trial was conducted in a controlled laboratory setting at the Royal Netherlands Navy Diving Medical Centre. This trial was approved by the Medical Research Ethics Committee of the University of Utrecht (ref: NL87993.041.24/24-304/A) and the Surgeon General of the Ministry of Defence (ref: DOSCO 2025002961), and it follows the Declaration of Helsinki 2024.[13]

PARTICIPANTS

Eligible participants included healthy male diving personnel (e.g., navy divers, hyperbaric technicians, and hyperbaric medical staff) who were fit to dive. For simplicity, these participants are referred to as 'divers' throughout this study. Exclusion criteria were an upper or lower respiratory infection (within the last six weeks). Due to limited wetsuit size availability, a minimum height of 172 cm was required. Participants were recruited voluntarily through presentations and posters. Written informed consent was obtained from all participants before the study.

MEASUREMENTS

Baseline characteristics, including age, height, weight, chest circumference, and waist circumference, were recorded for all participants. Chest circumference was measured at nipple level, and the waist circumference was measured at the umbilicus. Body mass index (BMI) and body surface area (BSA)[14] were calculated for each participant. New 5 mm thick super stretch neoprene military wetsuits (Northern Diver, United Kingdom) were used to minimise variation due to degradation or usage. Suit sizes (ranging from M to XXL) were selected based on chest circumference, with adjustments for height if necessary.

Measurements were conducted during a single visit, both with and without the neoprene wetsuit. The order of testing (with or without the suit) was randomised for each participant to minimise learning effects or fatigue. To prevent the influence of spirometry manoeuvres on respiratory mechanics, these were assessed before spirometry.[15]

To address unresolved questions regarding R_{rs} and X_{rs}, FOT parameters were measured using a Resmon Pro V3 (Accuramed, Belgium) in accordance with ERS/ATS guidelines.[16] The enhanced optimised multi-frequency approach (5–11–19 Hz) was employed. R_{rs}, X_{rs}, the resonant frequency (Fres) where X_{rs} crosses zero, the reactance area (AX), and the difference between resistance at low and higher frequencies (R5–R19), a measure of possible airway heterogeneity, were recorded.[17] The device was calibrated prior to the first measurement of each testing day in accordance with the manufacturer's guidelines.

Spirometry, lung volumes, and diffusion capacity for carbon monoxide (DL_{CO}) were measured using the Vyntus Body (Jaeger Medical, the Netherlands), in accordance with the respective guidelines.[18–20] Spirometry performed in a seated position according to the ATS/ERS guidelines is the standard approach, as this posture promotes consistency, patient comfort, and reproducibility of measurements.

While the seated position may have a minor effect on the neck seal, this influence is generally minimal and considered acceptable within the guidelines. Calibration was performed once daily before the first measurements, corresponding with

the manufacturer's guidelines. A single experienced lung function technician performed all measurements to minimise interobserver bias. The DL_{CO} was corrected for haemoglobin (Hb), which was measured at the end of the test run for each participant. The results were interpreted in accordance with the reference values of the Global Lung Initiative.[21,22]

STATISTICAL ANALYSIS

Based on an anticipated 7% reduction in lung function (references 3 and 4) and assuming a normal distribution of the data, a sample size calculation indicated that at least 25 participants were required ($\alpha = 0.05$, $\beta = 0.80$). To account for potential dropouts or failed measurements, 20% more participants were included, resulting in a study population of at least 30 participants.

The normality of the data was evaluated using the Shapiro-Wilk test. Normally distributed data were analysed using paired *t*-tests, while non-normally distributed data were assessed using Wilcoxon signed-rank tests (full details of the statistics are available on reasonable request). The significance threshold (alpha) was set at 0.05. For normally distributed data, results are presented as mean (standard deviation) (SD), whereas non-normally distributed data are reported as median with interquartile range (IQR). Statistical analyses were performed using Stata (version 18.5, StataCorp, USA).

Results

STUDY POPULATION

The study was conducted in accordance with the protocol, and 31 healthy male volunteers were included. All participants were fit to dive, and baseline measurements were within normal ranges based on z-scores. Baseline characteristics are presented in Table 1.

Table 1
Baseline characteristics of the study population

***n* = 31**	**Mean (SD)**	**Range**
Age (years)	40.7 (11.1)	23–59
Height (cm)	184.3 (7.7)	166–198
Weight (kg)	85.8 (10.9)	69–109
Chest circumference (cm)	98.4 (7.1)	85–115
Waist circumference (cm)	89.3 (8.0)	76–107
Body mass index ($kg \cdot m^{-2}$)	25.2 (2.5)	20.1–30.8
Body surface area (m^2)	2.09 (0.16)	1.8–2.4

Changes in spirometry, lung volumes, airway resistance, and diffusion capacity while wearing a wetsuit are presented in Table 2. FVC and FEV_1 decreased statistically significantly by 0.16 L (2.8%) and 0.13 L (2.9%), respectively, when wearing a wetsuit. FRC and ERV also showed significant reductions (see Figure 1), whereas inspiratory capacity (IC) and tidal volume (VT) increased significantly. Alveolar volume (VA_{sb}) decreased significantly, which is consistent with a decreased FRC, but the DL_{CO} remained unchanged. This pattern suggests intact alveolar-capillary function, often seen in extrapulmonary restriction, such as a neoprene wetsuit.

Airway resistance (R_{aw} 0.5 in) measured in the flow range of -0.5 to 0.5 $L \cdot s^{-1}$ during a panting manoeuvre in the body plethysmograph, increased significantly relative to the resistance measured with FOT; see Table 3.

FOT parameters were assessed to evaluate airway resistance and reactance. Changes in these parameters while wearing a wetsuit are presented in Table 3. R_{rs} at 5 Hz, 11 Hz, and 19 Hz were not normally distributed. Wilcoxon Signed-Rank Tests revealed no significant differences. X_{rs} at 5 Hz, 11 Hz, and 19 Hz, along with Fres, AX, and R5-R19, were all normally distributed across participants. These parameters demonstrated significant changes when participants wore the neoprene wetsuit, indicating altered airway mechanics likely due to chest wall restriction, which may reflect increased stiffness of the lung. The reduction in lung volume and the supposed higher work of breathing resembles the mechanism of increased work of breathing during immersion.[12] During swimming, these effects may reinforce each other.

Discussion

This study was the first to investigate the influence of wearing a neoprene wetsuit on respiratory parameters, including dynamic and static lung volumes, impedance and diffusion capacity. This multimodal approach enabled us to assess both the mechanical constraints imposed by the wetsuit and the physiological consequences for divers' WOB.

Consistent with the existing literature, we observed a statistically significant decline in FVC and FEV_1 during neoprene suit wear. However, the approximately 3% decrease in both parameters is not clinically relevant and is slightly smaller than previously reported.[3,4] These findings may be attributed to our larger and more homogeneous study population (exclusively male participants) and the absence of a possible fatigue effect, due to randomisation. More notably, we observed significant declines in FVC, FRC, and ERV, alongside significant improvements in IC and VT, findings not previously reported.

The observed reduction in FRC and increase in VT indicate restricted thoracic and abdominal expansion due to external

Table 2

Respiratory parameters and change when wearing a wetsuit; *$P < 0.05$; DL_{CO} – diffusing capacity for carbon monoxide; $DL_{CO}Adj/VA_{sb}$ – the ratio between diffusing capacity for carbon monoxide adjusted for haemoglobin and alveolar volume measured in a single breath; ERV – expiratory reserve capacity; FEV_1 – forced expiratory volume in one second; FEV_1/FVC – the ratio between forced expiratory volume in one second and forced vital capacity; FRC – functional residual capacity; FVC – forced vital capacity; IC – inspiratory capacity; R_{aw} 0.5 in – airway resistance at a flow of 0 ± 0.5; sG_{aw} 0.5 in – specific airway conductance at a flow of 0 ± 0.5; TLC – total lung capacity; VA_{sb} – alveolar volume measured in a single breath; VT – tidal volume

Parameter	No wetsuit Mean (SD) [Range]	Change (wetsuit vs no wetsuit) Mean (SD)
Spirometry		
FVC (L)	5.75 (0.70) [4.06–7.14]	-0.16 (0.17)*
FEV_1 (L)	4.51 (0.58) [3.26–5.71]	-0.13 (0.15)*
FEV_1/FVC (%)	78.6 (5.3) [65.1–87.0]	0.00 (0.02)
Lung volumes		
TLC (L)	7.54 (0.85) [5.54–9.6]1	-0.01 (0.17)
IC (L)	3.54 (0.68) [2.10–4.87]	0.16 (0.42)*
VT (L)	1.15 (0.28) [0.69–1.82]	0.10 (0.18)*
FRC (L)	4.00 (0.76) [2.59–5.64]	-0.16 (0.40)*
ERV (L)	2.01 (0.55) [0.88–3.27]	-0.22 (0.35)*
Airway resistance		
R_{aw} 0.5 ($kPa \cdot s^{-1} \cdot L^{-1}$)	0.139 (0.047) [0.06–0.22]	0.02 (0.04)*
sG_{aw} 0.5 ($L \cdot kPa^{-1} \cdot s^{-1}$)	1.79 (0.65) [0.96–3.09]	-0.25 (0.57)
Diffusion capacity		
DL_{CO} ($mmol \cdot min^{-1} \cdot kPa^{-1}$)	10.36 (1.19) [8.18–13.44]	-0.01 (0.56)
VA_{sb} (L)	6.73 (0.78) [4.84–8.48]	-0.13 (0.36)*
$DL_{CO}Adj/VA_{sb}$ ($mmol \cdot min^{-1} \cdot kPa^{-1} \cdot L^{-1}$)	1.55 (0.19) [1.21–2.15]	0.02 (0.08)

compression from the neoprene wetsuit. Theoretically, these changes likely contribute to an increased WOB, which can be visualised in a Campbell diagram. As FRC decreases and VT rises, the respiratory system operates at a less efficient point on the pressure-volume curve, demanding greater effort from respiratory muscles. Over time, this can lead to respiratory muscle fatigue and dyspnoea.[23]

Additionally, airway resistance, measured by body plethysmography, reflects the resistance to airflow in the large airways early in the respiratory cycle, and its elevation suggests that the neoprene wetsuit compresses the chest wall or airways, thereby increasing airflow resistance and further contributing to the increased WOB.[24,25]

While these physiological adjustments may not pose clinically significant challenges under resting conditions, they could exacerbate the physical demands of strenuous diving activities, in which divers experience hydrostatic pressure and wear not only a neoprene wetsuit but also additional equipment, such as diving cylinders, weight systems, harnesses, and the hydrostatic compression of thorax and abdomen. Under these conditions, where increased respiratory effort is already required, the cumulative effect of restrictive equipment may further compromise lung mechanics and gas exchange.

Figure 1

The main lung function parameters significantly decreased by wearing a neoprene wetsuit; *$P < 0.05$; ERV – expiratory reserve volume; FEV_1 – forced expiratory volume in one second; FRC – functional residual capacity; FVC – forced vital capacity

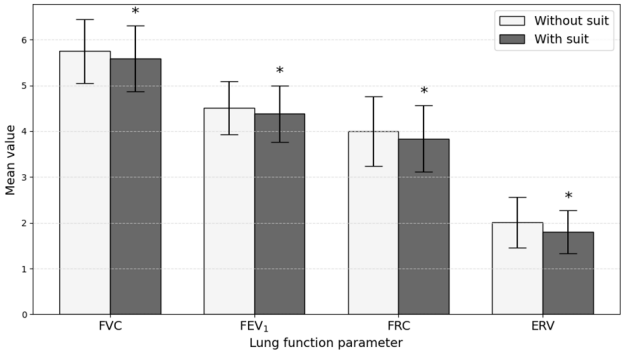


Table 3

Forced oscillation parameters; *$P < 0.05$; AX – reactance area the area under the reactance (X_{rs}) curve between 5 Hz and Fres; Fres insp – resonant frequency inspiratory (the frequency at which X_{rs} crosses zero); R_{rs} – resistance at frequencies 5, 11 and 19 Hz (measures airway resistance at specific frequencies); X_{rs} – reactance at frequencies 5, 11 and 19 Hz (represents the elastic and inertial properties of the respiratory system)

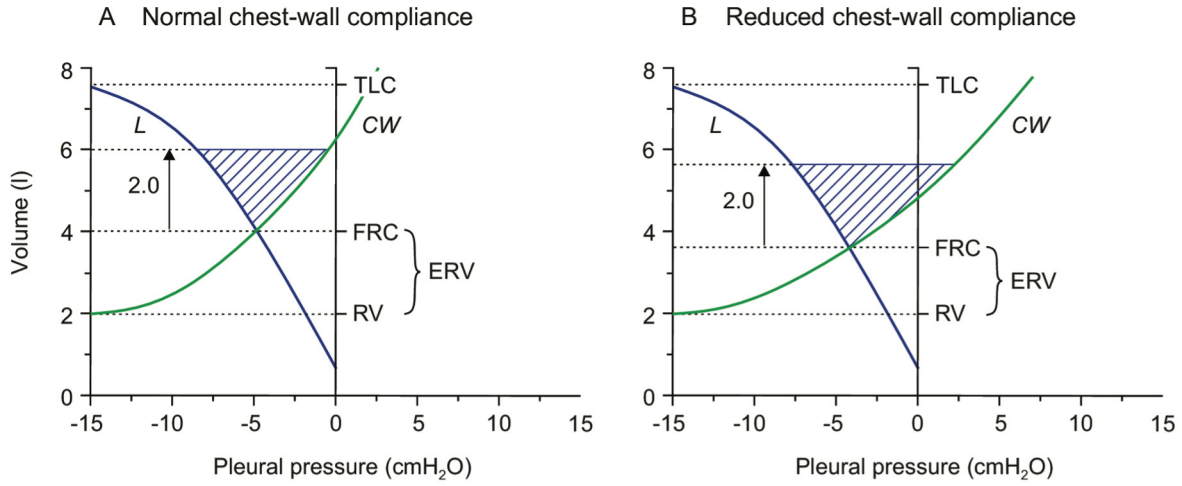
***n* = 31**	**No wetsuit Mean (SD) or Median [IQR]**	**Change (wetsuit vs no wetsuit) Mean (SD) or Median [IQR]**
R_{rs} 5 Hz (hPa·s^{-1}·L^{-1})		
R_{insp}*	0.213 [0.076]	-0.014 [-0.018]
R_{exsp}	0.238 [0.110]	+0.020 [0.001]
R_{tot}*	0.225 [0.091]	+0.019 [0.011]
R_{rs} 11 Hz (hPa·s^{-1}·L^{-1})		
R_{insp}	0.192 [0.074]	+0.001 [-0.015]
R_{exsp}	0.254 [0.106]	+0.004 [0.020]
R_{tot}	0.227 [0.087]	+0.004 [0.018]
R_{rs} 19 Hz (hPa·s^{-1}·L^{-1})		
R_{insp}	0.214 [0.071]	-0.010 [-0.007]
R_{exsp}	0.244 [0.082]	+0.003 [0.030]
R_{tot}	0.238 [0.085]	+0.000 [0.012]
X_{rs} 5 Hz (hPa·s^{-1}·L^{-1})		
X_{insp}	-0.055 (0.004)	-0.0122 (0.003)*
X_{exp}	-0.045 (0.004)	-0.008 (0.004)*
X_{tot}	-0.049 (0.004)	-0.010 (0.003)*
X_{rs} 11 Hz (hPa·s^{-1}·L^{-1})		
X_{insp}	0.030 (0.004)	+0.008 (0.003)*
X_{exp}	0.022 (0.004)	+0.013 (0.004)*
X_{tot}	0.025 (0.004)	+0.011 (0.003)*
X_{rs} 19 Hz (hPa·s^{-1}·L^{-1})		
X_{insp}	0.116 (0.006)	+0.010 (0.005)*
X_{exp}	0.098 (0.006)	+0.013 (0.005)*
X_{tot}	0.105 (0.006)	+0.012 (0.005)*
Derived parameters		
Fres insp (Hz)	8.831 (0.289)	+0.660 (0.190)*
AX insp (hPa·L^{-1})	0.122 (0.016)	+0.042 (0.136)*
R5 19 insp (hPa·s^{-1}·L^{-1})	-0.004 (0.004)	+0.009 (0.004)*

Additionally, the FOT results, particularly the increase in the reactance, suggest altered airway mechanics, likely due to mechanical compression of the chest wall or airways. While resistance may not show significant changes, the shift in reactance – especially at lower frequencies (e.g., 5 Hz) – often reflects increased stiffness of the respiratory system or peripheral airway narrowing, which may not be captured by resistance measurements (R_{rs} or R_{aw}) alone.[26] However, the broader implications of these FOT findings, including their effects on WOB, gas-exchange efficiency, and respiratory muscle fatigue, remain challenging to interpret. Further investigation is necessary to fully understand the implications for diver performance and safety, particularly under operational conditions where increased respiratory demand may exacerbate these effects.

The findings of this study indicate that wearing a neoprene wetsuit not only reduces spirometric parameters but also decreases the resting lung volume, thereby increasing WOB. This can contribute to dyspnoea and respiratory muscle fatigue, particularly during prolonged dives, with increased gas usage as a relevant consequence to divers. At greater depths, the neoprene wetsuit compresses and becomes thinner, potentially reducing its restrictive effect

Figure 2

Schematic pressure-volume relations for lung (L, blue) and chest wall (CW, green) in case of normal (A) and reduced chest-wall compliance (B); in both panels, the hatched area represents the elastic work of breathing needed to inhale 2.0 L of air from functional residual capacity (FRC). The volumes correspond to the mean values of Table 2. The volume change specified in these two diagrams depicts the group-mean results. The reduced chest-wall compliance appears as a flatter pressure-volume relation (the green curve in panel B). This leads to a lower intersection of both pressure-volume curves (a lower FRC, where the opposing elastic forces of lung and chest wall cancel each other out). The result is a greater work of breathing (the hatched area in panel B). ERV – expiratory reserve volume; RV – residual volume; TLC – total lung capacity



on breathing. However, WOB is likely more influenced by the increased density of breathing gases at depth. Less experienced or less trained divers may experience these effects more acutely, potentially impacting their operational effectiveness and safety.

Hypercapnia, or an elevated carbon dioxide (CO_2) level in the blood, is another critical concern in this context. The increased WOB imposed by the neoprene wetsuit's restrictive nature can lead to inadequate alveolar ventilation, resulting in CO_2 retention. This is particularly problematic during dives, where the combination of increased metabolic CO_2 production and restricted ventilation can rapidly elevate $PaCO_2$ levels. Hypercapnia can cause symptoms such as headache, confusion, and dyspnoea, and, in severe cases, loss of consciousness. Besides, an increase in $PaCO_2$ can also lead to a higher risk of decompression sickness.[27] Finally, for divers, this poses a significant risk, as impaired cognitive function and physical performance can compromise safety and operational efficiency.

STRENGTHS AND LIMITATIONS

To the best of our knowledge, this is the first study to assess the effects of neoprene wetsuits on resting lung volume using multiple modalities. The research was conducted in a controlled environment. All participants wore new wetsuits, and all measurements were performed in a pulmonary function laboratory under consistent conditions, using the same equipment and guidelines. This enhances the reliability of the findings. The study employed rigorous methodology, including randomisation of intervention order, standardised conditions, and measurements in a controlled environment, ensuring high scientific rigour.

However, several limitations should be acknowledged. First, given that all measurements were conducted in a controlled pulmonary laboratory, this is not the environment in which these neoprene wetsuits are worn. It would be valuable to investigate how immersion influences these results. Further studies are needed to examine the interactions among wetsuits, breathing effort, and physical exertion during actual diving.

Second, the Netherlands armed forces use 'one size fits all' wetsuits, based on chest circumference and height. While 17 of the 31 participants received a suit matching their chest circumference, the remaining participants required larger sizes due to their height. This resulted in a less pronounced but still statistically significant decline in FVC, FEV_1, FRC, and ERV, suggesting slightly reduced chest wall restriction in these individuals. However, the clinical or military relevance of these findings may be more nuanced, especially because neoprene wetsuits tend to be less restrictive in (deep) water due to pressure effects on the suit. Divers with pre-existing conditions, e.g., obesity, may experience more pronounced effects, whereas the impact on healthy individuals may only become clinically relevant during extended or high-intensity exercising dives. Although the dataset is not large enough to support valid subgroup analyses, the results for the aforementioned groups regarding matching suits are consistent with the manuscript's main conclusions.

Finally, there may have been a minor learning effect between pre- and post-measurements, particularly for spirometry. However, analysis of the data taking randomisation into account showed no learning effect. As all participants were experienced divers and, therefore, familiar with the tests, which are part of the annual medical assessment, we consider this risk is negligible.

Conclusions

This study shows that wearing a neoprene wetsuit leads to statistically significant reductions in spirometric parameters and lung volumes, particularly FRC and ERV, thereby increasing WOB. These changes may contribute to dyspnoea and respiratory muscle fatigue, especially during physically demanding or prolonged diving.

To better understand these dynamics, future research should examine these effects under submerged or immersed conditions, when environmental stressors, such as hydrostatic pressure, could exacerbate the physical challenges posed by restrictive dive gear.

References

1 Mitchell SJ, Bennett MH, Moon RE. Decompression sickness and arterial gas embolism. N Engl J Med. 2022;386(13):1254–64. doi: 10.1056/NEJMra2116554. PMID: 35353963.

2 Tetzlaff K. Pulmonary physiology and medicine of diving. Semin Respir Crit Care Med. 2023;44:705–18. doi: 10.1055/s-0043-1770065. PMID: 37369217.

3 Schellart NA, Sterk W. Influence of the diving wetsuit on standard spirometry. Diving Hyperb Med. 2016;46:138–41. PMID: 27723013. [cited 2026 Feb 18]. Available from: https://dhmjournal.com/images/IndividArticles/46Sept/Schellart_dhm.46.3.138-141.pdf.

4 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.

5 Blogg SL, Tillmans F, editors. DAN Annual diving report 2021 edition: A report on 2019 diving fatalities, injuries, and incidents. Durham (NC): Divers Alert Network; 2025.

6 Eberlein M, Schmidt GA, Brower RG. Chest wall strapping: An old physiology experiment with new relevance to small airways diseases. Ann Am Thorac Soc. 2014;11:1258–66. doi: 10.1513/AnnalsATS.201312-465OI. PMID: 25172621. PMCID: PMC5469355.

7 Pendergast DR, Lundgren CEG. The underwater environment: cardiopulmonary, thermal, and energetic demands. J Appl Physiol (1985). 2009;106:276–83. doi: 10.1152/japplphysiol.90984.2008. PMID: 19036887.

8 West JB. Respiratory physiology: The essentials, 9th ed. Philadelphia: Lippincott Williams, Wilkins; 2012.

9 Lumb AB. Nunn's applied respiratory physiology, 8th ed. Philadelphia: Elsevier; 2017.

10 Hlastala, MP, Berger AJ. Physiology of respiration, 2nd ed. Oxford University Press; 2001.

11 Oostveen E, MacLeod D, Lorino H, Farré R, Hantos Z, Desager K, et al. ERS Task Force on Respiratory Impedance Measurements. The forced oscillation technique in clinical practice: methodology, recommendations and future developments. Eur Respir J. 2003;22:1026–41. doi: 10.1183/09031936.03.00089403. PMID: 14680096.

12 Castagna O, Michoud G, Prevautel T, Delafargue A, Schmid B, Similowski T, et al. Broad individual immersion-scattering of respiratory compliance likely substantiates dissimilar breathing mechanics. Sci Rep. 2021;11(1):9434. doi: 10.1038/s41598-021-88925-x. PMID: 33941815. PMCID: PMC8093428.

13 World Medical Association. World Medical Association Declaration of Helsinki: Ethical principles for medical research involving human participants. JAMA. 2025;333:71–4. doi: 10.1001/jama.2024.21972. PMID: 39425955.

14 Du Bois D, Du Bois EF. A formula to estimate the approximate surface area if height and weight be known. 1916. Nutrition. 1989;5:303–11. PMID: 2520314.

15 Allen JL. Input oscillometry and the forced oscillation technique for assessing lung function in preschool children with asthma. Pediatr Investig. 2018;2:37–43. doi: 10.1002/ped4.12022. PMID: 32851228. PMCID: PMC7331333.

16 King GG, Bates J, Berger KI, Calverley P, de Melo P, Dellacà R, et al. Technical standards for respiratory oscillometry. Eur Respir J. 2020;55(2):1900753. doi: 10.1183/13993003.00753-2019. PMID: 31772002.

17 Lundblad LKA, Robichaud A. Oscillometry of the respiratory system: a translational opportunity not to be missed. Am J Physiol Lung Cell Mol Physiol. 2021;320:L1038–L1056. doi: 10.1152/ajplung.00222.2020. PMID: 33822645. PMCID: PMC8203417.

18 Graham BL, Steenbruggen I, Miller MR, Barjaktarevic IZ, Cooper BG, Hall GL, et al. Standardization of spirometry 2019 update. An official American Thoracic Society and European Respiratory Society technical statement. Am J Respir Crit Care Med. 2019;200(8):e70–e88. doi: 10.1164/rccm.201908-1590ST. PMID: 31613151. PMCID: PMC6794117.

19 Bhakta NR, McGowan A, Ramsey KA, Borg B, Kivastik J, Knight S, et al. European Respiratory Society/American Thoracic Society technical statement: standardisation of the measurement of lung volumes, 2023 update. Eur Respir J. 2023;62(4):2201519. doi: 10.1183/13993003.01519-2022. PMID: 37500112.

20 Graham BL, Brusasco V, Burgos F, Cooper, B., Jensen R, Kendrick A, et al. 2017 ERS/ATS standards for single-breath carbon monoxide uptake in the lung. Eur Respir J. 2017;49(1):1600016. doi: 10.1183/13993003.00016-2016. PMID: 28049168.

21 Quanjer PH, Stanojevic S, Cole TJ, Baur, X, Hall, GL, Culver, BH, et al. Multi-ethnic reference values for spirometry for the 3–95 year age range: the global lung function 2012 equations. Eur Respir J. 2012;40:1324–43. doi: 10.1183/09031936.00080312. PMID: 22743675. PMCID: PMC3786581.

22 Stanojevic S, Graham BL, Cooper BG, Thompson BR, Carter KW, Francis RW, et al. Official ERS technical standards: Global Lung Function Initiative reference values for the carbon monoxide transfer factor for Caucasians. Eur Respir J. 2017;50(3):1700010. doi: 10.1183/13993003.00010-2017. PMID: 28893868.

23 Gideon EA, Cross TJ, Coriell CL, Duke JW. The effect of estimating chest wall compliance on the work of breathing during exercise as determined via the modified Campbell diagram. Am J Physiol Regul Integr Comp Physiol. 2021;320(3):R268–R275. doi: 10.1152/ajpregu.00263.2020. PMID: 33356877.

24 Banner MJ, Jaeger MJ, Kirby RR. Components of the work of breathing and implications for monitoring ventilator-dependent patients. Crit Care Med. 1994;22:515–23. doi: 10.1097/00003246-199403000-00024. PMID: 8125004.
25 Storoni S, Treurniet S, Micha D, Celli M, Bugiani M, van den Aardweg J, et al. Pathophysiology of respiratory failure in patients with osteogenesis imperfecta: a systematic review. Ann Med. 2021;53:1676–87. doi: 10.1080/07853890.2021.1980819. PMID: 34569391. PMCID: PMC8477932.
26 Lappas AS, Tzortzi AS, Behrakis PK. Forced oscillations in applied respiratory physiology: clinical applications. Clin Res Pulmonol. 2014;2(2):1016. doi: 10.47739/2333-6625/1016.
27 Daubresse L, Vallée N, Druelle A, Castagna O, Guieu R, Blatteau J-E. Effects of CO_2 on the occurrence of decompression sickness: review of the literature. Diving Hyperb Med. 2024;54:110–9. doi: 10.28920/dhm54.2.110119. PMID: 38870953. PMCID: PMC11444918.

Acknowledgements

The authors would like to express their gratitude to all participants for their willingness to contribute to this study.

Conflicts of interest and funding: nil

Submitted: 19 February 2026
Accepted after revision: 16 May 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

Occupational health challenges in commercial diving: a qualitative study of psychological risk, training gaps, and organisational responsibilities

Holly Blake[1,2], Basharat Hussain[3], Lianne Hufton[4], Elisha Cousins[4], Graham Johnson[3,4], Andrew Tabner[3,4]

[1] *School of Health Sciences, University of Nottingham, Nottingham, UK*
[2] *NIHR Nottingham Biomedical Research Centre, Nottingham, UK*
[3] *School of Medicine, University of Nottingham, Nottingham, UK*
[4] *Emergency Department, University Hospitals of Derby and Burton NHS Foundation Trust, Derby, UK*

Corresponding author: *Professor Holly Blake, School of Health Sciences, University of Nottingham, Medical School, Queen's Medical Centre, Nottingham, NG7 2HA, UK*
ORCiD: *0000-0003-3080-2306*
holly.blake@nottingham.ac.uk

Keywords
Occupational stress; Organisational policy; Qualitative research; Training

Abstract
(Blake H, Hussain B, Hufton L, Cousins E, Johnson G, Tabner A. Occupational health challenges in commercial diving: a qualitative study of psychological risk, training gaps, and organisational responsibilities. Diving and Hyperbaric Medicine. 2026 30 September;56(3):226–236. doi: 10.28920/dhm56.3.226-236. PMID: 42743561.)
Introduction: Little is known about the occupational health needs of commercial divers, particularly regarding psychological wellbeing, trauma exposure, organisational support, and the adequacy of training for high-risk environments. This study qualitatively explored pathways into commercial diving, experiences of psychological strain and trauma, perceptions of support, and the relevance of training from the perspectives of divers, supervisors, and industry representatives.
Methods: This qualitative study used semi-structured interviews and focus groups with purposively sampled participants (n = 20 men). Data were analysed using reflexive thematic analysis.
Results: Three themes were identified: (1) pathways into a high-risk occupation shaped by financial incentives, risk acceptance, and informal entry routes; (2) psychological strain characterised by reported exposure to traumatic events, normalisation of distress, stigma surrounding mental health disclosure, and predominantly reactive organisational support; and (3) perceived inadequacies in training, described as generic, low-fidelity, and insufficiently aligned with the operational realities of offshore and saturation diving, alongside limited opportunities to practise key skills, contributing to feelings of unpreparedness during emergencies.
Conclusions: Participants described commercial diving as associated with substantial and often unaddressed psychological burden, compounded by cultural stigma, structural barriers to help-seeking, and predominantly reactive support systems. Training provision was perceived as outdated and insufficiently aligned with operational demands, with limited organisational investment in development. Findings suggest that industry-wide efforts may be needed to implement proactive mental-health strategies and improve the alignment of training with operational realities.

Introduction

Commercial diving is a highly specialised and inherently hazardous occupation, characterised by significant physical demands, complex technical tasks, and prolonged work in isolated, confined, and pressurised environments.[1–3] Offshore and saturation diving, in particular, require personnel to live and work under hyperbaric conditions for extended periods, often involving weeks of confinement, separation from family, and limited environmental stimulation. Empirical evidence indicates that saturation divers experience sustained physiological and psychosocial demands during prolonged exposure to these environments, where monotony, work–family conflict, and restricted living conditions contribute to cumulative psychological strain and necessitate substantial mental resilience.[1,3,4] Evidence from offshore oil and gas contexts further demonstrates that such environments are associated with fatigue, sleep disruption, and reduced performance, driven by long shifts, irregular schedules, and high operational demands.[5–7] In addition to these environmental and organisational stressors, divers operate in high-risk settings where exposure to traumatic incidents, including equipment failure, severe injury or fatality, is a recognised occupational hazard. Research on human performance in diving and comparable high-risk domains shows that acute stress responses, such as panic, increased cognitive load and impaired decision-making, can significantly affect operational safety.

Hyperbaric conditions introduce further psychophysiological challenges. Pressure-related factors, including nitrogen narcosis and high-pressure exposure, are known to impair

cognitive functioning, slow reaction times, and disrupt attention and information processing.[8–10] Experimental and observational evidence also indicates that prolonged dives are associated with physiological stress responses, including oxidative stress and fatigue, alongside adaptive shifts in cognitive performance under sustained pressure.[3,4] These interacting physiological and psychological stressors influence human performance in complex underwater environments and may amplify risk in already hazardous operational contexts.

Despite these well-recognised stressors, limited research has examined the broader occupational health needs of commercial divers, particularly in relation to psychological wellbeing, trauma exposure, and organisational support. Evidence from recreational diving identifies anxiety, depression, and stress-related responses associated with diving activities,[11] while studies in offshore and other high-risk industries report elevated psychological distress, including sleep disturbance and work-related stress, among personnel.[12,13] These findings highlight the importance of organisational and structural factors, including long shift rotations, isolation, high workload, job insecurity and limited access to support, in shaping mental health outcomes among offshore and high-risk workers.[12–14] High job demands, low control and inadequate organisational support are consistently associated with psychological distress, fatigue and impaired decision-making, with implications for workplace safety behaviour.[14–16] In offshore contexts, additional psychosocial hazards such as work–life interference, production pressures and fear of speaking up further contribute to risk and may increase the likelihood of accidents and errors.[13] Extending these findings to commercial diving, similar risks may be compounded by organisational and psychosocial factors, reinforcing the role of human and organisational influences as key determinants of safety outcomes in complex underwater environments.[13,15]

Given these gaps, there is a need for qualitative, experience-based research to better understand how these risks are experienced, interpreted, and managed in practice. The aim of this study was to explore commercial diving as a high-risk occupation by examining: (1) pathways into the profession; (2) experiences of psychological strain and trauma; (3) perceptions of organisational and peer support; and (4) the adequacy and relevance of current training for operational and health-related demands.

Methods

This study employed a qualitative design using data from individual semi-structured interviews and focus groups to explore participants' experiences and perspectives. All participants identified as male and were aged over 18 years. They were recruited using purposive sampling to ensure representation across key operational roles within commercial diving, including working divers, dive supervisors, and industry stakeholders involved in management, training, or safety oversight. Sampling criteria included role, years of experience, and exposure to different sectors of commercial diving (e.g., inshore, offshore, and saturation). Participants' experience ranged from a few years to approximately 40 years in the diving sector. We also sought variation in age and geographical work history to reflect the diversity of operational contexts in which psychological strain and training demands arise. Including both divers and supervisors enabled exploration of perspectives from those undertaking operational work and those responsible for decision-making, while industry stakeholders contributed organisational and policy-level insights.

Recruitment occurred via direct email invitation, and data were collected in April 2024. The divers were all recruited from one company operating in the North Sea, on a voluntary basis. Participants were recruited to take part in a parent study aimed at developing a resuscitation algorithm for divers in difficulty (i.e., to determine optimal approaches to delivering cardiopulmonary resuscitation [CPR] in hyperbaric environments). As part of this study, participants were interviewed about their experiences and perspectives. The present study represents a secondary qualitative analysis of these interview data.

Divers, dive supervisors and industry representatives were invited to take part in individual interviews with one of two researchers (LH, EC) trained in qualitative interview techniques. Additional industry representatives were invited to participate in one of two focus groups facilitated by both researchers. In this study, 'industry representatives' referred to individuals attending to provide senior operational advice on the algorithm for the parent study. They all had substantial prior experience as commercial divers and had all worked in the sector for several decades. At the time of the study, they held senior roles within diving organisations, including operational management, training oversight, and safety governance. Their inclusion allowed us to capture perspectives from those responsible for shaping organisational policies, training standards, and support structures, thereby complementing the lived experiences of divers and dive supervisors.

Sample size was guided by the concept of information power.[17] A total of 20 participants took part in the study (all men). Fifteen participants completed individual interviews, comprising 11 divers, two dive supervisors, and two industry representatives. An additional five industry representatives participated in one of two focus groups (group 1: $n = 2$, group 2: $n = 3$). Interviews were conducted during a week of activities developing an algorithm as part of the parent study. They took place away from the participants' normal place of work (e.g., a bespoke setup, specifically for the project). Participation, including interviews, took place during a dedicated project week and was considered paid work; participants were released from their usual duties by their employer to attend

Interview durations ranged from 11 to 49 minutes (average 24 minutes), and each focus group lasted approximately 85 minutes. All interviews took place in person and followed a semi-structured interview schedule that allowed for probing and follow-up questions. Sessions were audio-recorded with consent, and transcribed verbatim. Data were analysed using reflexive thematic analysis (RTA).[18] RTA was used because it provides a flexible, experience-focused approach suited to exploring divers' subjective experiences and perceptions without imposing predefined categories. The process involved familiarisation with the data, generating initial codes, constructing themes, reviewing and refining themes, and producing analytic narratives. Coding was conducted inductively by two members of the research team, with regular discussions to compare interpretations, refine codes, and develop themes. Differences in interpretation were resolved through iterative dialogue, supporting depth and coherence of the analysis. NVivo software[19] supported systematic data organisation. Reflexive notes and an audit trail of analytic decisions were maintained throughout, alongside regular team discussions, to enhance analytic rigour and transparency.

All participants provided informed consent and were assured of confidentiality and voluntary participation. The data were originally collected as part of a wider project,[20] and the interview questions were focused on experiences of and training for medical emergencies during saturation diving (*Supplementary file 1). Due to arising discussions around psychological risk during the interviews, ethical approval was sought for secondary analysis of this qualitative dataset to explore this further and was obtained from the University of Nottingham Faculty of Medicine and Health Sciences Research Ethics Committee on 26 February 2026 (REC ref no FMHS 118-0226). Confidentiality was protected using pseudonyms and the removal of identifying details during transcription. Audio files and transcripts were stored on secure, password-protected university servers accessible only to the research team. Given the sensitive nature of discussing psychological strain and traumatic operational experiences, participants were reminded that they could pause or stop the interview at any point, and signposting to appropriate support services was provided if distress arose. Participants were informed that they could request withdrawal of their data up to the point of anonymisation, after which removal was no longer possible due to de-identification.

REFLEXIVITY

Interviews were conducted by two female research nurses from a single UK hospital trust who were trained in qualitative interviewing. The interviewers had no prior professional or personal relationships with participants and no previous experience of, or involvement in, the commercial diving sector. This positioning supported a degree of analytic openness, reducing the likelihood of pre-existing assumptions shaping questioning or interpretation. However, it is acknowledged that differences in gender and professional background between interviewers (healthcare) and participants (commercial diving and industry leadership roles) may have introduced perceived power imbalances in both directions; for example, participants may have viewed interviewers as external to the industry and therefore either moderated or amplified their accounts. To mitigate potential bias and undue influence, interviewers followed a semi-structured guide, used open and non-leading questioning, and encouraged participants to elaborate on their experiences in their own terms. Reflexive practices were embedded throughout, including memo-writing, maintenance of an audit trail, and regular team-based analytic discussions to support critical reflection on coding and theme development. These approaches are consistent with reflexive thematic analysis,[18] where researcher subjectivity is recognised and actively engaged as part of the analytic process rather than treated as a source of error.

Results

Analysis of the data identified three overarching themes: motivation and pathways into the diving occupation; prevalence of psychological strain and its management; and divers' training associated with occupational health and wellbeing. Here, 'divers' refers to the broader diving community and 'participants' or 'interviewees' refers to research participants. While this qualitative study does not represent all commercial divers, the findings reflect patterns of meaning within this sample, which align with known structural features of the commercial diving sector (e.g., employment precarity, trauma exposure, training structures).

THEME 1: MOTIVATION AND PATHWAYS INTO THE DIVING OCCUPATION

Our research participants identified several factors that motivated them to pursue a career in diving. For many, the primary incentive was the high earning potential, particularly in offshore roles. Others were drawn to the demanding and adventurous nature of the work, viewing it as an opportunity to test their abilities and engage in tasks perceived as risky. Additional motivations included the enjoyable and flexible working conditions, as well as the chance to travel and work in diverse locations around the world.

> *Money was obviously massively enticing, and I was getting a month at home, six weeks at home to do with whatever I wanted. (Diver-3)*
> *[I joined diving] because I enjoy it…Yes, genuinely still enjoy it and the money's great and I don't have a plan b and that's it. (Diver-4)*

***Footnote:** Supplementary file 1 is available online on our website: https://www.dhmjournal.com/index.php/journals?id=434

I just like being in the water so that's what I do (Diver-2)
I fancied working offshore away from home… So, if you're going to be away, you may as well get paid more money for doing it. So that was something I factored in (Diver-9)

Most participants reported being introduced to diving through a family member or friend already working in the industry. A smaller number learned about the opportunity independently, often through newspapers or magazines. Prior to entering the diving profession some had served in the Royal Navy, while others had gained early exposure through recreational scuba diving during childhood.

The background of that is my two brothers were divers at that time as resettlement courses from the marines and the paras. I was given the opportunity because I couldn't see me going anywhere significant or exciting with the role I was taking at that time and so I took the opportunity and then signed up. (Diver-5)
I spent six years having a bit of fun in the navy and then decided I needed to grow up, and I left and became a commercial diver, I've been doing that since then for years, a long time now. (Diver supervisor-1)

THEME 2: PREVALENCE OF PSYCHOLOGICAL STRAIN AND ITS MANAGEMENT

Psychological strain among divers was reported as highly prevalent. Both divers and industry providers described offshore diving as frightening and, at times, traumatising - particularly in situations where divers attempted to rescue colleagues during emergencies but were unable to achieve a positive outcome. Participants also noted that divers often work far from their families and are deployed to various parts of the world. In some geographic regions, extreme heat, long working hours (often around 12 hours per day), and poor food and living conditions further exacerbate these psychological challenges.

I will show you pictures of the third-world hell holes that I've been to. That's actually trauma. That's more trauma than anything else that I've experienced working… Nobody could eat the food. It was 30-degree heat. You were working 12-hour shifts. It was constant relentless just hell. (Diver-11)
In my eyes it's not necessarily the physicality of the job, it's the psychological aspect of the job, away from your family, you've been away for so long, putting yourself inside a chamber for 28 days at a time. (Diver-10)

Participants described that divers form a close-knit community, which intensifies the trauma when they witness a close colleague coming to harm or dying in the workplace. It was noted that divers often experience the so-called 'second victim' phenomenon in such situations (i.e., emotional harm experienced by a responder involved in a traumatic or adverse event); however, awareness of this issue was perceived to be limited, with support mechanisms largely lacking.

The more you know someone obviously the more affected you are, but in this game, you see trauma all the time… [I] went to a lot of funerals, and you just start getting upset in the end. (Diver-6)
We never really look at second victim or anything like that. To be honest, the first time I heard the phrase second victim was here this week… They were like second victim. What's that? Honestly, that's the first time I've ever heard that term, so I probably don't know too much about that. (Diver-9)

High levels of stigma appeared to be attached to expressing psychological or mental health concerns in the diving workplace. Participants indicated that divers who disclose such difficulties are often perceived as weak and may be ostracised. Many also feared breaches of confidentiality and worried that raising psychological concerns could jeopardise future employment contracts.

You've got to remember the diving population is generally very, not introverted necessarily but they're a team together and they're very close and they really, really don't want to admit weakness because admitting weakness at 200 metres is not a good idea. So, they all tighten up and they're all Mr bravo and macho and everything else. (Industry-4)
If you were to go up to the offshore project manager and say that exact thing, he would say no problem at all. He would get you the first helicopter off that vessel to go home. Then you've got the stresses of losing the money and they've still got bills to pay so that's add its own element of stress and everything else. (Diver-10)

Participants described the industry as lacking a strong track record of providing mental health support to its workforce. Our interviewees frequently reported feeling powerless and unheard. Nonetheless, they referred to a small number of individuals within the sector who were actively championing mental health initiatives. These keen and enthusiastic advocates were described to be pushing the agenda forward - for example, one participant referred to a diver who encouraged their colleagues to engage in mental-health-focused journaling. In our sample, there was a recognition of the importance of such support, and how this was growing among diver managers more broadly. However, despite some companies beginning to offer counselling services, participants in our sample believed that divers rarely receive help at the time they need it most. Moreover, it was perceived that mental health support is typically provided only when divers request it, reflecting a reactive rather than proactive or preventive approach across the industry.

I know that there has been posters put up now on two of the vessels that I've been on recently about mental health kind of coaching and stuff. (Diver-1)
I just think it's a bit more open and acceptable now because of the generation thing again, all these closed books dinosaur people with these attitudes are now out the industry and the whole industry is changing for it as well there's a whole new outlook on people's wellbeing yes. (Diver-5)
I think the industry is slowly progressing but it's the whole argument of proactive versus reactive. At the minute we just solely focus on reactive measures and we're not very proactive as an industry. (Diver-10)

Participants reported that divers could be required to recover a colleague who is experiencing serious health difficulties or life-threatening complications. They spoke of a strong expectation that the recovery diver will go above and beyond to rescue the person in danger. However, participants reported that during such operations they often experience intense breathing effort, high stress, and significant performance pressure. In some cases, according to participant's accounts, divers attempting a recovery have themselves become immobilised or "*frozen*" underwater due to the overwhelming stress of the situation.

It was raised that companies employing divers were largely unwilling to engage in conversations about mental health or post-traumatic stress. Interviewees discussed the common practice following a traumatic incident - to send divers home and occasionally check on them by phone. Participants reported that organisations do not provide pre- or post-job psychological screening and had observed that no structured debriefing is offered to discuss how incidents affect those involved in the recovery.

We're never asked how we feel. Or when you come out of SAT, how are you physically? But we're never asked how do you feel mentally. How was that SAT psychologically? Was it mentally draining the team that you were in? Did you have to look after those divers? Did you have to carry them? They do not care (Diver-10)

In the absence of formal mental health or counselling support, it was perceived that divers are left to manage their traumatic experiences on their own or rely on family members - most often their wives - to discuss their psychological difficulties.

THEME 3: DIVERS' TRAINING ASSOCIATED WITH OCCUPATIONAL HEALTH AND WELLBEING

Participants reported that first aid/health and safety training within the diving industry is insufficient. The equipment used was described as low-tech and lacking fidelity. Compared with previous years, current training was perceived as increasingly theory-based, with minimal opportunities for hands-on experience.

The only training you do is a first aid course or your offshore survival, they are mandatory but it's just a piece of paper at the end of the day, the training is just, it's neither here nor there. (Diver supervisor-1)
Well in an ideal world you would have a setup like this, yes. Some places, some training centres do have a diving bell in there so you can get inside and you can practice but not all of them do. There's maybe just one or two that do. The place I go to, it's close to water. It's a good training centre. It used to be run by a paramedic. He's left now so I don't know what it's like now, but I felt like the trauma side of things, the training for trauma was really, really good. Anything to do with resuscitation, CPR, I thought was great as well. (Diver-8)

In the absence of adequate occupational health and safety/first-aid training, divers in our sample reported feeling unprepared to deliver effective care or achieve positive outcomes when incidents occur in the diving bell or underwater. It was also noted that, within the saturation environment, there is very little that can be done to assist a diver experiencing a medical emergency.

It's not adequate and it's not job specific. It's almost like your first aid at work. Do you know what I mean? It's as basic as that…It was one of the worst courses for medic training that I've ever been. The guy literally read off a PowerPoint presentation and then spun a few stories that he'd obviously heard about, not experienced himself or anything, very little practical work. They had a doctor come in and talk about diving physiology in a very monotone voice. So, there was nothing interesting about it. You're just sitting there for four hours. (Diver-11)

It was broadly believed by participants that divers generally have limited medical knowledge and are not trained healthcare professionals. Industry providers in our study noted that there is only so much emergency-care training these divers can realistically receive. They also reported that some divers are also reluctant to engage in training involving patients, blood, or first aid. Study participants indicated that, despite the requirement for biennial training refreshers, often completed primarily to secure higher pay, divers have limited opportunities to practise the diver medic technician (DMT) skills they acquire. As a result, these skills can fade over time.

So, over a two-year period, if they've not done anything and then suddenly, they have to do something, it's incredibly difficult. So, on average I would say less than 50% of the time that I ask for an IV to be put up, can one be set up. Indeed, once I ask for a urinary catheter to be put in, they couldn't do that either. (Industry-4)

Across the sample, companies employing divers were described as unwilling to invest in training or research related to the diving industry. Current training provision was

also perceived to offer poor value for money, particularly where courses are delivered online. Although most existing courses, including DMT training, were considered relatively inexpensive and accessible, divers noted that they are often responsible for organising their own attendance. Participants emphasised that employers should allocate dedicated budgets for diver training and make use of periods when divers are off-shift to provide it. Participants observed that the industry is slow to implement change and expressed scepticism about whether companies would invest meaningfully in training. Nonetheless, some remained hopeful that the arrival of a new generation of divers may bring positive developments for the sector.

> *We try to better it but until they kind of old dinosaurs that are running it move out of the way the new generation can't to change it. I'm hoping that as a new generation comes in, they will change it for the better, like training like this is a massive advancement and that's kind of where we want to be going. (Diver-2)*

Participants felt that the current training provision for divers is overly generic and does not reflect the realities of their working environment. They offered several suggestions for improvement, including the need for specialised training in resuscitation within saturation diving, as well as instruction on safely handling an individual in and out of a diving bell. Participants also recommended making the existing DMT training more practical and hands-on. Additionally, they suggested expanding the training content to include topics such as managing burns, responding to trauma, and using chest compression devices.

> *It needs to be way more practical, way more dive related and practical drills, so the current DMT course definitely needs [to be] reformed. (Diver-2)*
> *You don't just need to be looking at PowerPoints to be absorbing the theory behind it. You could be working a practical and still absorbing that information. We used to, on the DMT course, we used to witness an autopsy. We then used to work in the evenings in A&E [Accident and Emergency]. So again, you're putting that practice into real life situations. You're not asked to do very much, CPR [cardiopulmonary resuscitation], cannulations but still, your experience and the stress, the anxiety that comes with things like that in a real-life incident really. (Diver-10)*

Discussion

This study identified several key themes relating to participants' experiences, including pathways into the role and the psychological strain encountered during participation. Given that divers were recruited from a single North Sea operator, these findings should be interpreted within that organisational and geographic context, although they may reflect patterns relevant to similar commercial diving settings. Overall, findings highlight the complex interplay between anticipated benefits and lived experiences, with participants often negotiating both practical and emotional challenges. Motivations and pathways into the role (e.g., financial incentives and risk acceptance) may also shape participants' tolerance and appraisal of the psychological strain described in later themes. This aligns with prior research showing that saturation divers are motivated by high wages and job prestige, while simultaneously developing psychological endurance and adaptability to manage extreme occupational demands in a high-risk environment.[1,7]

Participants highlighted substantial structural, cultural, and operational challenges, particularly in relation to psychological wellbeing and training provision. In their accounts, divers depicted working environments characterised by high exposure to physical hazards (e.g., risks of entrapment, barotrauma, decompression related injury, and impact trauma associated with offshore and saturation operations), alongside significant emotional strain and risk of exposure to traumatic events. These findings align with existing commercial diving literature demonstrating the combined physiological and psychosocial demands of offshore and saturation environments, including exposure to multiple physical hazards and sustained environmental stressors, as well as psychosocial challenges such as confinement, monotony, and emotional strain.[1,7,21]

The close-knit nature of the diving community further intensifies the psychological impact of witnessing or responding to critical incidents, including unsuccessful rescue attempts (or the possibility of this occurring). Despite the clear prevalence of psychological strain described by participants, accounts suggested that a pervasive culture of stigma may discourage divers from disclosing mental-health concerns. This mirrors patterns seen across other high-risk, high-responsibility professions such as the military[22] or law enforcement,[23] where safety depends on trust, and cultural norms emphasise toughness, reliability, and emotional control. Fears of being labelled weak, ostracised by peers, or denied future employment were described as contributing to a pattern of silence and under-reporting.

Organisational responses to trauma were described by participants as inconsistent and largely reactive. Within this sample, formal mental-health support was perceived to be limited, often inaccessible at the point of need, and rarely embedded within routine operational practice. The reported absence of structured debriefing, psychological screening, or proactive monitoring was described as leaving divers reliant on informal support networks, particularly family members, to manage the emotional consequences of their work. Although a small number of individuals within the industry were reported to be advocating for improved mental-health provision, systemic change was perceived as slow. These observations are consistent with earlier investigations into occupational diving, which have highlighted a predominant

focus on physiological and physical health risks, with comparatively limited attention to psychological outcomes and mental-health support needs.[1,7] Commercial diving remains one of the most gender-imbalanced professions. United States workforce statistics broadly indicative of global trends suggest that 96.7% of divers are men;[24] this was reflected in our study's sample. A lack of preventive approaches to mental health has been observed in other male-dominated professions such as construction, forestry, mining, transport, and emergency services.[25,26]

Our findings reveal notable perceived inadequacies in current diver medical and/or first aid training provision. These results are consistent with other studies that have identified both knowledge gaps and a reduction in higher-level training capability,[27,28] including prior critiques of diver training frameworks and evolving competency requirements within commercial diving contexts.[28] Participants in our study consistently reported that current training is experienced as generic, outdated, and insufficiently aligned with the realities of saturation diving. Courses were described as overly theoretical, lacking practical components, and delivered with inadequate or outdated equipment. Divers also noted that limited opportunities to practise DMT skills may contribute to skill decay over time, a concern consistent with broader evidence that low-frequency, high-stakes medical skills are particularly vulnerable to rapid decay in the absence of recurrent practice and refresher training.[20,30] Skills decay is strongly associated with lack of practice[31] and has been identified in other medical and high-risk operational fields, such as emergency medicine,[32] military medical training,[33] prehospital care[34] and first-responder sciences.[35]

Participants' accounts highlighted not only concerns about the adequacy of current training but also uncertainty regarding the level of medical competence realistically expected of divers. Skills such as establishing an intravenous (IV) line or performing invasive procedures are taught within some DMT courses, yet divers are not trained healthcare professionals, nor are they expected to maintain competencies equivalent to those of an emergency physician, nurse, or land-based paramedic. This tension reflects broader discussions within diving medicine regarding the scope and limits of diver medic capability in hyperbaric environments, where operational constraints may further restrict intervention options. Several participants described feeling unprepared or anxious when required to perform rarely practised skills, suggesting that the challenge may lie as much in unclear or inconsistently defined competency expectations as in the quality of training itself.

These findings indicate that, within this sample, divers may sometimes expect more of themselves than is feasible within the constraints of their role, particularly given the limited opportunities for practice and the unique operational limitations of the saturation environment. Hyperbaric conditions, including cognitive effects associated with pressure and gas exposure, may further shape performance and decision-making under stress, reinforcing the need for training aligned with realistic operational capability. Accordingly, rather than concluding that training programmes should deliver a specific set of advanced clinical skills, our findings suggest that to improve training fidelity, programmes may need to incorporate clearer definitions of expected competence, more realistic scenario-based practice, and training content that aligns with what divers can reasonably perform *in situ*.

Financial investment from major employers, including oil companies, was described by participants as minimal, with divers often responsible for organising and funding their own training. While some optimism exists that newer generations entering the profession may drive cultural and structural improvements, the current pace of change was perceived by participants as inadequate.

A further complexity shaping the wellbeing and safety experiences of commercial divers relates to the industry's employment structure. Historically, many divers have worked as independent contractors rather than salaried employees, creating a form of precarious work characterised by limited job security, ambiguous employment relationships, and restricted access to social protections such as paid sick leave or income support when removed from duty. This mirrors broader patterns observed across high-risk maritime and offshore sectors, where atypical or contingent employment arrangements are common and have been shown to heighten vulnerability to occupational health and safety risks.[36] As participants in this study described, being taken off a vessel for psychological or medical reasons can result in immediate financial loss due to the absence of employer-provided sick pay; an outcome that reflects the structural features of self-employment rather than individual employer misconduct. Although precarious work is associated with higher risk for physical and mental ill-health,[37] such arrangements may shift responsibility for health-related downtime onto divers themselves, potentially limiting incentives for companies to invest proactively in psychological support and discouraging individuals from reporting distress.

This dynamic is not unique to diving; offshore oil and gas research identifies work status (particularly insecure or casual employment arrangements) as a significant psychosocial stressor, contributing to feelings of insecurity and vulnerability and occurring alongside a broader workplace culture characterised by fear of speaking up about safety and health concerns.[13] Precarious employment has also been associated with heightened safety risks in the wider maritime workforce, where uncertainties around scheduling, deployment, and vessel assignment – often involving short-notice placement and insufficient rest – contribute to reduced wellbeing and increased perceived risk to safety.[36] From a regulatory standpoint, diving operations are

governed by robust safety frameworks that define minimum standards for operational planning, equipment, emergency preparedness, and medical fitness.[38,39] However, these regulatory instruments focus primarily on physical risks and operational hazards, and they do not explicitly address organisational responsibilities for supporting psychological wellbeing, reflecting a broader tendency within occupational safety and health frameworks to prioritise physical hazards over psychosocial risks.[40,41] A clearer distinction therefore emerges between regulatory responsibility – compliance with statutory safety requirements - and moral responsibility, which encompasses the broader duty of care organisations have toward ensuring the mental, emotional, and financial security of those whose labour underpins offshore operations. Evidence from offshore oil and gas research highlights that fear of raising concerns is a key psychosocial hazard affecting safety, while broader organisational research identifies psychological safety as fundamental to maintaining a safe and high-performing workforce.[13,42] Given divers' exposure to extreme physiological and psychological demands, and the precarious employment context in which many operate, improving workforce sustainability may require employers, particularly major contracting and operating companies, to move beyond minimal regulatory compliance. This includes recognising that wellbeing is not simply an individual responsibility but may also require coordinated organisational and industry-level approaches to support a competent, stable, and psychologically resilient diving workforce.

Based on these findings, Figure 1 presents a conceptual model of occupational health challenges in commercial diving, illustrating key stressors, mediating factors, and outcomes, and the interplay between organisational and individual influences.

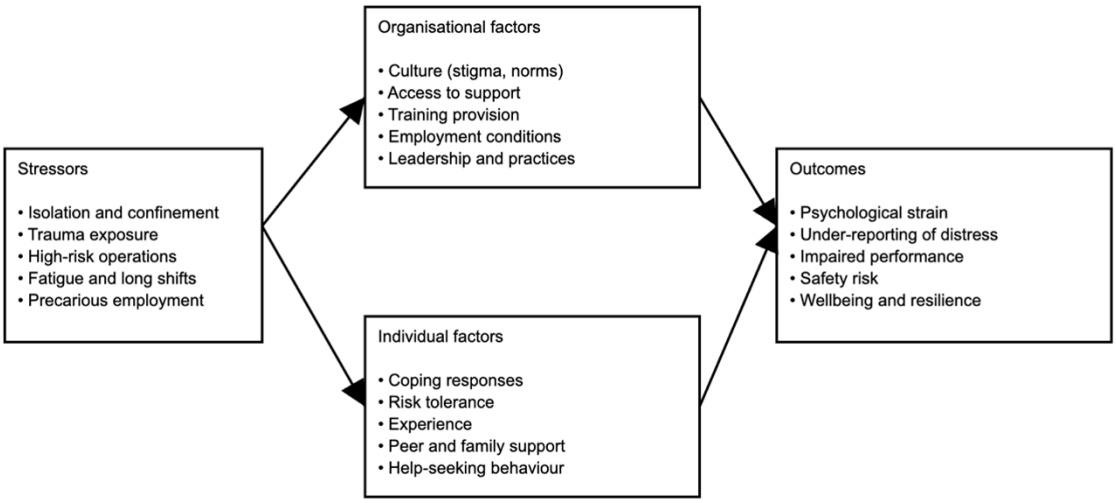
LIMITATIONS

Data were drawn from interviews and focus groups, which enriched the dataset but also introduced differing social dynamics that may have shaped responses.[43] The sample was purposively recruited from a single commercial diving company operating in the North Sea, which may limit transferability to other organisations, regions, or types of diving. This context also raises the possibility of selection bias and may have influenced participants' accounts of organisational practices. The dataset was originally collected as part of a broader study focused on resuscitation in diving, meaning psychological issues were not the primary focus of the interview schedule. This may have shaped both the direction of discussion and the salience of medical or emergency-related experiences (contextual priming). Although steps were taken to encourage candid responses, the shared project setting may also have influenced how participants framed their views. The study did not include perspectives from regulators, independent training providers, or employers beyond the participating organisation. As a result, findings may reflect a partial view and should not be interpreted as representative of the industry as a whole. More broadly, the sample was limited to men, reflecting industry demographics but restricting wider applicability, and findings were based on self-reported experiences, which may be influenced by recall or social desirability.

Conclusions

Several recommendations emerge from participants' accounts for strengthening psychological support, improving training quality, and fostering a safer and more sustainable diving workforce. First, findings suggest that the industry may benefit from adopting a more proactive approach to mental-health provision. This could include implementing

Figure 1
Occupational health challenges in commercial diving



routine psychological screening before and after high-risk operations, establishing structured debriefing processes following traumatic events, and improving timely access to qualified mental-health professionals. Normalising conversations about psychological wellbeing and reducing stigma may be supported through leadership engagement, peer-support initiatives, and targeted awareness programmes.

Second, participants' accounts indicate the potential value of greater organisational investment in mental-health infrastructure to move beyond ad-hoc or reactive support. This may involve developing clearer pathways for confidential reporting of psychological concerns, supporting help-seeking without adverse employment consequences, and more explicitly integrating mental-health considerations into operational planning. The development of more consistent standards for mental-health provision across organisations may also help reduce variability in access to support.

Third, findings suggest that medical training provision would benefit from further development to improve relevance, quality, and practical applicability. Training programmes could incorporate more realistic, scenario-based exercises reflecting the complexities of saturation and offshore environments. Particular emphasis may be placed on emergency response, resuscitation in hyperbaric conditions, casualty handling in and out of the bell, and the management of burns, trauma, and decompression-related injuries. Enhancing practical components of DMT training and providing more regular opportunities for skill maintenance may help mitigate skill degradation.

Fourth, participants highlighted the perceived importance of greater investment in diver training and research. This could include improved access to modern training equipment, such as high-fidelity simulation tools, to support preparedness and confidence. Scheduling training during off-shift periods may also improve accessibility without compromising operational demands.

Finally, findings suggest that ongoing cultural and organisational development may be important for supporting long-term improvements in wellbeing, safety, and professional development. As new generations enter the profession, there may be opportunities to further embed more progressive approaches to mental health and safety. Supporting such developments through policy evolution, leadership training, and collaborative industry initiatives may help facilitate sustained change.

References

1 Romsbotn S, Eftedal I, Vaag JR. A work environment under pressure: psychosocial job demands and resources among saturation divers. Front Public Health. 2022;10:765197. doi: 10.3389/fpubh.2022.765197. PMID: 35570940. PMCID: PMC9095950.

2 Risberg J, Midtgaard H. Decompression sickness in surface decompression breathing air instead of oxygen. Diving Hyperb Med. 2024;54:242–8. doi: 10.28920/dhm54.3.242-248. PMID: 39288932. PMCID: PMC11444916.

3 Barassi A, Brizzolari A, Rubino F, Caretti A, Nenna F, Egi M, et al. Psychophysical stress during a 24 h dive: a case study of an older male diver. Physiol Rep. 2026;14(6):e70836. doi: 10.14814/phy2.70836. PMID: 41872979. PMCID: PMC13098038.

4 Deb SK, Swinton PA, Dolan E. Nutritional considerations during prolonged exposure to a confined, hyperbaric, hyperoxic environment: recommendations for saturation divers. Extreme Physiol Med. 2016;5:1. doi: 10.1186/s13728-015-0042-9. PMID: 26744625. PMCID: PMC4704397.

5 Santos WUF, Stolz CM, Amario M. Fatigue and sleep deprivation in the offshore oil and gas industry: a systematic review of health, performance and safety implications. Safety. 2026;12:45. doi: 10.3390/safety12020045.

6 Kang J, Payne SC, Sasangohar F, Mehta RK. The impacts of rotating shiftwork on worker fatigue levels and associated adaptations: a naturalistic study across offshore platforms in the Gulf of Mexico. Hum Factors. 2025;68:109–22. doi: 10.1177/00187208251358635. PMID: 40653711.

7 Imbert JP, Balestra C, Kiboub FZ, Loennechen Ø, Eftedal I. Commercial divers' subjective evaluation of saturation. Front Psychol. 2019;9:2774. doi: 10.3389/fpsyg.2018.02774. PMID: 30692957. PMCID: PMC6340096.

8 Karakaya H, Aksu S, Egi SM, Aydin S, Uslu A. Effects of hyperbaric nitrogen narcosis on cognitive performance in recreational air SCUBA divers: An auditory event-related brain potentials study. Ann Work Expo Health. 2021;65:505–15. doi: 10.1093/annweh/wxaa132. PMID: 33942846.

9 Sharma RI, Marcinkowska AB, Mankowska ND, Waśkow M, Kot J, Winklewski PJ. Cognitive functions in scuba, technical and saturation diving. Biology (Basel). 2023;12:229. doi: 10.3390/biology12020229. PMID: 36829505. PMCID: PMC9953147.

10 Bliznyuk A, Hollmann M, Grossman Y. High pressure stress response: Involvement of NMDA receptor subtypes and molecular markers. Front Physiol. 2019;10:1234. doi: 10.3389/fphys.2019.01234. PMID: 31611813. PMCID: PMC6777016.

11 St Leger Dowse M, Whalley B, Waterman MK, Conway RM, Smerdon GR. Diving and mental health: the potential benefits and risks from a survey of recreational scuba divers. Diving Hyperb Med. 2019;49:291–7. doi: 10.28920/dhm49.4.291-297. PMID: 31828748. PMCID: PMC7039781.

12 Baygi F, Mohammadian Khonsari N, Seif E, Asayesh H, Qorbani M. The mental health status of offshore oil platform workers during the COVID-19 pandemic. Front Psychiatry. 2022;13:1009602. doi: 10.3389/fpsyt.2022.1009602. PMID: 36304563. PMCID: PMC9593038.

13 D'Antoine E, Jansz J, Barifcani A, Shaw-Mills S, Harris M, Lagat C. Psychosocial safety and health hazards and their impacts on offshore oil and gas workers. Safety. 2023;9:56. doi: 10.3390/safety9030056.

14 Asare BY, Kwasnicka D, Powell D, Robinson S. Health and well-being of rotation workers in the mining, offshore oil and gas, and construction industry: a systematic review. BMJ Glob Health. 2021;6;7:e005112. doi: 10.1136/bmjgh-2021-005112. PMID: 34301674. PMCID: PMC8311322.

15 Derdowski LA, Mathisen GE. Psychosocial factors and safety in high-risk industries: a systematic literature review. Saf Sci. 2023;157:105948. doi: 10.1016/j.ssci.2022.105948.

16 Berthelsen M, Pallesen S, Bjorvatn B, Knardahl S. Shift schedules, work factors, and mental health among onshore and offshore workers in the Norwegian petroleum industry. Ind Health. 2015;53:280–92. doi: 10.2486/indhealth.2014-0186. PMID: 25740007. PMCID: PMC4466879.
17 Malterud K, Siersma VD, Guassora AD. Sample size in qualitative interview studies: Guided by information power. Qual Health Res. 2016;26;13:1753–60. doi: 10.1177/1049732315617444. PMID: 26613970.
18 Braun V, Clarke V. Reflecting on reflexive thematic analysis. Qual. Res. Sport Exerc. Health. 2019;11;4:589–97. doi: 10.1080/2159676X.2019.1628806.
19 Lumivero. NVivo (Version 14) [Computer software]; 2023. [cited 2026 Jun 11]. Available from: https://lumivero.com.
20 Johnson G, Tabner A, Tilbury N, Wesson A, Hughes GD, Elder R, et al. Development of an algorithm to guide management of cardiorespiratory arrest in a diving bell. Resusc Plus. 2024;19:100724. doi: 10.1016/j.resplu.2024.100724. PMID: 39100390. PMCID: PMC11295632.
21 Monnoyer R, Lautridou J, Deb S, Hjelde A, Eftedal I. Using salivary biomarkers for stress assessment in offshore saturation diving: a pilot study. Front Physiol. 2021;12:791525. doi: 10.3389/fphys.2021.791525. PMID: 34916964. PMCID: PMC8669759.
22 Bogaers R, Geuze E, van Weeghel J, Leijten F, Rüsch N, van de Mheen D, et al. Decision (not) to disclose mental health conditions or substance abuse in the work environment: a multiperspective focus group study within the military. BMJ Open. 2021;11;10:e049370. doi: 10.1136/bmjopen-2021-049370. PMID: 34706950. PMCID: PMC8559108.
23 Richard C, Corbière M, Fiset-Renaud H, Caiada M, Lamontagne J, Diotte F, et al. Disclosure impact of mental health conditions in the workplace: a scoping review and a thematic analysis. J Occup Rehabil. 2026;36:131–66. doi: 10.1007/s10926-025-10288-1. PMID: 40192917.
24 Zippia. Commercial diver demographics and statistics in the US; 2026. [cited 2026 Jun 11]. Available from: https://www.zippia.com/commercial-diver-jobs/demographics/.
25 Roche E, Richardson N, Sweeney J, O'Donnell S. Workplace interventions targeting mental health literacy, stigma, help-seeking, and help-offering in male-dominated industries: A systematic review. Am J Mens Health. 2024;18;2:15579883241236223. doi: 10.1177/15579883241236223. PMID: 38581228. PMCID: PMC10998494.
26 Seaton CL, Bottorff JL, Oliffe JL, Medhurst K, DeLeenheer D. Mental health promotion in male-dominated workplaces: Perspectives of male employees and workplace representatives. Psychol. Men Masc. 2019;20;4:541–52. doi: 10.1037/men0000182.
27 Karadurmuş U, Bıçaklı R, Erdoğan G. Knowledge-level assessment of commercial divers: a focus on fundamental decompression procedures. Int J Occup Saf Ergon. 2025;31;3:672–9. doi: 10.1080/10803548.2025.2455865. PMID: 39935218.
28 Smart D. Back to the future: occupational diver training in Australia. Diving Hyperb Med. 2017;47:214–5. doi: 10.28920/dhm47.4.214-215. PMID: 29241229. PMCID: PMC6706341.
29 Anderson GS, Gaetz M, Masse J. First aid skill retention of first responders within the workplace. Scand J Trauma Resusc Emerg Med. 2011;19:11. doi: 10.1186/1757-7241-19-11. PMID: 21303536. PMCID: PMC3044091.
30 Klostermann M, Conein S, Felkl T, Kluge A. Factors influencing attenuating skill decay in high-risk industries: a scoping review. Safety. 2022;8:22. doi: 10.3390/safety8020022.
31 Winfred A, Day EA. Skill Decay: The science and practice of mitigating skill loss and enhancing retention. In: Ward P, editor. The Oxford handbook of expertise. Oxford Academic; 2018. [cited 2026 Jun 11]. Available from: https://academic.oup.com/edited-volume/34285/chapter-abstract/290673398?redirectedFrom=fulltext.
32 Clyne B, Barber Doucet HB, Brown L, Musits A, Jacobs E, Merritt C, et al. Maintaining procedural skills for academic emergency medicine faculty: A needs assessment. AEM Educ Train. 2021;5(4):e10648. doi: 10.1002/aet2.10648. PMID: 34853821. PMCID: PMC8609535.
33 Linde AS, Caridha J, Kunkler KJ. Skills decay in military medical training: A meta-synthesis of research outcomes. Mil Med. 2018;183(1-2):e40–e44. doi: 10.1093/milmed/usx023. PMID: 29401337.
34 Maddocks W. Skill fade in military medical training: a literature review of supraglottic airway use in the prehospital environment. J Mil Vet Health. 2020;28;3:34–41. doi: 05.2021-54148286/JMVH Vol 28 No 3.
35 Woodman S, Bearman C, Hayes P. Understanding skill decay and skill maintenance in first responders. Aust J Emerg Manage. 2021:44–9. doi: 10.47389/36.4.44.
36 Devereux H, Wadsworth E. Work scheduling and work location control in precarious and 'permanent' employment. Econ Labour Relat Rev. 2021;32;2:230–46. doi: 10.1177/10353046209814O5.
37 Kinman G. Creating better quality work and workplaces. Society of Occupational Medicine; 2021. [cited 2026 Jun 11]. Available from: https://www.som.org.uk/Creating_better_quality_work_and_workplaces.pdf.
38 Occupational Safety and Health Administration. (2021–2022). Commercial diving – Overview. [cited 2026 Jun 11]. Available from: https://www.osha.gov/commercial-diving
39 Electronic code of federal regulations. 29 CFR Part 1910 Subpart T – Commercial Diving Operations; 2026. [cited 2026 Jun 11]. Available from: https://www.ecfr.gov/current/title-29/subtitle-B/chapter-XVII/part-1910/subpart-T
40 Azzi M. Psychosocial risks at work: why they must be treated as core occupational safety and health hazards. British Safety Council / International Labour Organization; 2026. [cited 2026 Jun 11]. Available from: https://www.britsafe.org/safety-management/2026/psychosocial-risks-at-work-why-they-must-be-treated-as-core-occupational-safety-and-health-hazards
41 Di Tecco C, Persechino B, Iavicoli S. Psychosocial risks in the changing world of work: Moving from the risk assessment culture to the management of opportunities. Med Lav. 2023;114(2):e2023013. doi: 10.23749/mdl.v114i2.14362. PMID: 37057349. PMCID: PMC10133769.
42 Edmondson AC. Psychological safety and learning behavior in work teams. Admin Sci Q. 1999;44:350–83. doi: 10.2307/2666999.
43 Guest G, Namey E, Taylor J, Eley N, McKenna K. Comparing focus groups and individual interviews: findings from a randomized study. Int J Soc Res Methodol. 2017;20:693–708. doi: 10.1080/13645579.2017.1281601.

Acknowledgements

The authors thank the organisation from which participants were recruited for supporting staff participation in the study. For the purpose of open access, the authors have applied a Creative Commons Attribution (CC BY) licence to any Author Accepted Manuscript arising from this submission, in accordance with the University of Nottingham Open Access Policy.

Conflicts of interest and funding

GJ received funding for this study from TAC Healthcare, Aberdeen. The project was supported by: Professional Diving Academy, Submarine Manufacturing and Products, Boskalis, NUI, BP, DFS Diving, Equinor, IMCA, K-Subsea, KD Marine, Rever, RockSalt Subsea, Shelf Subsea, Subsea 7, TechnipFMC, Total Energies, Unique Hydra (PTY) Ltd, Well-Safe Solutions.

The funders had no role in the design or conduct of the study, data collection, analysis or interpretation, or the decision to publish. Representatives from several funding organisations contributed in a professional capacity by sharing their experience and expertise; however, they had no influence over the study design, data analysis, interpretation, or publication. Participation by all research team members and industry representatives was either voluntary or undertaken as part of their normal employment, and no additional payments were provided.

Submitted: 9 March 2026
Accepted after revision: 5 July 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

A calculation method for inert gas tension based on a multi-tissue model

Shuai Di[1], Chumeng Liu[1], Jinghua Gu[1]

[1] *Naval Medical Center, Naval Medical University, Shanghai, China*

Corresponding author: *Professor Jinghua Gu, 880 Xiangyin Road, Yangpu District, Shanghai, China*
ORCiD: *0009-0007-9622-7822*
gujingh@163.com

Keywords
Decompression; Decompression sickness; Diving; Diving medicine; Nitrogen; Pressure

Abstract
(Di S, Liu C, Gu J. A calculation method for inert gas tension based on a multi-tissue model. Diving and Hyperbaric Medicine. 2026 30 September;56(3):237–244. doi: 10.28920/dhm56.3.237-244. PMID: 42743562.)
Introduction: This study developed a mathematical model capable of accurately estimating inert gas tension within human tissues under continuously changing ambient pressure.
Methods: Based on Haldane's decompression theory and a multi-tissue model, and grounded in the principle that the rate of gas diffusion is proportional to the pressure gradient, a differential equation was derived and solved. Analytical solutions were obtained for four prototypical ambient pressure patterns: constant pressure, exponential compression, linear compression, and linear decompression. The model's validity was confirmed by comparison with the piecewise approximation method using a sufficiently small step interval.
Results: The model allows precise calculation of theoretical tissue inert gas tension at any given time. In air-breathing fast buoyant ascents, for fast tissues with half-times of 30 s and 80 s, the inert gas tensions calculated using the conventional method (374 kPa and 233 kPa) were notably higher than those obtained via the proposed model (331 kPa and 217 kPa), with a maximum difference of 13%. The proposed model closely matched the piecewise method (1 s interval), differing by only 1 kPa. For tissue half-times exceeding 5 min, all three methods produced similar results. In a standard air diving decompression schedule, both the proposed and conventional methods yielded initial decompression stop depths rounded to 18 m, showing no significant difference.
Conclusions: The model demonstrates clear advantages for fast tissues, and under varying ambient pressures, establishes a theoretical foundation for the dynamic prediction of tissue inert gas tension across diverse diving scenarios. It can be utilised in safety assessments of fast buoyant ascent, offering substantial theoretical relevance and practical applicability.

Introduction

Inert gases, characterised by their low chemical reactivity and lack of involvement in metabolic processes, exist exclusively in a physically dissolved form within the body. Within the context of diving medicine, these gases encompass nitrogen, helium, neon, argon, krypton, and xenon. Nitrogen, constituting the predominant component of atmospheric air, represents the principal inert gas encountered in conventional air diving. The human body neither metabolises nor regulates nitrogen, thereby posing distinct challenges in the field of diving medicine. In the early twentieth century, Haldane and his colleagues conducted analyses of air diving experiences alongside animal studies to elucidate the behaviour of nitrogen within the body during diving. This work culminated in the formulation of decompression theory, which serves as the foundational framework for contemporary understanding of inert gas dynamics in diving medicine.

Under stable ambient pressure conditions, gas exchange within bodily tissues attains an equilibrium state. When ambient pressure changes, nitrogen diffuses bidirectionally between the alveoli, bloodstream, and tissues, from blood to tissues during compression, and from tissues to alveoli during decompression. The rate of diffusion is directly proportional to the pressure gradient. The time required for an inert gas to reach 50% saturation in a tissue is defined as the half-saturation time (or half time), which varies according to tissue composition. Haldane's multi-tissue model categorises tissues into five hypothetical compartments with half-times of 5 min, 10 min, 20 min, 40 min, and 75 min, independent of pressure. These compartments are referred to as "*theoretical tissues*".[1] The model remains a cornerstone in the global diving community, underpinning recreational, technical, commercial, and military diving, with modern refinements dating to the 1960s.[2–4]

The functional definition of theoretical tissues is straightforward: their uptake and elimination of inert gases proceed in a fully symmetrical fashion. Studies have shown that the random movement and exchange of inert gas molecules between regions of different concentrations are driven by local pressure gradients, specifically, the difference between arterial inert gas tension and instantaneous tissue inert gas tension. To quantitatively describe the kinetics of

inert gases in the human body, a conventional mathematical model with respect to time *t* has been established, as shown in Eq. (1).[1–6]

$$P_T = P_a + \left(P_{T_i} - P_a\right)e^{-kt} \tag{1}$$

where:
P_T is the inert gas tension in a given tissue at time *t*;
P_a is the arterial inert gas tension at time *t* (assumed to be equal to the inert gas tension in the breathing gas);
P_{Ti} is the initial tissue inert gas tension;
k is the saturation rate constant, related to the half-saturation time of the corresponding tissue.

The conventional model demonstrates accuracy solely under conditions of constant ambient pressure. However, actual diving scenarios involve continuous fluctuations in pressure during both descent (characterised by increasing pressure) and ascent (characterised by decreasing pressure). Existing methodologies face challenges in precisely capturing the dynamics of inert gas tension throughout these phases. In practice, several approximations are frequently employed, including adding descent time to bottom time to approximate total underwater exposure for tissue inert gas saturation; using the average of initial and final pressures during ascent as a surrogate for a constant equivalent pressure; or segmenting continuous pressure changes into multiple intervals of steady pressure. These approximations implicitly assume that the human body is subjected instantaneously to a constant-pressure environment, thereby disregarding the continuous nature of pressure variation. Given that instantaneous pressurisation or depressurisation is physiologically unfeasible, such simplifications inevitably lead to discrepancies between theoretical predictions and actual physiological responses. To address this limitation, the present study aimed to develop a concise mathematical model capable of accurately calculating inert gas tension in human tissues under conditions of continuously varying ambient pressure.

Methods

Let $f(x)$ denote the ambient pressure, specifically the pressure of the breathing gas, as a function of time x, and let $g(x)$ represent the pressure within a particular tissue over the same temporal variable, with $g'(x)$ signifying the rate of change of $g(x)$. According to the theoretical framework underpinning this analysis, variations in $f(x)$ elicit corresponding changes in $g(x)$, which serve to reduce the pressure difference between $g(x)$ and $f(x)$. Moreover, the rate of change $g'(x)$ is directly proportional to this pressure gradient. These considerations lead to the derivation of Eq. (2).

$$g'(x) = k\left[f(x) - g(x)\right] \tag{2}$$

Where:
The function $f(x)$ represents the ambient pressure, predicated on the assumption that arterial inert gas tension is equal to the inert gas tension in the breathing gas. It is expressed in kPa. The corresponding inert gas tension is then obtained by multiplying $f(x)$ by the fractional concentration of the inert gas in the gas mixture. For clarity and ease of understanding, this study defines 100 kPa as one standard atmosphere and assumes that pressure increases by 100 kPa for every 10-metre descent in water depth. This assumption is made independently of the water's density.

The function $g(x)$ represents the pressure within a given tissue, measured in kPa. Its derivative, $g'(x)$, denotes the rate of change of tissue pressure with respect to time, expressed in $kPa \cdot s^{-1}$.

The parameter *k* is defined as the saturation rate constant, a dimensionless quantity with positive values.

From Eq. (2), Eq. (3) is derived:

$$g'(x) + kg(x) = kf(x) \tag{3}$$

Eq. (3) denotes a first-order nonhomogeneous linear differential equation, and its standard form is given by Eq. (4):

$$\frac{dy}{dx} + P(x)y = Q(x) \tag{4}$$

Its general solution is given by Eq. (5):

$$y = Ce^{-\int P(x)dx} + e^{-\int P(x)dx}\int Q(x)e^{\int P(x)dx}dx \tag{5}$$

By comparing Eq. (5) with Eq. (3), we have $P(x) = k$ and $Q(x) = kf(x)$. Substituting these into Eq. (5) yields:

$$g(x) = e^{-kx}\left(\int kf(x)e^{kx}dx + C\right) \tag{6}$$

Here, *C* is an arbitrary constant that is determined by the initial conditions. For any ambient pressure function $f(x)$, Eq. (6) represents the general solution of $g(x)$.

CONSTANT AMBIENT PRESSURE FUNCTION

Consider the function $f(x) = P_1$, where P_1 represents a constant ambient pressure to which an individual is exposed for a specified duration. Substituting $f(x) = P_1$ into Eq. (6) yields Eq. (7):

$$g(x) = P_1 + \frac{C}{e^{kx}} \tag{7}$$

When $x = 0$ s, substituting into Eq. (7) gives $g(0) = P_1 + C$, so $C = g(0) - P_1$. At this point, the saturation is 0%. Here, $g(0)$ represents the known initial tissue pressure.

As $x \to +\infty$, substituting into Eq. (7) yields $g(+\infty) = P_1$, indicating 100% saturation. Over the entire time course, $g(x)$ increases from $g(0)$ to P_1.

Let T be the time at which 50% saturation is reached, corresponding to the half-saturation time of a given theoretical tissue. Substituting $C = g(0) - P_1$ into $g(T)$ yields $k = \ln 2/T$. Substituting these into Eq. (7) yields:

$$g(x) = P_1 - \frac{P_1 - g(0)}{2^{\frac{x}{T}}} \tag{8}$$

Eq. (8) is consistent with Eq. (1), and the commonly used saturation formula in diving medicine corresponds to these two equations, as shown in Eq. (9):

$$S = (1 - 0.5^n) \times 100\% \tag{9}$$

EXPONENTIAL AMBIENT PRESSURE FUNCTION

Consider the function $f(x) = P_0 \times 2^{x/n}$, which represents an individual exposed to an initial ambient pressure P_0, where the pressure doubles every n seconds. Substituting $f(x) = P_0 \times 2^{x/n}$ into Eq. (6) yields:

$$g(x) = \frac{nkP_0}{\ln 2 + nk} \cdot 2^{\frac{x}{n}} + C \cdot e^{-kx} \tag{10}$$

Given that $g(0)$ is known, substituting $x = 0$ into Eq. (10) yields:

$$C = g(0) - \frac{nkP_0}{\ln 2 + nk} \tag{11}$$

Substituting Eq. (11) into Eq. (10) yields:

$$g(x) = \frac{nkP_0}{\ln 2 + nk}(2^{\frac{x}{n}} - e^{-kx}) + g(0)e^{-kx} \tag{12}$$

Generally, $g(0) = P_0$— this indicates that, at the moment ambient pressure begins to change, tissue pressure is already in full equilibrium with the ambient pressure. If, at that point, the individual has experienced repeated diving or multiple compression/decompression cycles in a chamber, the value of $g(0)$ should be determined based on actual conditions. Substituting $g(0) = P_0$ into Eq. (11) yields:

$$C = \frac{P_0 \ln 2}{\ln 2 + nk} \tag{13}$$

Substituting Eq. (13) into Eq. (10) yields:

$$g(x) = P_0 \frac{nk \cdot 2^{\frac{x}{n}} + \ln 2 \cdot e^{-kx}}{\ln 2 + nk} \tag{14}$$

LINEAR AMBIENT PRESSURE FUNCTION

1. Linear ambient pressure decrease

Consider the function $f(x) = P_1 - ax$, which represents an individual initially exposed to an ambient pressure P_1, with the pressure decreasing at a rate of a kPa per second, where $a > 0$. Substituting $f(x) = P_1 - ax$ into Eq. (6) yields:

$$g(x) = P_1 - \frac{a(kx-1)}{k} + C \cdot e^{-kx} \tag{15}$$

Given that $g(0)$ is known, substituting $x = 0$ s into Eq. (15) yields:

$$C = g(0) - P_1 - \frac{a}{k} \tag{16}$$

Substituting Eq. (16) into Eq. (15) yields:

$$g(x) = P_1 - \frac{a(kx-1)}{k} + \left(g(0) - P_1 - \frac{a}{k}\right)e^{-kx} \tag{17}$$

2. Linear ambient pressure increase

Consider the function $f(x) = P_0 + bx$, which represents an individual initially exposed to an ambient pressure P_0, with the pressure increasing at a rate of b kPa per second, where $b > 0$. Substituting $f(x) = P_0 + bx$ into Eq. (6) yields:

$$g(x) = P_0 + \frac{b(kx-1)}{k} + C \cdot e^{-kx} \tag{18}$$

Given that $g(0)$ is known, substituting $x = 0$ s into Eq. (18) yields:

$$C = g(0) - P_0 + \frac{b}{k} \tag{19}$$

Substituting Eq. (19) into Eq. (18) yields:

$$g(x) = P_0 + \frac{b(kx-1)}{k} + \left(g(0) - P_0 + \frac{b}{k}\right)e^{-kx} \tag{20}$$

Generally, $g(0) = P_0$. In this case, $g(x) = P_0 + b(kx-1)/k + b \times e^{-kx}/k$.

In fact, Howel, Weber and Vann et al. have already provided analytical solutions for tissue inert gas tension under linear compression and decompression conditions,[7] and the results independently derived in this study are in complete agreement with their conclusions.

The definitive mathematical model is presented in Table 1. When the breathing gas is compressed air, it is necessary to multiply by 0.79 (or approximately 0.8) when calculating tissue nitrogen tension.

Table 1
Mathematical model based on different forms of ambient pressure variation; see text for additional symbol definitions

Forms of ambient pressure variation	Ambient pressure function	Tissue pressure function (multiply by 0.79 to obtain tissue nitrogen tension)	Remarks
Constant pressure	P_1	$g(x) = P_1 - \frac{P_1 - g(0)}{2^{\frac{x}{T}}}$	$\boldsymbol{P_1}$: initial ambient pressure, generally **not** atmospheric pressure $\boldsymbol{T}$: half-saturation time of a given tissue $\boldsymbol{g(0)}$: initial tissue pressure
Exponential compression	$P_0 \times 2^{x/n}$	$g(x) = \frac{nkP_0}{\ln 2 + nk}(2^{\frac{x}{n}} - e^{-kx}) + g(0)e^{-kx}$	$\boldsymbol{n}$: pressure doubles every n seconds during the compression phase $\boldsymbol{k}$: saturation rate constant $\boldsymbol{P_0}$: initial ambient pressure, generally atmospheric pressure $\boldsymbol{g(0)}$: initial tissue pressure
Linear decompression	$P_1 - ax$	$g(x) = P_1 - \frac{a(kx-1)}{k} + \left(g(0) - P_1 - \frac{a}{k}\right)e^{-kx}$	$\boldsymbol{k}$: saturation rate constant $\boldsymbol{P_1}$: initial ambient pressure, generally **not** atmospheric pressure $\boldsymbol{g(0)}$: initial tissue pressure
Linear compression	$P_0 + bx$	$g(x) = P_0 + \frac{b(kx-1)}{k} + \left(g(0) - P_0 + \frac{b}{k}\right)e^{-kx}$	$\boldsymbol{k}$: saturation rate constant $\boldsymbol{P_0}$: initial ambient pressure, generally atmospheric pressure $\boldsymbol{g(0)}$: initial tissue pressure

CRITERION FOR MODEL EVALUATION

The piecewise approximation method, a common approach for calculating inert gas tension, converges to the exact solution as the step interval decreases. Therefore, to rigorously assess the accuracy of the proposed model, we compare its results with those obtained from the piecewise method using a step interval of 1 s. The primary metric for evaluating model performance is the level of agreement between the two methods, quantified as the maximum deviation in tissue nitrogen tension across the range of half-times considered.

Results

THEORETICAL TISSUE NITROGEN TENSIONS FROM THREE CALCULATION METHODS

The initial pressure was 100 kPa, with exponential compression following $2^{t/4}$. The target depth was 100 m, followed by a high-pressure dwell time of 4 s. Decompression was performed at a rate of 30 kPa·s^{-1}, with a nitrogen concentration of 80%. Six theoretical tissues, with half-saturation times ranging from 30 s to 20 min, were selected to calculate their surfacing nitrogen tension. During the ambient constant-pressure phase, all methods employed the classical half-saturation formula to determine nitrogen tension. For the ambient compression and decompression phases, the computational approaches differed as follows:

Conventional method: the duration of compression or decompression was converted into an equivalent exposure at a constant pressure equal to the average of the initial and final pressures for that phase.

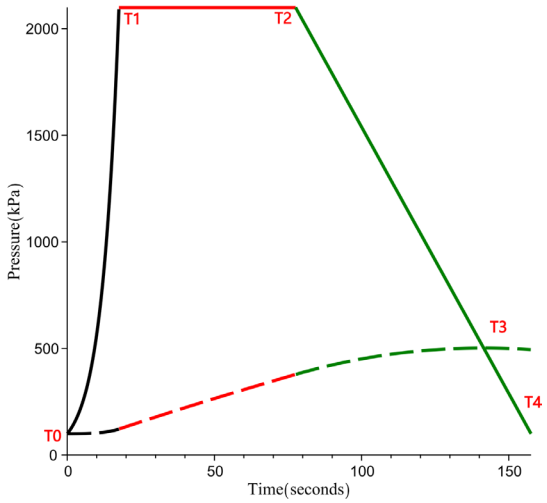
Piecewise method: the ambient compression or decompression process was divided into 1-s intervals, each treated as a constant-pressure step, with the classical half-saturation formula applied to each step.

Proposed model method: parameters were directly substituted into the mathematical model developed in this study.

Table 2
Theoretical tissue nitrogen tension values obtained by different calculation methods

Calculation method	Theoretical tissue nitrogen tension (kPa)					
	30 s	80 s	120 s	5 min	10 min	20 min
Conventional method	374	233	189	127	104	92
Piecewise method	332	218	179	123	102	91
Proposed model method	331	217	178	123	102	91

Figure 1
Variation of ambient pressure and tissue pressure over time; solid black line – ambient pressure during the compression phase; solid red line – ambient pressure during the constant-pressure phase; solid green line – ambient pressure during the decompression phase; dashed black line – tissue pressure during the compression phase; dashed red line – tissue pressure during the constant-pressure phase; dashed green line – tissue pressure during the decompression phase



The calculation results are presented in Table 2.

NITROGEN TENSION VARIATION CURVES AND THEIR CHARACTERISTICS OBTAINED BY THE PROPOSED MODEL METHOD

The initial pressure was 100 kPa, with exponential compression following $2^{t/4}$. The target depth was 200 m, followed by a high-pressure dwell time of 60 s. Decompression was performed at a rate of 25 kPa·s^{-1}. The tissue half-time was 300 s, and the nitrogen concentration was 79%. Incorporating these parameters into the mathematical model produced the curves shown in Figure 1, with the corresponding results summarised in Table 3.

At time *T*0, the ambient compression phase begins. At this moment, ambient pressure equals tissue pressure, and the rate of increase of tissue pressure is zero.

The interval from *T*0 to *T*1 is the ambient compression phase. During this phase, tissue pressure increases in a concave manner, with both ambient pressure and tissue pressure rising rapidly. However, the rate of increase of tissue pressure is always lower than that of ambient pressure, causing the pressure difference between them to gradually widen.

At time *T*1, the pressure difference reaches its maximum, and simultaneously, the rate of increase of the tissue pressure function also peaks.

The interval from *T*1 to *T*2 is the ambient constant-pressure phase: ambient pressure remains constant, while tissue pressure increases in a convex manner (with *T*1 as the inflection point), continuing to rise toward ambient pressure, but its rate of increase gradually slows.

The interval from *T*2 to *T*3 is the ambient decompression phase: ambient pressure decreases rapidly, while tissue pressure continues to rise, with the tissue pressure function remaining convex during this phase, until time *T*3.

At time *T*3, as shown in Figure 1, the ambient pressure curve intersects with the tissue pressure curve. At this point, the two pressures are equal, tissue pressure reaches its maximum, and its rate of increase drops to zero.

The interval from *T*3 to *T*4 is the ambient decompression phase: both ambient pressure and tissue pressure begin to decrease, but the rate of decrease of ambient pressure exceeds that of tissue pressure. Consequently, the pressure difference between them continues to widen, while the rate of decrease of tissue pressure gradually accelerates.

Discussion

The four types of ambient pressure functions selected in this study are all representative to some extent. When ambient pressure is constant, it indicates that the pressure remains stable over a specified period, corresponding to the constant-pressure phase. The associated tissue pressure function is consistent with the conventional saturation formula based on half-saturation time. When ambient pressure changes exponentially (typically with a base of two), it represents a rapid pressure increase, effectively

Table 3

Ambient pressure, tissue pressure, and tissue nitrogen tension at different time points; at time T0, ambient pressure begins to increase. At T1, the increase in ambient pressure stops and the constant-pressure phase begins. At T2, the constant-pressure phase ends and the ambient pressure decrease phase begins. At T3, tissue pressure and tissue nitrogen tension reach their maximum values. At T4, the ambient decompression phase ends

Category	Time point				
	0 s (*T*0)	**17.57 s (*T*1)**	**77.57 s (*T*2)**	**141.48 s (*T*3)**	**157.57 s (*T*4)**
Ambient pressure (kPa)	100.0	2100.0	2100.0	502.4	100.0
Tissue pressure (kPa)	100.0	122.4	378.4	502.4	495.0
Tissue nitrogen tension (kPa)	79.0	96.7	298.9	396.9	391.0

doubling every few seconds. This pattern is uncommon in standard diving conditions and primarily pertains to rapid compression in, for example, a submarine escape chamber. The linear function is further divided into two forms: constant-rate compression and constant-rate decompression. Constant-rate compression corresponds to a diver descending at a constant speed underwater or the pressure in a chamber increasing at a constant rate; constant-rate decompression corresponds to a diver ascending at a constant speed underwater or the pressure in a chamber decreasing at a constant rate. Collectively, these four functional forms effectively cover the typical pressure change patterns encountered in various diving scenarios.

AIR-BREATHING FAST BUOYANT ASCENT

The diving mode most suitable for the proposed model is the air-breathing fast buoyant ascent. It should be noted that fast buoyant ascent refers to a procedure in which personnel, after rapid compression in a chamber, directly enter seawater at a certain depth and ascend rapidly to the surface without any in-water decompression stops. During the ambient compression phase, personnel are in the chamber environment rather than the underwater environment; therefore, this is not a conventional diving mode. Hyperbaric exposure followed by fast buoyant ascent carries a risk of decompression sickness, with the initial bubble load considered to originate mainly from fast tissues.[8] Decompression sickness (DCS) is believed to be caused by the formation of bubbles when tissue gases become supersaturated.[9] Murphy et al. studied five novel pharmacokinetic structures with compartments coupled by perfusion and applied them to predict the probability and time of onset of decompression sickness in humans.[10] Theoretically, the incidence of decompression sickness is closely related to tissue nitrogen tension; however, during fast buoyant ascent, the tension at and after surfacing, rather than during ascent, is considered the dominant factor. Since actual tissue nitrogen tension is difficult to measure, the calculation of theoretical tissue nitrogen tension is essential. The higher the accuracy of these calculations, the more accurate the assessment of personnel safety during fast buoyant ascent from different depths. The current commonly used calculation method estimates tissue nitrogen tension upon surfacing based on the multi-tissue half-saturation theory. This method is uncontroversial for the constant-pressure phase. However, for the compression and decompression phases, where ambient pressure is not constant, the common practice is to convert these continuously changing phases into equivalent constant-pressure phases for approximate calculation. Because this treatment differs from the actual pressure variation pattern, discrepancies between calculated results and true physiological values are inevitable.

A fast buoyant ascent such as during submarine escape typically consists of three distinct phases: a compression phase, a constant-pressure phase, and a decompression phase. The compression and constant-pressure phases take place inside a chamber, where ambient pressure is fully controllable. Furthermore, the ascent rate during the decompression phase can be adjusted within certain limits through buoyancy control, thereby enabling accurate calculation of tissue nitrogen tension throughout the entire process. In practical calculations, the appropriate model is selected based on the specific characteristics of the ambient compression and decompression phases, and the final surfacing nitrogen tension is obtained through cumulative, segment-by-segment calculation.

Analysis of the data presented in Table 2 reveals several notable features when comparing the three calculation methods.

1) The difference between the conventional method and the other two approaches is most evident in fast tissues. For fast tissues with half-saturation times of 30 s and 80 s, the nitrogen tension values calculated by the conventional method are markedly higher than those obtained from the piecewise method and the proposed model. This indicates that during rapid ambient pressure changes, the conventional method overestimates the actual nitrogen tension in fast tissues because it neglects the continuity of ambient pressure variation.

2) The results obtained using the piecewise method and the proposed model are in close agreement. Across all six theoretical tissues, the values generated by these two methods are nearly identical, with a maximum difference

of only 1 kPa. This finding not only confirms the accuracy of the mathematical model developed in this study but also demonstrates that, in practical engineering applications or calculations, when the step interval is sufficiently small, the piecewise approximation method yields results sufficiently close to those of the exact model, offering practical utility.

3) When the half-saturation time reaches 5 min or more, the differences among the three methods are minimal, with a maximum difference not exceeding 1 kPa. This is because slow tissues are less sensitive to dynamic pressure changes, so the errors introduced by simplification are diluted.

In summary, the conventional method overestimates inert gas tension in fast tissues, rendering it unsuitable for approximate calculation in practical applications. The proposed method more accurately characterises the dynamics of tissue inert gas tension under continuous ambient pressure variations and exhibits strong concordance with the piecewise approximation method, thereby confirming its reliability and practical utility. It should be noted that in the piecewise method, reducing the time interval improves the accuracy of the results but increases computational complexity. When the step interval approaches zero, the outcomes converge with those obtained from the proposed model. Furthermore, as shown in Figure 1, the maximum nitrogen tension occurs at time $T3$ rather than at $T4$ (surfacing); during the interval from $T3$ to $T4$, the tissue is already undergoing decompression, indicating that surfacing nitrogen tension is not the maximum. In contrast, the conventional method assumes that the tissue remains in a compression state throughout the entire process.

CONVENTIONAL DIVING

A conventional diving process typically consists of one or more descent (compression) phases, one or more bottom (constant-pressure) phases, and multiple ascent phases. During ascent, the diver's movement between decompression stops constitutes the decompression phase, while the stationary periods at these stops constitute the constant-pressure phase.

The proposed method enables accurate and convenient calculation of tissue inert gas tension during compression and decompression phases. A conventional air diving decompression schedule is used as an example for illustration: a diver descends from the surface to a depth of 50 m at a constant rate over 3 min, and then remains at that depth for 13 min of bottom work. The diver breathes compressed air, with a nitrogen percentage of 80%. A conventional air diving process generally consists of a descent phase, a bottom phase, multiple travel phases, and staged decompression phases. In the conventional method, the formula for the constant-pressure phase is directly applied to calculate tissue nitrogen tension during the descent and bottom phases, yielding nitrogen tensions of 216 kPa and 437 kPa for 5 min tissue at 3 min (end of descent) and 16 min (end of bottom phase), respectively. In contrast, using the linear compression mathematical model, the nitrogen tension of the same tissue at 3 min is 153 kPa, which is only 70.8% of the value obtained by the conventional method. During the subsequent constant-pressure phase (3 min to 16 min), the calculation of nitrogen tension is the same as that in the conventional method, giving a value of 426 kPa at 16 min, approximately 97.5% of the conventional method's result. The difference between the two methods at the end of the constant-pressure phase is small, mainly because the constant-pressure phase gradually reduces the deviation accumulated in the previous phase — the longer the constant-pressure phase, the closer the results of the two methods become.

Taking the safe coefficient of supersaturation as 1.6, the first decompression stop depth calculated by the conventional method is 17.2 m, while that obtained using the linear compression model is 16.6 m. After rounding, both methods give a first decompression stop depth of 18 m. It should be noted that during ascent, except for the first phase, the remaining ascent phases are often neglected in conventional calculations because of their short duration, making it impossible to optimise these phases using the proposed model. In summary, the principal advantage of the proposed model lies in its ability to accurately calculate tissue inert gas tension during ambient compression and decompression phases. In conventional diving applications, however, divers spend most of their time in the ambient constant-pressure phase, and the compression and decompression phases account for a relatively small proportion of the total time, resulting in no significant overall difference in the final decompression schedule. Moreover, the calculation principles of decompression schedules are inherently conservative, with depths and durations being rounded up by default when determining decompression stops, which further reduces the practical difference between the two methods.

Conclusions

The mathematical model, developed on the premise that the diffusion rate of a gas is proportional to the pressure gradient, provides an accurate and convenient calculation method that addresses the limitations in calculating inert gas tension during ambient compression and decompression phases. It offers a theoretical framework for the dynamic prediction of tissue inert gas tension across a range of diving scenarios. The exponential model, in particular, provides a significant improvement in the fidelity of the model to the likely physiological state. Compared with the conventional exponential response function employed during constant-pressure phases, the accuracy of this method improves with an increased proportion of ambient compression and decompression phases, as well as with shorter tissue half-saturation times.

References

1 Boycott AE, Damant GC, Haldane JS. The prevention of compressed-air illness. J Hyg (Lond). 1908;8:342–443. doi: 10.1017/s0022172400003399. PMID: 20474365. PMCID: PMC2167126.

2 Behnke AR. The Harry G. Armstrong Lecture. Decompression sickness: advances and interpretations. Aerosp Med. 1971;42:255–67. PMID: 4926522.

3 Bühlmann AA, Frei P, Keller H. Saturation and desaturation with N2 and He at 4 atm. J Appl Physiol. 1967;23:458–62. doi: 10.1152/jappl.1967.23.4.458. PMID: 6053671.

4 Wienke BR. Equivalent multi-tissue and thermodynamic decompression algorithms. Int J Biomed Comput. 1989;24:227–45. doi: 10.1016/0020-7101(89)90019-6. PMID: 2606565.

5 Thalmann ED. Testing of decompression algorithms for use in the U.S. Navy underwater decompression computer. Research Report NEDU TR 1-84. Washington DC: Navy Experimental Diving Unit; 1984.

6 Workman RD. Calculation of decompression schedules for nitrogen--oxygen and helium- oxygen dives. Research Report NEDU TR 6-65. Washington DC: Navy Experimental Diving Unit; 1965.

7 Howle LE, Weber PW, Vann RD. A computationally advantageous system for fitting probabilistic decompression models to empirical data. Comput Biol Med. 2009;39:1117–29. doi: 10.1016/j.compbiomed.2009.09.006. PMID: 19853847.

8 Gennser M, Loveman G, Seddon F, Thacker J, Blogg SL. Oxygen and carbogen breathing following simulated submarine escape. Undersea Hyperb Med. 2014;41:387–92. PMID: 25558547.

9 Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. Lancet. 2011;377(9760):153–64. doi: 10.1016/S0140-6736(10)61085-9. PMID: 21215883.

10 Murphy FG, Hada EA, Doolette DJ, Howle LE. Probabilistic pharmacokinetic models of decompression sickness in humans, part 1: Coupled perfusion-limited compartments. Comput Biol Med. 2017;86:55–64. doi: 10.1016/j.compbiomed.2017.04.014. PMID: 28505552.

Conflicts of interest and funding: nil

Submitted: 4 April 2026
Accepted after revision: 6 July 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

A feasibility study of combined hyperbaric oxygen treatment and a lifestyle medicine program for multimorbid conditions

Elizabeth Elliott[1], Sam Manger[2], John Mercer[3], John Stevens[4]

[1] *Department of Diving and Hyperbaric Medicine, Royal Hobart Hospital, Hobart, Tasmania, Australia*
[2] *James Cook University College of Medicine and Dentistry, Douglas, Queensland, Australia*
[3] *Allied Health Metabolic Clinic, Tasmania Health Service, Launceston, Tasmania, Australia*
[4] *Mullumbimby, New South Wales, Australia*

Corresponding author: *Dr Elizabeth Elliott, Department of Diving and Hyperbaric Medicine, Royal Hobart Hospital, Liverpool St, Hobart, Tasmania 7000, Australia*
ORCiD: *0009-0005-3679-621X*
elizabeth.elliott@ths.tas.gov.au

Keywords
Education; Health; Hyperbaric research; Medical conditions and problems; Questionnaire

Abstract
(Elliott E, Manger S, Mercer J, Stevens J. A feasibility study of combined hyperbaric oxygen treatment and a lifestyle medicine program for multimorbid conditions. Diving and Hyperbaric Medicine. 2026 30 September;56(3):245–256. doi: 10.28920/dhm56.3.245-256. PMID: 42743563.)

Introduction: Multimorbidity and polypharmacy are highly prevalent among patients receiving hyperbaric oxygen treatment (HBOT). Lifestyle Medicine (LM) interventions improve health outcomes in chronic disease management, but their implementation in tertiary hospital settings remains poorly studied and not at all in hyperbaric therapy. We evaluated the feasibility and acceptability amongst patients and staff of delivering a Lifestyle Medicine Program (LMP) within a small hyperbaric medicine department.

Methods: A six-week, open-label, feasibility study was conducted at the Department of Diving and Hyperbaric Medicine, Royal Hobart Hospital. The LMP comprised weekly group education sessions on the six pillars of LM, underpinned by motivational interviewing, and supported by newsletters. Acceptability (participant satisfaction ratings, recommendation rates, perceived value) and feasibility (enrolment, session attendance, retention, engagement, resource use, cost analysis, program delivery, and safety) were assessed via mixed methods data analysis on participant and staff feedback through questionnaires and focus groups.

Results: Twelve patients were recruited via rolling recruitment; 11 completed post-program questionnaires. Participants perceived the LMP as acceptable, safe, and valuable, with 11/11 recommending the program and 10 reporting it beneficial. Qualitative themes from short answers and focus groups indicated that the group-based education fostered social connection and self-agency, though challenges in accessibility, program framing, and therapeutic alliance with HBOT highlighted areas for improvement to enhance feasibility and integration within clinical care. Seventeen staff completed their questionnaire, indicating that the LMP was perceived as beneficial, valuable, and achievable with the hyperbaric setting, provided that minor logistical and structural modifications are made.

Conclusions: Delivering a LMP alongside HBOT is feasible in a tertiary hospital setting, with potential to enhance patient wellbeing and complement clinical care. Larger, multi-site trials are needed to evaluate scalability, inclusivity, and long-term feasibility outcomes.

Introduction

Multimorbid conditions affect 38% of Australians (9.2 million people in 2022) and are defined by the presence of two or more chronic illnesses.[1,2] Polypharmacy, the use of more than five medications, is common among patients with multimorbidity.[3,4] These conditions disproportionately affect individuals with lower social determinants of health, advanced age, frailty, impaired development, or cognition, comorbid mental health conditions fragmented medical care, and institutionalisation.[1,2,4]

Hyperbaric medicine is accessible in every state capital of Australia (plus Darwin and Townsville), is primarily utilised for hypoxic tissue repair and healing, including diabetic foot wounds and soft tissue radiation injury.[5] The Department of Diving and Hyperbaric Medicine at the Royal Hobart Hospital services the population of Tasmania, approximately 576,000 people, providing upward of 2,500 hyperbaric treatments annually.[6] From the principal investigator's (PI's) clinical experience, patients undergoing hyperbaric oxygen treatment (HBOT) typically present with multimorbidity and polypharmacy. HBOT is an acceptable and well tolerated therapy for patients.[7–10]

Table 1
The six pillars of Lifestyle Medicine

Pillar	Description
Nutrition	Emphasises whole, real food, minimally processed dietary patterns tailored to culture and context of the person, with limiting ultra-processed foods and excess added sugar.
Physical activity	Regular, enjoyable movement and structured exercise to improve cardiovascular, metabolic and musculoskeletal health, thereby reducing sedentary behaviour, and support functional capacity.
Sleep	Addressing sleep quantity and quality with assessment and management of sleep timing, maintenance and behaviours as restorative sleep supports metabolic, cognitive and emotional health.
Stress management	Structured approaches to reduce adverse psychological and physiological effects of negative stress integrated into person-centred care.
Addressing risky behaviours/ substances	Identification and reduction/elimination of tobacco, harmful alcohol consumption and other risky substance use and behaviours, including gambling, screen usage, etc.
Social connection	Generating and nurturing meaningful personal and social relationships and community engagement (including cultural/community supports).

Lifestyle medicine (LM) is an emerging discipline shown to improve health parameters for both prevention and management of chronic conditions, with benefits extending to patient wellbeing and health equity, deprescribing, health system sustainability, and healthcare provider satisfaction.[4,11–21]

LM incorporates environmental, motivational, and behaviour-change principles delivered with structured interventions and evidence-based clinical applications of healthy lifestyle behaviours to prevent, treat, and often reverse chronic disease alongside conventional medical care.[13,15] The healthy lifestyle behaviours are often referenced as the 'six pillars' of LM, including nutrition, physical activity, sleep, stress management, social connection, and avoidance of risky substances and behaviours (Table 1).[22] These six pillars are applied to the individual according to their biopsychosocial, cultural and spiritual needs.

While LM has been extensively studied in chronic disease disorders where "*the goal is to establish LM as a bedrock and foundation of primary care*", there is a paucity of evidence exploring its implementation in tertiary or hospital-based contexts.[21,23,24] No published research has examined the integration of lifestyle medicine programs (LMPs) into hyperbaric departments, despite their patient populations having a high degree of multimorbidity and therefore theoretically well-suited for such interventions. This research and practice gap limits the ability to extend LM approaches into specialised hospital environments.

This unique hospital setting and treatment course provides an opportunity for an education-based LM approach that can be embedded into standard HBOT care with the prospect of minimal interruption to services, staff, patients, or budget. Establishing determinants of feasibility (acceptability, demand, implementation, practicality, integration and limited-efficacy testing) for this study through feedback from participants and staff utilising a rigorous mixed methods approach will provide a template to generate enhanced insights to inform future implementation trials.[25] This work aligns with the 2021–2030 National Preventative Health Strategy, using lifestyle improvements as an affordable and scalable approach to chronic disease management.[26]

AIMS

The primary aim for the study was to determine the feasibility of delivering a LMP to patients and staff in the Department of Diving and Hyperbaric Medicine, Royal Hobart Hospital. This included assessing participant acceptability, satisfaction, perceived value, and engagement (recruitment, retention, questionnaire completion, resource use, and session attendance), staff acceptability, and perspectives on workload and integration, and the practicality of inserting education sessions on the six pillars of LM, underpinned by motivational interviewing, into routine hyperbaric services (*Supplement 1).[25] The study assessed outcome measures from participants and staff, and recruitment strategies; evaluated the impact on service delivery; and supported development for future pilot trials for implementing LM into tertiary care contexts.[27]

***Footnote:** Supplement 1 is available online on our website: https://www.dhmjournal.com/index.php/journals?id=435

This study was informed by current knowledge and application of LM, implementation science, patient-centred care, and motivational interviewing. The predicted benefits were multi-layered. For patients, the LMP may improve recovery, quality of life, and self-management skills. With staff, the LMP offers tools and strategies to enhance wellbeing while providing holistic patient care, potentially reducing burnout. Regarding hospitals, the integration of LM supports innovation, aligns with broader health system goals (preventative healthcare, decreased disease burden, improved staff wellbeing), and may reduce long-term healthcare costs.

Methods

ETHICS

Dual ethics approval was obtained through the Tasmanian Low Risk Human Research Ethics Committee (2024/ETH02748) as the location for the study was at the Royal Hobart Hospital (RHH), and James Cook University Human Resource Ethics Committee (25H-0092) as the principal investigator's (PI's) Master's qualification was administered by the university. Governance was approved by the RHH Governance Committee.

STUDY DESIGN

This pilot program was conducted as a prospective, open label, non-controlled, non-randomised, small-scale clinical study, implemented over six weeks delivered alongside HBOT to evaluate operationalising a LMP for participants and staff. The six weeks aligned with the six pillars of LM topics, and an extra seventh week facilitated focus group attendance. A single LMP was provided to capture initial feasibility determinants. Data were sourced from a convenience sample of participants and staff using purpose-designed questionnaires, verbal commentary, and formalised focus groups. Quantitative and qualitative approaches were combined to capture measurable outcomes of feasibility (acceptability, demand, implementation, practicality, adaptation, integration and limited-efficacy testing) through metrics of enrolment, session attendance, retention, engagement, resource use, program delivery, participant satisfaction ratings, recommendation rates, perceived value, and safety to generate insights into suitability from participants and staff.[28] As a feasibility study, the sample size was not powered to detect intervention effectiveness. Quantitative outcomes are therefore reported descriptively, with percentages used to summarise feasibility rather than to support inferential or causal conclusions. The study framework was guided by LM principles, behaviour change theory (motivational interviewing), and implementation science concepts of feasibility.

The LMP was provided as a weekly group education and interactive discussion (six sessions, twice weekly, 30–40 minutes each) delivered in the department sub-waiting room prior to HBOT morning and afternoon chamber runs. LMP sessions were conducted by three HBOT staff, the PI (author EE) as the LM educator, a senior nurse/registrar as facilitator, and a nurse/registrar as scribe. Extra staffing was not required. The sessions were incorporated into patient care with the intent to minimally interrupt or inconvenience the running of the hyperbaric chamber or the participants' day (adding on an extra 20 minutes to patient contact time outside of standard care). Patients typically present 20–30 minutes prior to their daily HBOT to allow for changing out of street clothes into hyperbaric approved clothing, check in with nursing and medical staff to determine daily fitness for HBOT, and other individualised interventions (blood sugar level measurement, wound dressing change, etc).

Six weekly topics covered the six pillars of LM (nutrition, physical activity, enhancing sleep, decreasing stress, limiting addictive behaviours, and maintaining community connectedness), with delivery incorporating motivational interviewing techniques. Session notes captured by the scribe were summarised with supporting documentation by the PI into a weekly newsletter (printed or emailed to participants) to reinforce content and support accountability, building a LMP library.

PARTICIPANTS AND RECRUITMENT

Eligible participants were invited to attend the LMP via an opportunistic rolling recruitment prior to and during the study supported by poster advertisement in the department. Patients commenced and completed their hyperbaric treatment at different times during the trial, depending on timing of HBOT assessment, HBOT prescription (20–40 week-daily treatments), patient availability, hyperbaric chamber occupancy, and urgency of treatment. The individualised staggered start and cessation of HBOT for patients and their recruitment to the LMP was expected to impact on attendance rates (which was not a reflection of retention), therefore, attendance of the full program was unlikely. Given the chamber capacity (maximum six patients in the multiplace chamber and one patient in the monoplace chamber across two daily runs) and anticipated opt-out rates (~25%), sample size was also expected to be small during the six-week LMP feasibility study. Medical staff obtained written consent from participants on patient information and consent forms prior to engagement in the LMP with the option to withdraw from the study at any stage during the program without impacting their HBOT.

Eligibility criteria for the intervention included any patient commencing HBOT during the study period (six weeks) as many patients being referred for HBOT have multimorbid conditions. Exclusion criteria included age < 18 years, inability to consent (intubated patients), and a past or current history of eating and/or addiction disorders (as per low-risk ethics requirements). All attendees were from English speaking backgrounds.

QUESTIONNAIRE AND FOCUS GROUPS

All participants were asked to complete a feasibility questionnaire (responses were on a five-point Likert scale and short answers) following their completion of the program, regardless of number of sessions attended (*Appendix 1). A sample of 12 participants was targeted with a threshold for questionnaire completion, recruitment and retention set at 80% to support validation of the primary aim.[29] The participants who completed at least three of the six LMP sessions were deemed 'completers' of the program and were invited to the focus groups in week seven. This low attendance number criteria for completers anticipated the potential for attrition of participants during the program to ensure there would be eligible participants for the focus groups. Questionnaires and focus group questions were purpose-designed for this feasibility study, with the participant questionnaire adapted from the Food For Thought Questionnaire, 2015, provided through the Tasmanian Health Service's Northern Integrated Care Service (*Appendix 1&3).[30] Survey items were tailored to the pilot objectives of feasibility as there was no established questionnaire available for this specific context (Appendix 1).

Open-ended interview questions in the two 25-minute focus groups conducted by the PI sought to determine the participants' opinions as to which specific aspects of the LMP made it feasible, and what could be done for future iterations of the LMP (*Appendix 3). The focus groups were conducted 30 minutes prior to HBOT in week seven, to facilitate participant attendance. The focus group conversations were recorded by a Smartphone program and deleted following artificial intelligence transcription with the PI editing the transcription verbatim. The edited transcript was reviewed by co-author, JM. All results from questionnaires and focus groups were deidentified and recorded in the Department of Health Research Electronic Data Capture (REDCap) as per ethics requirements.

STAFF AND RECRUITMENT

Department of Diving and Hyperbaric Medicine staff fulfilled the roles in the LMP sessions including the LM educator (PI and hyperbaric consultant), facilitator (senior nurse/registrar), and scribe (nurse/registrar). The PI held dual roles as the LM educator and researcher as this was their capstone project to achieve a Masters in Lifestyle Medicine. Staff were proactively recruited by the PI depending on availability, providing their verbal consent with their roles clearly outlined. All department staff involved directly (provision or support of the LMP) or indirectly (supervision of HBOT) with the LMP were asked to complete the staff purpose-designed questionnaire (five-point Likert scale and short answers) at completion of the LMP to obtain a broader scope of interdepartmental acceptance of the LMP (*Appendix 2). Staff HBOT employment (doctor, nurse, technician, administration) varied from < 1 year to > 30 years, with no prior LM experience. A threshold of 80% was prospectively set for staff feasibility measured as acceptability, alignment of the LMP with the Organisation's values, and future recommendation of patient participation by staff determined through the questionnaire (*Appendix 2).

DATA ANALYSIS

Feasibility was evaluated through quantitative metrics such as recruitment and retention of participants, and satisfaction and recommendation rates, analysed descriptively and reported as percentages.[28] Qualitative responses (short answers and focus group transcripts) employed inductive thematic analysis, guided by Braun and Clarke's six-phase framework.[31] The PI immersed themselves in the raw data from participant short answers and focus groups, and the staff short answers, seeking a critical appraisal of the dataset. This helped initial data coding and theme development as they emerged from the raw data through a process of highlighting text and determining codes by repeated comments and salient points from respondents.[32] These codes were then analysed to find broader patterns of commonality to support evolution of themes to describe what emerged out of the dataset. Themes were further refined using reflexive journalling and a thematic map.[32] The final thematic analysis involved evolution of the themes and subthemes to ensure capture of nuanced perspectives provided by the responders, supported by illustrative quotes. As the themes for the participant short answers and focus groups were well aligned, they were combined for the qualitative assessment. This process implemented reflexivity throughout the six phases, which was then reviewed by co-author JM for validity of participant and staff feedback into the thematic constructs to restrict PI bias.[32,33] This mixed-methods approach attempted to capture both measurable feasibility outcomes and rich, contextual insights into acceptability through convergent parallel analysis for the small sample size. Questionnaires and focus group questions could be further refined in designing future pilot trials to help validate the assessment tools.

COST ANALYSIS

Cost of implementing a LMP was provided as 'time in kind' with staff, expertise, and resources all sourced within the department and provided alongside standard patient care. The PI allocated two hours per week to prepare and conduct the LMP sessions, document, and electronically distribute the weekly newsletter to support consolidation of information from the group education and discussion session. Newsletters were formulated from key learning objectives for each pillar and conversational points from participants supported by factual information that arose during the education and discussion session. The educational

***Footnote:** Appendices 1–3 are available online on our website: https://www.dhmjournal.com/index.php/journals?id=436

framework and information guide based on the six pillars of LM was formulated as part of the ethics application. The framework, guides, and newsletters established a LMP library for future use. Staff involvement was within normal working hours (for up to 40 minutes per session, twice weekly), with no significant increase in duties and minimal preparation required for the program. The total additional hours allocated to the LMP for all staff involved in the program was approximately 5.5 hours per week during normal working hours (divided among the PI, facilitator, and scribe). Participants might have been out-of-pocket for a small increase in vehicle parking fees. No extra funding was sought for the program.

Results

RECRUITMENT AND RETENTION

Fourteen patients were eligible for the LMP alongside their HBOT during the recruitment period with 12 participants consented to the program, achieving the goal of 12 participants for this small feasibility study (recruitment was > 80%) (Figure 1). The two other eligible patients declined consent to be involved in the LMP. No-one withdrew their consent for participation during the program (retention was successful at > 80%).[29] LMP attendance was 100% despite varied HBOT commencement and cessation dates amongst participants (Table 2). One participant did not complete the questionnaire due to unplanned cessation of HBOT and was unable to be contacted. Their data are not included in the results. Six participants attended three or more sessions ('completers') during their HBOT, with two completing the full six sessions. Four out of six 'completers' attended the two focus groups held prior to HBOT facilitated by the PI. One 'completer' was unavailable due to work commitments and the other was travelling interstate. Ten participants were receiving HBOT for latent effects of soft tissue radiation (predominantly radiation cystitis), and two had complications of a diabetic foot ulcer (both conditions typically receive 40 HBOT sessions). Seventeen staff completed questionnaires, ten medical and nursing staff directly involved in the provision of the LMP (59%) and seven medical, nursing, technical and administrative staff not directly involved in the program (41%) but had regular participant contact during the LMP (Table 3).

QUANTITATIVE OUTCOMES

Participants

Of the 11 respondents (out of 12 consented participants), seven were male (64%, 75–83 years old) and four were female (36%, 64–69 years old). All resided in socioeconomically advantaged areas (Socio-Economic Indexes for Areas quintiles 4–5).[34] Acceptability was high (11/11, 100%), with 10 participants (91%) finding the LMP beneficial, nine (82%) reporting improved understanding of health behaviours, and 10 (91%) valued the newsletters (Table 4). In fact, all participants requested newsletters for topics that they had missed the education sessions for both prospectively and retrospectively. While all sessions were rated as relevant, application of knowledge to daily life varied (Table 4). However, four participants (36%) reported feeling unsupported by the facilitators, and two participants (18%) felt unsupported and uncomfortable contributing to the education sessions (Table 4). Three participants (27%) also felt they didn't receive enough information prior to joining the LMP (Table 4). Yet, eight participants (73%) expressed

Figure 1
Overview of participants and attendance

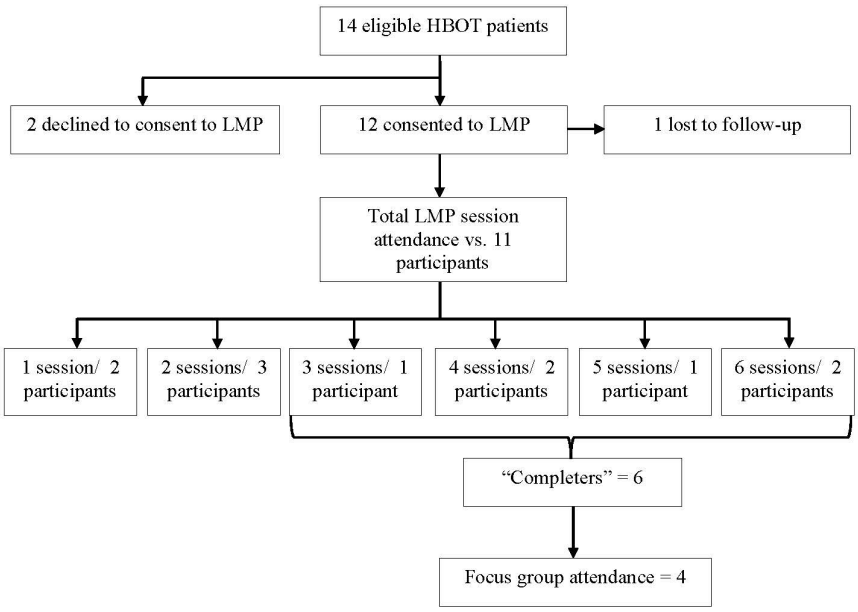


Table 2
Characteristics of participant attendance

Week	Topic	Participant attendance
1	Nutrition	6
2	Physical activity	8
3	Sleep	7
4	Stress management	5
5	Addressing addiction	5
6	Enhancing community	5

Table 3
Characteristics of staff in the lifestyle medicine program (LMP)

Occupation	*n* (%)
Doctor	4 (23%)
Nurse	9 (53%)
Technician	3 (18%)
Administration	1 (6%)
Total	17
Involvement in the LMP	
Yes	10 (59%)
No	7 (41%)

interest in further programs, with three participants (27%) neutral (Table 5). All participants would recommend the LMP (Table 4).

Staff

Staff also rated the LMP highly with 17/17 (100%) reporting acceptability and alignment with the organisations' values and mission (Table 5). The utility of the LMP effectively addressing patient needs was also high, which was reflected in the likelihood of staff recommending the program to patients (Table 5).

QUALITATIVE OUTCOMES

Participants

Coding participant verbal feedback and triangulation with short answer responses via thematic analysis resulted in five overarching themes with subthemes (Table 6). Participants generally valued the LMP integrated into their hyperbaric treatment, finding the information, social support and connection as beneficial for their health and wellbeing (Table 6). However, they did struggle with the perceived connection of the LMP to hyperbaric therapy, highlighting the need for clarity with program framing and communication (Table 6). The social connection and shared experiences emerged as core components to the success of the LMP (Table 6). Reflexively, the PI's positive priming during the focus groups may have shaped participant responses, shifting the conversation toward affirmation as opposed to critique. Thematically, this analysis provides recommendations for improved transparency, structure, and therapeutic alliance with implementing a LMP within hospital-based care.

Staff

Thematic analysis for staff short answers from the questionnaire revealed four themes with descriptors regarding their perspective of the implementation of the LMP into the Department of Diving and Hyperbaric Medicine (Table 7). From a reflexive perspective, the staff data analysis indicated that the LMP was well-received and feasible with workflow, identifying strong participant engagement and staff enthusiasm as enablers, while barriers included participant readiness, scheduling and access constraints, with suggestions for improved timing, pre-intervention information, and multidisciplinary input to strengthen future implementation (Table 7). The inductive interpretation of the dataset captures the intrinsic alignment between LM's principles (prevention, empowerment, behaviour change) and the department's culture of patient-centred, holistic care.[11,14]

Discussion

These results suggest that integrating a LMP into standard HBOT care was feasible for participants and staff within this small and intimate hyperbaric setting. Feasibility metrics of acceptability, demand, implementation, practicality, integration and limited-efficacy testing were achieved through high attendance and the absence of withdrawals, plus participant and staff appreciation for LMP structure and resources (particularly newsletters). Both groups also reported strong satisfaction with the LMP and willingness to recommend the program. The need to provide adequate explanation for the intent and purpose of the LMP with onboarding, clearer framing of the LMP-HBOT relationship and checking in during the discussions to ensure understanding and clarity were identified by both participants and staff. This could reduce the risk of participants feeling unsupported and uncomfortable during the group sessions, allowing for a more supportive, sharing environment (Table 4). Future studies could incorporate participant assessment of readiness for behavioural change to strengthen study design and success. Both parties provided support and recommendations for future implementation of the program in the department.

As the first study of its kind, this feasibility study addresses a gap in the literature and aligns with national health priorities. Regular HBOT attendance appeared to facilitate program delivery, while the mixed-methods design enabled a broader understanding of both quantitative viability information and qualitative suitability insights from participants and staff. While the PI was the only staff member trained in LM, there

Table 4
Results of the participant questionnaire; LMP – lifestyle medicine program

Question	*n* (%)					
	Strongly agree	**Agree**	**Neutral**	**Disagree**	**Strongly disagree**	**No response**
Overall, I had benefit from the LMP	4 (36%)	6 (55%)	–	–	–	1 (9%)
Overall, the LMP was acceptable	3 (27%)	8 (73%)	–	–	–	–
I have a better understanding of how I can improve my health and wellbeing	5 (46%)	4 (36%)	2 (18%)	–	–	–
I found the weekly structure and content of the LMP to be helpful	2 (18%)	7 (64%)	–	–	–	2 (18%)
I found the weekly newsletter/readings/resources to be helpful	4 (36%)	6 (55%)	–	–	–	1 (9%)
I found the setting in which the LMP was provided to be appropriate	5 (45%)	5 (45%)	–	–	–	1 (10%)
I felt that I didn't receive enough information about the program and my role in it before starting the LMP	2 (18%)	1 (9%)	1 (9%)	6 (55%)	–	1 (9%)
I have no regrets about taking part in the LMP	7 (64%)	4 (36%)	–	–	–	–
I felt unsupported and uncomfortable sharing my views and experiences during the education and discussion sessions in the LMP	–	2 (18%)	2 (18%)	4 (36%)	3 (28%)	–
I felt unsupported by facilitators	1 (9%)	3 (27%)	–	4 (36%)	3 (28%)	–
I would recommend the LMP to other people	4 (36%)	7 (64%)	–	–	–	–
If given the opportunity, I would do more LMPs	3 (27%)	5 (46%)	3 (27%)	–	–	–
I have made/am making change in my daily life because of what I learned in the LMP	3 (27%)	5 (46%)	2 (18%)	1 (9%)	–	–
I found the nutritional content to be helpful and relevant (6/11 participants)	2 (33%)	3 (50%)	1 (17%)	–	–	–
I found the physical activity content to be helpful and relevant (8/11 participants)	3 (38%)	5 (62%)	–	–	–	–
I found the stress management content to be helpful and relevant (5/11 participants)	1 (20%)	4 (80%)	–	–	–	–
I found the sleep optimisation content to be helpful and relevant (7/11 participants)	3 (43%)	4 (57%)	–	–	–	–
I found the addressing addictive behaviours content to be helpful and relevant (5/11 participants)	1 (20%)	2 (40%)	2 (40%)	–	–	–
I found the enhancing community connectedness content to be helpful and relevant (5/11 participants)	2 (40%)	2 (40%)	–	–	–	1 (20%)

are educational opportunities in the form of an e-learning module and workshops (< 60 minutes duration) provided by the Tasmanian Health Service on the Six Pillars of Wellbeing designed to enhance staff knowledge and expertise within the LM realm. Support of staff with accessible and affordable inhouse training could promote multiplication of sustainable leadership with provision of LM education and discussion sessions.

The combination of education sessions and newsletters appeared to offer a practical, low-cost intervention model that can be adapted across clinical settings. Health-literate newsletters could be further developed for pre- and post-session resources, translated for diverse cultural and language backgrounds, and supplemented with digital tools. This could further enhance reinforcement of learning aims and accountability for participants and support staff

Table 5
Results of the staff questionnaire; LMP – lifestyle medicine programme

Question	*n* (%)				
	Very acceptable/ Aligned/ Effective/ Likely	**Acceptable/ Aligned/ Effective/ Likely**	**Neutral**	**Unacceptable/ Misaligned/ Ineffective/ Unlikely**	**Very unacceptable/ Misaligned/ Ineffective/ Unlikely**
Rate your overall acceptability of the LMP	12 (71%)	5 (29%)	–	–	–
Does the LMP align with our organisations values and mission?	16 (94%)	1 (6%)	–	–	–
How effective do you think the programme is in addressing patient needs?	9 (52%)	8 (48%)	–	–	–
How likely are you to recommend the programme to patients?	13 (76%)	4 (24%)	–	–	–

familiarisation of education topics. Further clarification of optimising patient access to such resources needs to be explored in future research, particularly considering the existing high workload on healthcare professionals.

Future research could also consider a hybrid model tailored to patient access and needs for virtual (e.g. video vignettes, online resources) and face-to-face integration of education. The model needs to align with participant and staff recommendations to preserve interpersonal benefits and motivational interviewing from face-to-face sessions. This can assist with addressing issues regarding the setting, patient transport, and staffing fluctuations around departmental demands.

LIMITATIONS

There were several limitations with this study. The small sample size and single-site design limit transferability and generalisability of the findings and restrict the ability to draw causal inferences. Health characteristics beyond their HBOT indication of the participants were not obtained due to ethics restrictions, limiting applicability of the LMP to patients with multimorbidity and polypharmacy. Percentages reported should be interpreted with caution due to low absolute numbers. While opportunistic rolling recruitment provided pragmatic implementation and created variability in participant exposure to the full program, this may have introduced selection bias and limited diversity within the sample.

The consent process could also be improved with pre-information patient resources (brochure, website). The short duration of the study and lack of follow-up also limits the assessment of longer-term implementation sustainability, scalability and real-world impact.

All participants were English speaking and resided in areas of relative socioeconomic advantage resulting in lack of heterogenicity of participants and restricting interpretation of results to disadvantaged groups.[34] This substantially limits applicability of this study, particularly as LM interventions aim to address inequities among disadvantaged populations who have the greatest burden of multimorbidity and constitute the greatest proportion of the population in Tasmania.[34] This generalisability gap in the recruitment impacts on the interpretation of feasibility of the study amongst all patients referred for HBOT, further highlighting equity and health literacy deficits in the provision of healthcare nationally.

Potential researcher bias in data collection and interpretation is another consideration, as the PI held dual roles as the LM educator and researcher. This may have influenced participant engagement and interpretation of qualitative data despite efforts to minimise bias irrespective of peer review. Feedback from staff, while valuable, may have been influenced by departmental culture as opposed to organisational or universal feasibility. There is also acknowledgement for professional alignment with the PI for staff responses. The small size of the department, intimate social relationship between staff, participants, and the PI, and the level of socioeconomic advantage of the cohort could have also lent itself to social desirability bias. Efforts were made to minimise bias through recruitment strategies, structured facilitation, use of independent questionnaires, and reflexive, systematic thematic analysis with co-author oversight. As the PI was the only trained LM practitioner in the department this is also a limitation in that there is a risk of lack of quality control and continuity for the provision of LMP across staff involved in the LMP.

This feasibility study could be further expanded into a pilot trial to determine and further elaborate on feasibility metrics with implementing a LMP into standard HBOT care. The limitations could also be addressed with enrolling more participants, providing multiple iterations of the program, creating multi-site designs, extended trial period, expansion on delegation of roles including triangulation, independent facilitation of qualitative data collection, targeted strategies

Table 6
Thematic analysis of participant short answers and focus groups; FG – focus group; HBOT – hyperbaric oxygen treatment; SA – short answer

Themes	Subthemes	Illustrative quotes
Social connection and peer support with learning	Social sharing	*"It breaks down those barriers a little bit for chatting around the subjects."* (Female, FG) *"Everyone seemed engaged and happy to share."* (Female, SA)
	Lived shared experience	*"It gets you through to talk about your condition... connection helped me through mine"* (Male, FG) *"People build such good relationships because you've got the time and you've got that shared experience."* (Male, FG)
	Therapeutic relationships	*"Lifestyle, nutrition for prevention of disease would be a really good thing for doctors to be able to teach patients."* (Female, short answer) *"The friendly, convivial attitude of staff is infectious."* (Male, SA)
Interpersonal and social aspects of learning	Delivery preferences	*"Excellent presentations of invaluable information."* (Female, SA) *"In those group sessions, there's always someone who wants to talk."* (Male, FG) *"I received helpful newsletters on some of the sessions I missed."* (Female, SA)
	Enhanced wellbeing and coping	*"Before I started, there was a lot of pain…there's no pain anymore."* (Male, FG) *"There's been lots of health concerns….it helped me to cope with that."* (Male, FG)
Participant engagement and knowledge development	Empowerment	*"I'm reading up on it now to see what else I can do."* (Male, FG)
	Self-directed learning	*"Manageable goals/ways of implementing."* (Female, SA)
	Information load	*"I tuned out for quite a bit of it."* (Male, FG) *"Very, very foreign topics."* (Male, FG)
	Educational accessibility	*"Sessions informative and some very helpful information shared… helpful newsletters."* (Female, SA) *"Advantageous to have your summarise given to us first."* (Male, FG)
Perceived relevance and integration	Variable usefulness	*"Better clarification about what was involved in the program."* (Male, FG) *"I found it a tad difficult."* (Male, FG) *"Presentations were aimed at the correct level."* (Female, SA)
	Relevance with HBOT	*"I don't know what connection there is with hyperbaric."* (Male, FG)
	Personalisation of information	*"I think that sort of information is good for everyone… a general holistic approach."* (Female, FG) *"Because that list that you showed me, that was of interest. I didn't know I was committing to a six-week session."* (Male, FG)
Program delivery, timing and logistical considerations	Multimodal delivery	*"The printed information sent by email is invaluable, because you have time to absorb it."* (Male, SA) *"Face to Face is better for discussions."* (Female, SA)
	Timing	*"Timing worked out pretty well… that was fortunate."* (Male, FG) *"It would be good to know what days this was going to happen so you can make arrangements."* (Female, FG)
	Accessibility barriers	*"I couldn't hear it properly."* (Male, FG) *"That works if you're driving, that doesn't work so much if you're relying on other people for transport."* (Female, SA)

Table 7
Thematic analysis of staff short answers; HBOT – hyperbaric oxygen

Themes	Subthemes	Illustrative quotes
Departmental workflow and logistical implications	Service flow Scheduling	*Didn't interrupt the flow of the unit.*" (Technician) *"Early timing incompatible with some, especially for community transport"* (Doctor)
	Adequate staffing	*"Timing can be tight for nurses in organising other patients."* (Nurse)
	Spatial configuration	*"Doing the sessions in a difference space from the waiting area to psychologically separate the program from 'waiting for HBOT'."* (Doctor)
Patient preparedness and readiness for behaviour change	Targeted recruitment	*"Potential patients could be identified and sent information brochures prior to assessment."* (Doctor)
	Variable readiness	*"One or two patients would not consider… not willing to change"* (Nurse) *"Some patients pre-contemplative."* (Doctor) *"Patients very, very engaging"* (Technician) *"The program was received very well from our patients."* (Administration)
Communication and education resources	Effective communication	*"Receiving the weekly newsletter with the resources that are sent to the participants." (Nurse)* *"Perhaps a brochure if it does become permanent"* (Doctor)
	Staff awareness	*"So staff can keep abreast of what is being discussed with patients and learn too!"* (Nurse)
	Access to materials	*"A website patients can visit."* (Technician)
Future implementation and integration	Staff optimism	*"I really enjoyed participating."* (Nurse) *"A great program, well received by patients and staff alike."* (Technician)
	Continuation	*"I'd be happy to be involved with this again."* (Nurse) *"Hoping it continues"* (Nurse)
	Multidisciplinary inclusion	*"Maybe input from other specialties, e.g. nutritionists, physiotherapists."* (Nurse)

to engage socioeconomically disadvantaged participants, and staff education in the field of LM. Outcomes can target inclusivity, validation of assessment tools (to measure pre-program population profile against post-program content retention/learnings), patient-reported outcomes in the short and long-term, staff satisfaction, sustained behaviour change, and cost-effectiveness to inform scalability of LMPs in clinical care.

Conclusions

This feasibility study demonstrated that integrating a LMP alongside HBOT can be feasible, where resources are available for English speaking and socioeconomically advantaged patients and staff in a small hyperbaric department in a tertiary hospital setting potentially improving preventative health behaviours, wellbeing, and patient and staff satisfaction. Education sessions supported by newsletters provided a practical, potentially low-cost, and well-received model that fostered collegiality, enhanced coping strategies, and promoted self-agency. Staff recognised the program's alignment with organisational values and its potential to complement HBOT without disrupting service delivery. Limitations, including small sample size, single-site design, and participant socioeconomic homogeneity, restricted generalisability. Future research could build on these findings through a pilot trial with a larger sample size conducted over a longer duration to address equity gaps, optimise integration into standard patient care and resource delivery, and evaluate longer-term outcomes such as behaviour change, patient-reported health outcomes (including morbidity and mortality metrics), and cost-effectiveness. This study provides a foundation for extending LM into hospital-based care, with relevance for chronic disease management across hyperbaric and other tertiary settings.

References

1 Aramrat C, Choksomngram Y, Jiraporncharoen W, Wiwatkunupakarn N, Pinyopornpanish K, Carson Mallinson PA, et al. Advancing multimorbidity management in primary care: A narrative review. Prim Health Care Res Dev. 2022; 23:e36. doi: 10.1017/S1463423622000238. PMID: 35775363. PMCID: PMC9309754.

2 Australian Institute of Health and Welfare. Multimorbidity. AIHW, Australian Government. 2024. [cited 2024 Oct 23]. Available from: https://www.aihw.gov.au/reports/australias-health/multimorbidity.

3 Masnoon N, Shakib S, Kalisch-Ellett L, Caughey GE. What is polypharmacy? A systematic review of definitions. BMC Geriatr. 2017;17:230. doi: 10.1186/s12877-017-0621-2. PMID: 29017448. PMCID: PMC5635569.

4 Srivastava SB. Polypharmacy, unintended consequences, and impact of lifestyle medicine. Am J Lifestyle Med. 2023;18:54–7. doi: 10.1177/15598276231207302. PMID: 39184275. PMCID: PMC11339771.

5 Sethuraman KN, Smolin R, Henry S. Is there a place for hyperbaric oxygen therapy? Adv Surg. 2022;56:169–204. doi: 10.1016/j.yasu.2022.02.011. PMID: 36096567.

6 Australian Bureau of Statistics. National, state and territory population. Canberra: ABS; 2025. [cited 2026 Jan 14]. Available from: https://www.abs.gov.au/statistics/people/population/national-state-and-territory-population/latest-release.

7 Simman R, Mihani A, Michalak J, Heyboer M. Pearls in hyperbaric oxygen therapy: a challenging case series. Wounds. 2022;34(9):E78–E84. doi: 10.25270/wnds/21049. PMID: 36252269.

8 MacInnes L, Baines C, Bishop A, Ford K. Patient knowledge and experience of hyperbaric oxygen treatment. Diving Hyperb Med. 2021;51:72–7. doi: 10.28920/dhm51.1.72-77. PMID: 33761544. PMCID: PMC8313768.

9 Teguh DN, Bol Raap R, Koole A, Knippenberg B, Smit C, Oomen J, van Hulst RA. Hyperbaric oxygen therapy for nonhealing wounds: Treatment results of a single center. Wound Repair Regen. 2021;29:254–60. doi: 10.1111/wrr.12884. PMID: 33377598. PMCID: PMC7986203.

10 Howell RS, Criscitelli T, Woods JS, Gillette BM, Gorenstein S. Hyperbaric oxygen therapy: Indications, contraindications, and use at a tertiary care center. AORN J. 2018;107:442–53. doi: 10.1002/aorn.12097.

11 Babich JS, McMacken M, Correa L, Polito-Moller K, Chen K, Adams E, et al. Advancing lifestyle medicine in New York City's public health care system. Mayo Clin Proc Innov Qual Outcomes. 2024;8:279–92. doi: 10.1016/j.mayocpiqo.2024.01.005. PMID: 38828080. PMCID: PMC11141270.

12 Bodai BI, Nakata TE, Wong WT, Clark DR, Lawenda S, Tsou C, et al. Lifestyle medicine: A brief review of its dramatic impact on health and survival. Perm J. 2018;22:17–25. doi: 10.7812/TPP/17-025. PMID: 29035175. PMCID: PMC5638636.

13 Egger GJ, Binns AF, Rossner SR. The emergence of "lifestyle medicine" as a structured approach for management of chronic disease. Med J Aust. 2009;190:143–5. doi: 10.5694/j.1326-5377.2009.tb02317.x. PMID: 19203313.

14 Egger G, Egger S. Lifestyle medicine: The Australian experience. Am J Lifestyle Med. 2012;6:26–30. doi.org/10.1177/1559827611405287.

15 Egger G. Lifestyle medicine: The 'why', 'what', and 'how' of a developing discipline. Aust J Gen Pract. 2019;48;665–8. doi: 10.31128/AJGP-06-19-4955. PMID: 31569327.

16 Fields D, Arnold M, Karlsen M, Kelly J. A lifestyle medicine approach to medication deprescribing: An introduction. J Fam Pract. 2022;71(1):E100–4. doi: 10.12788/jfp.0259. PMID: 35389855.

17 Merlo G, Rippe J. Physician burnout: A lifestyle medicine perspective. Am J Lifestyle Med. 2021;15:148–57. doi: 10.1177/1559827620980420. PMID: 33790702. PMCID: PMC7958216.

18 Patel P, Madrid M. Successfully integrating lifestyle medicine into health systems. Am J Lifestyle Med. 2021;15:510–13. doi: 10.1177/15598276211008397. PMID: 34646099. PMCID: PMC8504335.

19 Petersen MR, Freeman AM, Madrid M, Aggarwal M. Strategies for incorporating lifestyle medicine in everyday hospital practice. Am J Lifestyle Med. 2021;15:531–7. doi: 10.1177/15598276211006664. PMID: 34646102. PMCID: PMC8504330.

20 Sun X, Feng X, Hu S. Lifestyle medicine in China: The Fuwai Hospital lifestyle medicine initiative. Am J Lifestyle Med. 2025;20(1):15598276251371687. doi: 10.1177/15598276251371687. PMID: 40895502. PMCID: PMC12394202.

21 Yeoh EK, Wond MCS, Wong ELY, Yam C, Poon CM, Chung RY, et al. Benefits and limitations of implementing chronic care model (CCM) in primary care programs: A systematic review. Int J Cardiol. 2018;258:279–88. doi: 10.1016/j.ijcard.2017.11.057. PMID: 29544944.

22 Egger G, Binns A, Rössner S, Sagner M, editors. Lifestyle medicine: lifestyle, the environment and preventive medicine in health and disease, 3rd ed. London: Academic Press; 2017.

23 Lippman D, Stump M, Veazey E, Tanigawa Guimarães S, Rosenfeld R, Kelly JH, et al. Foundations of lifestyle medicine and its evolution. Mayo Clin Proc Innov Qual Outcomes. 2024;8:97–111. doi: 10.1016/j.mayocpiqo.2023.11.004. PMID: 38304165. PMCID: PMC10831813.

24 Kamarajah SK, Lampridou S, Soysa ND, Glasbey JC, Nepogodiev D, Blackwell S, et al. Implementation of hospital-initiated complex interventions for adult people with multiple long-term conditions: a scoping review. BMC Health Serv Res. 2025;25:290. doi: 10.1186/s12913-025-12424-2. PMID: 39980012. PMCID: PMC11843763.

25 Bowen DJ, Kreuter M, Spring B, Cofta-Woerpel L, Linnan L, Weiner D, et al. How we design feasibility studies. Am J Prev Med. 2009;36:452–7. doi: 10.1016/j.amepre.2009.02.002. PMID: 19362699. PMCID: PMC2859314.

26 Australian Government, Department of Health. National preventive health strategy 2021–2030. 2021. [cited 2024 Nov 9]. Available from: https://www.health.gov.au/sites/default/files/documents/2021/12/national-preventive-health-strategy-2021-2030_1.pdf.

27 Eldridge SM, Chan CL, Campbell MJ, Bond CM, Hopewell S, Thabane L, et al. on behalf of the PAFS consensus group. CONSORT 2010 statement: extension to randomised pilot and feasibility trials. BMJ. 2016;355:i5239. doi: 10.1136/bmj.i5239. PMID: 27777223. PMCID: PMC5076380.

28 Aschbrenner KA, Kruse G, Gallo JJ, Plano Clark VL. Applying mixed methods to pilot feasibility studies to inform intervention trials. Pilot Feasibility Stud. 2022;8(1):217. doi: 10.1186/s40814-022-01178-x. PMID: 36163045. PMCID: PMC9511762.

29 Montgomery R. Sample size justification in feasibility studies:

moving beyond published guidance. Pilot Feasibility Stud. 2025;11(1):88. doi: 10.1186/s40814-025-01675-9. PMID: 40551266. PMCID: PMC12186404.

30 Ogden K, Woodroffe J, Mercer J. 'Food for thought': Implementation and development of a new interdisciplinary clinical pathway for obesity management in Tasmania. A demonstration project of the vTAHSP 2014–2015. University of Tasmania, Tasmania. 2015 (Final report – Government Research).

31 Braun V, Clarke V. Using thematic analysis in psychology. Qual Res Psychol. 2006;3:77–101. doi: 10.1191/1478088706qp063oa.

32 Ahmed SK, Mohammed RA, Nashwan AJ, Ibrahim RH, Abdalla AQ, Ameen BMM, et al. Using thematic analysis in qualitative research. J Med Surg Public Health. 2025;6:100198. doi: 10.1016/j.glmedi.2025.100198.

33 Ogden K, Barr J, Rossetto G, Mercer J. A "messy ball of wool": a qualitative study of the dimensions of the lived experience of obesity. BMC Psychol. 2020;8(1):67. doi: 10.1186/s40359-020-00416-2. PMID: 32586401. PMCID: PMC7318440.

34 Australian Bureau of Statistics (ABS). Socio-economic advantage and disadvantage. Cat. No. 2071.0 – Reflecting Australia: Stories from the Census, 2016. 2017. [cited 2025 Jul 27]. Available from: https://www.abs.gov.au/ausstats/abs@.nsf/Lookup/by%20Subject/2071.0~2016~Main%20Features~Socio-Economic%20Advantage%20and%20Disadvantage~123#:~:text=SEIFA%20quintiles%20are%20based%20on,.gov.au/census.

Acknowledgements

Deep gratitude and thanks to the patients and staff of the Department of Diving and Hyperbaric Medicine, Hobart, who were involved in and supported the study.

Conflicts of interest and funding: nil

Submitted: 14 February 2026
Accepted after revision: 6 July 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

No difference in core temperature or thermal sensation in male and female scuba divers using wetsuits or semi-drysuits in 22.6°C sea water

Fabian N Möller[1,2,3], Kavi Dodge[1], Tucker Orman[2], Igor Glavičić[4], Željko Dujić[5], Nikola Foretić[6], Andrew T Lovering[1]

[1] *University of Oregon, Department of Human Physiology, Eugene, OR, USA*
[2] *Massachusetts Institute of Technology, Department of Aeronautics and Astronautics, Aerospace Physiology Lab, Boston, MA, USA*
[3] *German Sport University, Cologne, Institute of Professional Sport Education and Sport Qualification, Cologne, Germany*
[4] *Department of Marine Studies, University of Split, Croatia*
[5] *Department of Integrative Physiology, University of Split School of Medicine, Split, Croatia*
[6] *Faculty of Kinesiology, University of Split, Split, Croatia*

Corresponding author: *Professor Andrew Lovering, Department of Human Physiology, 1240 University of Oregon, Eugene OR, 97403-1240, USA*
ORCiD: *0000-0001-7826-4290*
lovering@uoregon.edu

Keywords
Dive suit; Equipment; Immersion; Safety; Scuba diving; Thermal protection

Abstract
(Möller FN, Dodge K, Orman T, Glavičić I, Dujić Z, Foretić N, Lovering AT. No difference in core temperature or thermal sensation in male and female scuba divers using wetsuits or semi-drysuits in 22.6°C sea water. Diving and Hyperbaric Medicine. 2026 30 September;56(3):257–264. doi: 10.28920/dhm56.3.257-264. PMID: 42743564.)
Introduction: Scuba diving in water below thermoneutral temperature (< 30°C) can reduce core temperature and impair comfort and performance. Semi-drysuits are presumed superior to wetsuits because they presumably limit water entry into the suits, but changes in core temperature and thermal sensation have not been previously published in males and females wearing semi-drysuits. This study assessed the influence of anthropometrics and thermal protection from wetsuits and semi-drysuits on core temperature and thermal sensation in males and females performing recreational scuba dives.
Methods: Core temperature (telemetric pill) and thermal sensation (Young's scale) were measured in 39 recreational divers (17 females) wearing wetsuits (mean 6 [SD 1] mm thickness, n = 19, 6 females) or semi-drysuits (7 [1] mm, n = 20, 11 females) during a 45-minute dive in mean 22.6 (1.1)°C water.
Results: The wetsuit group had approximately 2 mm thinner suits ($P < 0.001$) and a 0.2 m² greater body surface area ($P = 0.035$), while age, body mass index, body fat percentage, and water temperature did not differ between groups. No significant differences were observed between suit types for absolute core temperature change or thermal sensation. In semi-drysuit divers, body fat percentage negatively correlated with the change in temperature per minute of dive time (rate of temperature change, $R^2 = 0.29$, $P = 0.014$). However, when anthropometric values were multiplied by suit thickness, there were no significant correlations with the rate of temperature change in either suit type.
Conclusions: Semi-drysuits did not confer superior thermal protection in 22.6°C water during recreational scuba diving activities.

Introduction

Decreases in body temperature from cold air or water exposure in professional, occupational, and/or recreational settings lead to physiological adaptations and symptoms ranging from discomfort and shivering to decreased physical and psychological performance.[1] The severity of possible symptoms depends on the air and/or water temperature, thermal protection, and time of exposure, as well as on body composition and physiological reactions for heat loss prevention that include peripheral vasoconstriction and shivering or non-shivering thermogenesis.[2]

With scuba diving, individuals choose to wear various types of diving suits for thermal protection. Conductive heat loss is increased in water compared to air, depending on the temperature difference between the skin and the surrounding medium, the material's thickness and thermal conductivity, and the medium's conductance.[3] Furthermore, high body surface area to mass ratios tend to create the greatest decreases in core temperature in cold environments due to the greater surface area for heat conduction,[4,5] and it has been suggested that skin temperature and the locations of greatest heat loss might differ between sex assigned at birth.[6] Acknowledging the relationships of these factors with the chosen suit type might improve thermal protection

during cold water immersion. However, current operational exposure criteria in scuba diving and cold-water immersion research generally define limits in terms of physiologic responses (e.g., acceptable changes in core temperature) and do not formally incorporate diver anthropometrics, suit type, or sex into the definition of thermal exposure limits.[7] While the type of suit may play a role in preventing decreases in body core temperature and thermal sensation, the studies performed thus far have focused on wetsuits and drysuits under various conditions in studies with mostly male subjects that were within a narrow range of demographic parameters like age, height, and body composition,[8–10] and under extreme conditions that are not likely for the majority of recreational scuba divers.[11] Previous work from our group reported temperature changes in recreational divers using wetsuits[12] and drysuits.[13] Still, no data exist on the impacts of wetsuits versus semi-drysuits on changes in core temperature and thermal sensation in male and female recreational scuba divers.

When considering suit types, thermal protection in wetsuits is primarily achieved by limiting convective heat loss through minimising water circulation between the skin and the suit by a snug fit. Nevertheless, during a dive, some degree of intermittent water exchange with the ambient environment can occur, particularly in wetsuits without an ideal fit, which can lead to repeated bouts of cooler ambient water entering the suit and increased heat loss. Conversely, suits commonly referred to as 'semi-dry' are designed to further restrict water inflow and outflow compared with conventional wetsuits through close-fitting smooth-skin neoprene seals at the wrists, ankles, and neck, typically combined with a watertight back- or front-entry zipper, sealed seams, and, in some models, an attached or integrated hood, rather than through fundamentally different insulation principles. Both suit types use closed-cell neoprene of comparable construction, so the dominant insulation mechanism, the reduction of conductive and convective heat loss via the trapped gas in the neoprene matrix, is shared between designs. As a result, and assuming an ideal fit, semi-dry suits may reduce the magnitude of water exchange with the environment and thereby attenuate temperature fluctuations inside the suit following the initial heat conduction after water entry, as minimal as it may be. To date, a search on PubMed for "*semi-drysuits*" and "*dive*" (with or without hyphen, plural or singular) yields just one publication that examined the utility of using heating pads in seven male divers wearing semi-drysuits in 7.2°C water.[14] Accordingly, there are currently no data-based recommendations for choosing semi-drysuits over wetsuits for scuba diving in any water temperature, particularly with respect to the inclusion of both male and female participants.

The purpose of this study was to measure core temperature and thermal sensation before and after a ~45-minute scuba dive in ~22.6°C water temperature in males and females scuba divers using either semi-drysuits or wetsuits Furthermore, we sought to quantify individual anthropometrics and suit thickness on the rate of change in core temperature per minute of dive time. It was hypothesised that the (I) decrease in core temperature and thermal sensation would be less with semi-drysuits than wetsuits, and (II) that a higher body mass index, higher percentage of body fat, and a lower body surface area to mass ratio would be associated with a reduced change in core temperature per minute of dive time.

Methods

This study was approved by the University of Oregon's Office for Protection of Human Subjects (RB# 00000951), the University of Split (#2181205020523020), and the German Sport University Cologne (108/2023). All subjects had English language proficiency, were provided with documents outlining the study in English, and gave written approval prior to participating in the study. All experimental procedures were conducted in accordance with the Declaration of Helsinki, except for registration of this study.

The study included 39 divers with dives times set for 45 minutes. Data were collected in September 2023 in Roses, Spain, and Bol, Croatia. Twenty subjects used semi-drysuits that allowed only minimal water exchange with ambient water, and 19 subjects used wetsuits without seals at the neck, arms, or legs. All participants were healthy, active, and without any self-reported cardiovascular or pulmonary disease.

All dives were conducted under optimal visual and water conditions without any exhausting tasks and using open-circuit apparatus with air as breathing gas. No further restrictions were placed on the dive profile. Maximum depth, mean depth, water temperature, and dive time were assessed at 1-second intervals by individual dive computers. Tank pressure was recorded before and after each dive, and total air consumed reported normalised to 1 bar ambient pressure. Temperature was assessed directly before water ingress and after egress by a single measurement using a telemetric pill (Jonah core body temperature capsule; Mini Mitter, Bend, OR) and by statement of thermal sensation on a scale from 0 (unbearably cold) to 8 (unbearably hot).[15]

The changes in core temperature ($\Delta Tc \cdot min^{-1}$) and thermal sensation ($\Delta Ts \cdot min^{-1}$) were normalised for total dive time in minutes to acknowledge the entire exposure duration impact on these variables and allow future comparisons to various dive durations.

Thermal protection was categorised as either wetsuits or semi-drysuits and total suit thickness was stated as the maximum thickness in millimetres around the core from all suit layers combined. All wetsuits and ~10% of the semi-drysuits were the personal property of the divers, while the other 90% of the semi-drysuits were provided by a university diving group.

Body mass index (BMI) was assessed indirectly as a calculation of body mass (BM) in kg divided by height $(m)^2$, and body surface area (BSA) by the du Bois formula where BSA (m^2) = 0.007184 (x height$^{0.725}$) x (weight$^{0.425}$).

Regression analyses were calculated as $\Delta Tc \cdot min^{-1}$ with the anthropometric variables BM, BMI, BSA, BSA/mass, and suit thickness. Additional analyses were calculated with anthropometric variables normalised for suit thickness.

An estimation of physical exertion during the dive, ventilation ($\dot{V}_E$, $L \cdot min^{-1}$) was calculated as total air consumption × dive time^{-1} × $P_{Mean\ depth}^{-1}$, with total air consumption derived from the dive tank pressure delta multiplied by the tank volume ($\Delta P_{tank} \times V_{tank}$), like before.[16]

The percentage of body fat (BF%) was calculated using [(1.39 x BMI) + (0.16 x age) - (10.34 x sex) - 9],[17] where males are depicted by '1' and females by '0', respectively.

Differences in all anthropometric and descriptive data between sex and suit types were analysed using Student's *t*-tests. Repeated measures ANOVAs were calculated for thermal sensation (Ts) and core temperature (Tc) on the factors time (pre-dive vs. post-dive) and suit type (semi-drysuit vs. wetsuit), based on post-hoc power analyses using G*Power on $\Delta Tc \cdot min^{-1}$ (Effect size: 0.51, $\alpha = 0.05$, Power: 0.99) and $\Delta Ts \cdot min^{-1}$ (Effect size: 0.51, $\alpha = 0.05$, Power: 0.72). Sex differences were not investigated due to insufficient statistical power.

The relationships between the rate of change in Tc ($\Delta Tc \cdot min^{-1}$) and wetsuit thickness (mm), anthropometric factors, including, body mass (kg), BSA (m^2), BMI, %BF and BSA to mass ratio (BSA/BM), were analysed using linear regressions. The potential effect of ventilation per minute ($\dot{V}_E$, $L \cdot min^{-1}$), as an indicator of physical activity and metabolic demand, on $\Delta Tc \cdot min^{-1}$ was investigated using linear regressions.

Statistical significance was accepted at $P < 0.05$. All data are presented as mean (SD). All analyses were completed in GraphPad Prism 10.2.0 (GraphPad Software, La Jolla, CA).

Results

Out of the 39 divers that participated in the study, 22 were males and 17 females, aged 27 (10) years. Twenty subjects (11 females) used semi-drysuits and 19 subjects (six females) used wetsuits. All dives were no-decompression dives conducted between 09:30 and 10:30 in the morning or between 15:30 and 16:30 in the afternoon. The core temperature (Tc) pill was ingested 6–10 hours before the start of the dive. All wetsuit dives were conducted within 18 days at a measured water temperature of 22.8 (1.2)°C, while semi-drysuit dives were conducted over eight days at a water temperature of 22.3 (0.9)°C. Water temperatures were not significantly different between suit types. For all participants combined, the dive time was 45 (7) min ($P = 0.468$ between groups), with a depth of 10.4 (3.2) metres of seawater (msw) ($P = 0.999$ between groups) and a water temperature of 22.5 (1.0)°C ($P = 0.168$ between groups). The thermocline ranged between 24 (1.5)°C on the surface and 21 (1.8)°C at maximum depth (Table 1).

Males and females were represented in both suit type groups (semi-drysuit vs. wetsuit). The two suit type groups differed only in suit thickness, hood thickness, and BSA. While BSA was calculated and is presented using the DuBois equation, repeating the between-group comparison with the Mosteller and Haycock equations yielded virtually identical estimates, $\Delta = 0.158$ and 0.165 m^2, respectively, vs. 0.145 m^2 for DuBois, with t = 2.15–2.17 and $P = 0.038$–0.040 in all three cases; pairwise $r \geq 0.99$. Wetsuits and hoods were both approximately 2 mm thinner than in those wearing semi-drysuits ($P < 0.001$). Ancillary protective equipment (e.g., hoods and handwear) varied between individuals; however, no group differences were observed except for greater hood thickness in the semi-dry suit group. BSA was 0.2 m^2 higher in the wetsuit group than in the semi-drysuit group ($P = 0.035$). Dive suit groups were not different in age ($P = 0.119$), BMI ($P = 0.119$), body fat percentage ($P = 0.390$), or water temperature ($P = 0.157$). $\dot{V}_E$ was not significantly different between the two groups ($P = 0.169$). That did not change when normalising ventilation for body weight ($P = 0.999$). Please refer to Table 1 for full anthropometrics, dive data and thermal protection characteristics.

All participants had a significantly reduced post-dive core temperature (Tc) (Δ -0.4 (0.5)°C; $P < 0.001$) and thermal sensation (Ts) (Δ -1.0 (0.7); $P < 0.001$) with no effect of suit type (wetsuit: Δ -0.4 (0.6)°C Tc, Δ -1.1 (0.7) a.u. Ts; semi-drysuit: Δ -0.4 (0.4)°C Tc, Δ -0.9 (0.7) a.u. Ts) (Table 1).

The rate of core temperature change per minute was not different between suit types ($\Delta Tc \cdot min^{-1}$) ($P = 0.873$; wetsuits: $\Delta Tc \cdot min^{-1}$ = -0.008°C; semi-drysuits: $\Delta Tc \cdot min^{-1}$ = -0.008°C; Figure 1A). Nor was there a difference in change of thermal sensation change per minute ($\Delta Ts \cdot min^{-1}$) ($P = 0.509$; wetsuits: $\Delta Ts \cdot min^{-1}$ = -0.024 arbitrary units (a.u.); semi-drysuits: $\Delta Ts \cdot min^{-1}$ = -0.021 a.u.; Figure 1B).

No significant associations were observed for either ΔTs $a.u. \cdot min^{-1}$ or $\Delta Tc \cdot min^{-1}$ with BF% (Figure 2A+B), BM (Figure 2C+D), BMI (Figure 2E+F), or BSA divided by BM (Figure 2G+H) when normalised for suit thickness (Figure 2) to quantify the combined thermal protection effects of anthropometrics and suit characteristics (all $r < 0.180$; $P > 0.062$).

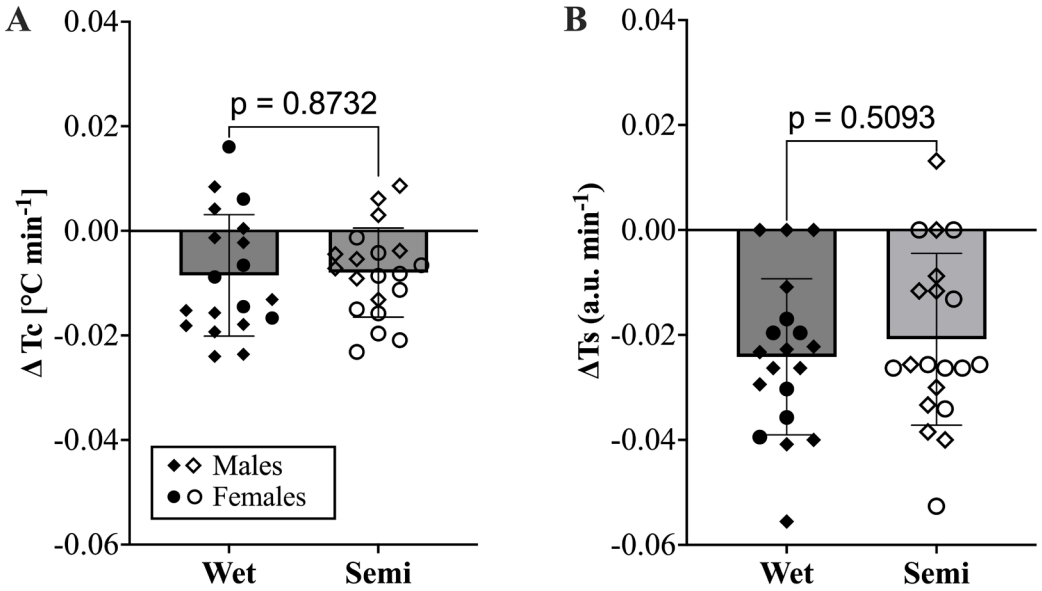
When not normalised for suit thickness, $\Delta Tc \cdot min^{-1}$ was significantly and negatively correlated to the BF% in the semi-drysuit group, with higher BF% increasing the rate of temperature loss ($P = 0.014$, $R^2 = 0.292$). In the wetsuit group, body fat percentage was positively correlated with $\Delta Tc \cdot min^{-1}$ though this association was not significant.

Table 1

Anthropometric data, body mass (BM), body surface area (BSA), body mass index (BMI), dive data, and specifics about thermal protection from all participants, separated by wetsuit and semi-drysuit, and males and females; data are presented as mean (standard deviation). Hoods, gloves, and boots are indicated with the number of participants who wore them and mean (SD) thickness (*n*; mm). Temperatures are displayed as pre-dive, post-dive, and delta values for core temperature and the statement of perceived thermal sensation on a scale from 0 (unbearably cold) to 8 (unbearably hot).[14] Significant differences between the suit type groups are depicted with *, and pre- to post-dive comparisons are indicated by #

Variable		Wetsuit, *n* = 19			Semi-dry suit, *n* = 20		
		All (19)	Women (6)	Men (13)	All (20)	Women (11)	Men (9)
		Anthropometric data					
Age, years		29.8 (13.2)	21.2 (3.0)	33.8 (14.3)	24.8 (4.9)	23.5 (2.1)	26.4 (6.7)
Height, cm		177.7 (8.3)	169.8 (5.2)	181.4 (6.9)	174.0 (8.5)	169.1 (6.7)	180.0 (6.5)
BM, kg		82.3 (22.0)	64.5 (12.9)	90.6 (20.5)	70.6 (10.7)	64.0 (5.3)	78.7 (10.2)
BSA, m^2		1.99 (0.24) *	1.74 (0.17)	2.10 (0.17)	1.84 (0.16) *	1.73 (0.09)	1.98 (0.13)
BMI, $kg \cdot m^{-2}$		26.0 (7.1)	22.3 (3.9)	27.8 (7.7)	23.3 (2.8)	22.4 (2.1)	24.3 (3.3)
BSA/BM, $m^2 \cdot kg^{-1}$		0.025 (0.003)	0.027 (0.003)	0.024 (0.003)	0.026 (0.002)	0.027 (0.001)	0.025 (0.002)
Body fat, %		24.9 (9.8)	25.3 (5.2)	24.7 (11.5)	22.7 (5.4)	26.0 (2.9)	18.7 (5.2)
		Dive data					
Water temperature, °C		22.8 (1.2)	23.6 (0.7)	22.5 (1.2)	22.3 (0.9)	22.0 (1.0)	22.7 (0.5)
Depth, m		10.3 (1.2)	9.1 (0.0)	10.8 (3.2)	10.3 (3.2)	10.9 (3.8)	9.5 (2.0)
Dive time, min		46.4 (7.7)	48.0 (10.3)	45.6 (6.4)	44.6 (7.2)	42.7 (7.7)	46.7 (6.4)
Air consumed, L		1824 (403)	1420 (334)	2011 (279)	1591 (339)	1435 (274)	1782 (325)
Ventilation, $L \cdot min^{-1}$		21 (8)	16 (5)	23 (8)	18 (4)	17 (4)	20 (3)
		Thermal protection					
Suit thickness, mm		5.8 (1.3) *	5.7 (1.2)	5.9 (1.3)	7.4 (0.8) *	7.5 (0.7)	7.2 (1.0)
Thickness/BSA, $mm \cdot m^{-2}$		3.0 (0.8)	3.3 (1.1)	2.8 (0.7)	4.0 (0.6)	4.4 (0.4)	3.7 (0.5)
Thickness/BMI, $mm \cdot kg^{-1} \cdot m^{-2}$		0.3 (0.1)	0.3 (0.1)	0.2 (0.1)	0.3 (0.1)	0.3 (0.1)	0.3 (0.1)
Hoods, *n*; mm		16; 4.0 (1.7) *	5; 4.0 (1.7)	11; 4.4 (1.5)	17; 6.3 (1.2) *	10; 6.6 (0.9)	7; 5.9 (1.6)
Gloves, *n*; mm		3; 3.5 (2.1)	N/A	3; 3.5 (2.1)	7; 4.0 (1.1)	4; 3.7 (1.2)	3; 4.3 (1.2)
Boots, *n*; mm		9; 4.9 (1.5)	1; 5.0 (-)	8; 4.9 (1.4)	19; 5.1 (1.2)	10; 5.2 (1.1)	9; 5.1 (1.4)
		Measured and perceived temperature changes					
Core temperature °C	Pre	37.5 (0.3)	37.7 (0.5)	37.5 (0.6)	37.6 (0.3)	37.7 (0.1)	37.5 (0.5)
	Post	37.1 (0.7)	37.5 (1.0)	36.9 (0.6)	37.3 (0.3)	37.2 (0.4)	37.4 (0.3)
	Δ	-0.4 (0.6) #	-0.2 (0.7)	-0.5 (0.5)	-0.4 (0.4) #	-0.5 (0.4)	-0.2 (0.4)
Perceived temperature scale	Pre	4.9 (0.8)	5.1 (0.7)	4.9 (0.9)	5.0 (0.5)	5.1 (0.3)	4.9 (0.7)
	Post	3.8 (0.6)	3.8 (0.4)	3.8 (0.7)	4.1 (0.6)	4.2 (0.7)	3.9 (0.2)
	Δ	-1.1 (0.7) #	-1.3 (0.4)	-1.0 (0.8)	-0.9 (0.7) #	-0.9 (0.8)	-1.0 (0.7)

Figure 1
Changes per minute of dive time in A: Core temperature ($\Delta Tc \cdot min^{-1}$) and B: Thermal sensation ($\Delta Ts \cdot min^{-1}$); males (diamonds) and females (circles) are depicted with full symbols for wetsuits (Wet) and empty symbols for semi-drysuits (Semi); a.u. – arbitrary units



Linear regressions of $\dot{V}_E$ and $\Delta Tc \cdot min^{-1}$ were not significant for females wearing wetsuits ($P = 0.503$, $R^2 = 0.119$) or semi-drysuits ($P = 0.542$, $R^2 = 0.043$) or males wearing wetsuits ($P = 0.071$, $R^2 = 0.266$) or semi-drysuits ($P = 0.946$, $R^2 < 0.001$).

Discussion

All participants combined experienced a significant decrease in core temperature (-0.4 [0.5]°C) and thermal sensation (-1.0 [0.7]) over the 45-minute dives in 22.6 (1.1)°C water temperature. Participants wearing wetsuits had a greater BSA than the semi-drysuit participants (2.1 m^2 vs. 1.8 m^2), and thermal protection that was 1.7 mm thinner. Under the study conditions, no differences were observed between wetsuits or semi-drysuits to maintain body core temperature within established US Navy exposure limits of 1°C.

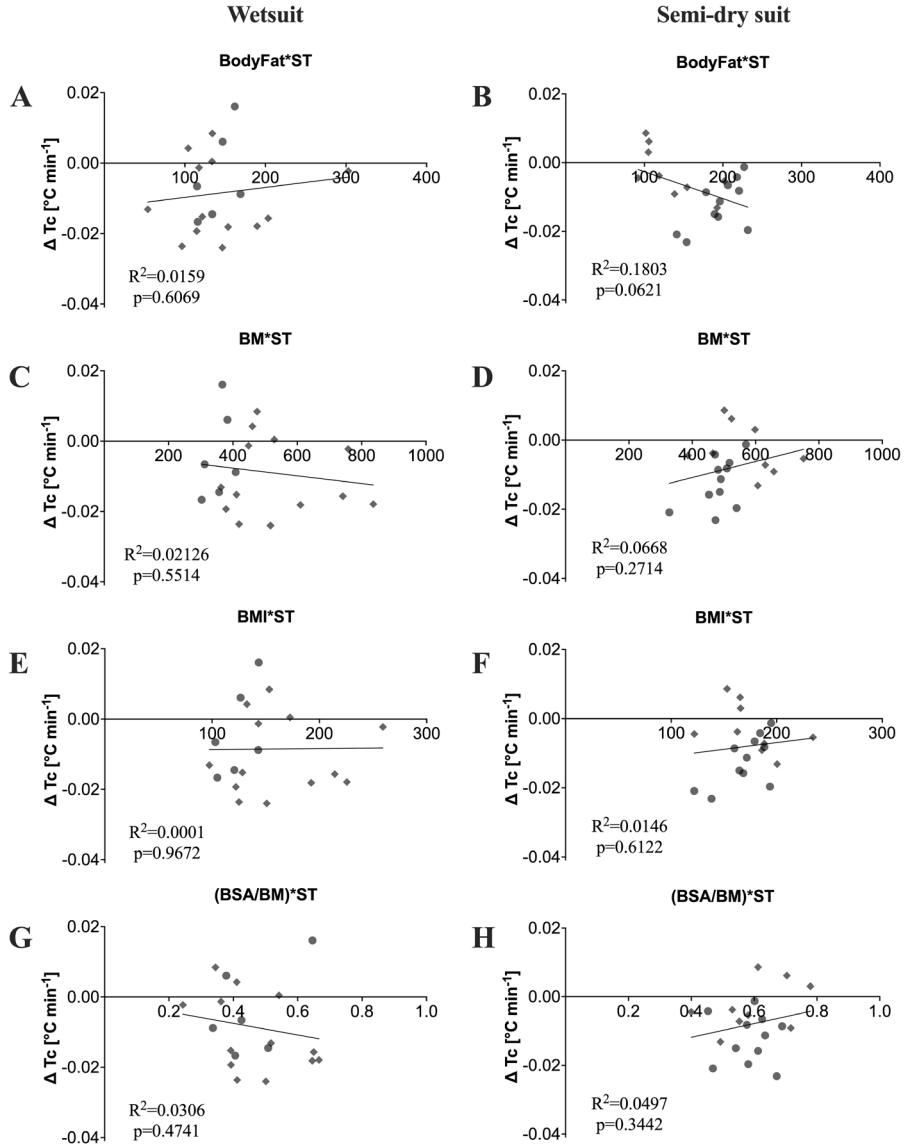
Many studies have examined thermoregulatory variables in scuba divers, however, previous reports typically had small numbers of participants who were mostly males diving under extreme conditions,[8,9] and/or highly specialised and selected groups, like Navy SEALs.[18] In some cases, these studies used specialised equipment such as argon as the insulation gas in a dry suit.[19,20] While being instrumental in laying the groundwork for male divers in extreme conditions, their applicability may be limited for the much greater percentage of male and female Scuba divers diving for recreational purposes in less extreme conditions. Additionally, no published study has directly compared wetsuits and semi-drysuits, despite both being commonly used in recreational diving. In part, this likely reflects the genuine difficulty of the comparison rather than a simple gap in the literature, as thermal protection depends not only on suit category and thickness but also on regional fit, flexibility, seal integrity, and gaps at the interface between skin and suit.

Previous work from our group reported temperature changes in scuba divers wearing wetsuits, normalised for bottom times of long, relatively shallow (47 minutes at 18 msw) and short, relatively deep dive profiles (20 minutes at 30 msw). These measurements were conducted in water ~4.0°C colder than in the current study and resulted in core temperature changes of -0.9 (0.5)°C.[12] This change in core temperature was more than twice that of the -0.4 (0.6)°C recorded in the wetsuit group in the current study, emphasising the significant influence of water temperature on core temperature maintenance. Measures of thermal sensation were not made in the previous study and were only assessed in the current study at ~22.6°C water temperature. However, we did not detect any differences in thermal sensation between wetsuits and semi-drysuits, aligning with the lack of differences in core temperature measurements. These findings indicate that any differences in skin and core temperature changes between wetsuits and semi-drysuit users were insufficient to be perceived as thermally different. Thus, the perceived and/or actual benefits of semi-drysuits over wetsuits might only emerge in colder water temperatures, which have not yet been studied.

The degree to which shivering, non-shivering thermogenesis, or physical activity contributed to maintaining core temperature is unclear. Earlier work in ~17.5°C water temperature found no difference in fibroblast growth factor

Figure 2

Linear regressions between the change in core temperature per minute of dive time ($\Delta Tc \cdot min^{-1}$) and the anthropometric factors body fat % (A+B), body mass (BM, kg; C+D), body mass index (BMI, $kg \cdot m^2$; E+F), and body surface area divided by BM (BSA/BM; G+H) multiplied by total suit thickness (mm) (ST). Males: diamonds. Females: circles. a.u. – arbitrary units



21 as an indicator for non-shivering thermogenesis, although studies in 13.5°C water did report differences,[21–23] again demonstrating the importance of water temperature in regulating the physiological responses during the dive. It can be assumed that shivering and non-shivering thermogenesis did not play a dominant role in the current study's ~22.6°C water temperature.

Thicker wetsuits, a higher body weight, a higher body mass index, or a lower body surface area to body mass ratio led to a reduced change in core temperature in previous work conducted in ~17–18°C water temperature,[12] suggesting that participants with these anthropometrics might be able to choose a thinner suit for dives in these water temperatures. This could not be replicated by the current findings in ~22.6°C water, with no associations between suit type, suit thickness, and core temperature change. Correlations between anthropometric factors and core temperature change emerged only for the percentage of body fat in semi-drysuit dives and remained non-significant when normalising body fat percentage for suit thickness (see Figure 2). While these data suggest that the suit types were adequately chosen for that water temperature and personal perception, the negative correlation of body fat% with the rate of temperature change in the semi-drysuits is counterintuitive, as the body fat should theoretically provide additional thermal insulation. When considering the previous study in 17–18°C water and the current study in ~22.6°C water, these two studies suggest that the significant effect of anthropometrics and wetsuit thickness might depend on water temperature. Specifically, at warmer water temperatures, a suit thickness range of 5–8 mm may be sufficient for maintaining core temperature

without considering anthropometrics. Conversely, at lower temperatures, a suit thickness range of 5–8 mm may be insufficient for maintaining core temperature without considering anthropometrics. The temperature threshold where this occurs has not yet been determined. In the current study, it can only be speculated that semi-drysuits might have presented a less ideal fit for individuals with a high body fat percentage, as that was negatively correlated to temperature loss. Commercially available suits are designed for males and females in different sizes, but few are individually tailored to account for individual anthropometrics, which might be addressed in the future by recent advances, such as 3D scanning.[24] Therefore, it must be noted that the range in suit thickness and suit fit offered by the manufacturer's rental equipment might not provide the ideal choice for everyone for a given set of environmental conditions. It should also be acknowledged that suit selection in practice involves an individual component: cold tolerance, prior thermal experience, and subjective comfort vary between divers, and no anthropometric formula will fully replace personal experience in choosing a suit. The approach taken here is therefore intended to complement, rather than substitute for, individual judgement, by identifying conditions under which anthropometric and suit characteristics meaningfully predict thermal responses and conditions under which they do not. Future studies should examine the effects of semi-drysuits on core temperature and thermal perception in colder water temperatures than those used for the current study.

LIMITATIONS

Several limitations should be acknowledged. The study did not employ a crossover design, limiting within-subject comparisons between suit types. The sample size was small relative to the number of influencing variables, reducing statistical power and limiting the ability to resolve interindividual variability. Thermal stress was modest, as dives were conducted in relatively warm water (22.6 °C) and exposure times were short, which may have limited the detection of differences in thermal strain between suit types. Dive profiles and underwater activity were not controlled, and depth varied between dives. As water depth influences neoprene compression, this likely introduced variability in suit insulation during exposure. The use of ancillary thermal protection (e.g., hoods and handwear) was not standardised and may have contributed additional variability in heat loss between participants. The insulation properties of individual suits were not quantified. Suit age, prior usage history, material degradation, and individual fit were not systematically controlled and likely influenced thermal insulation. These factors represent important, unmeasured sources of variability. Core temperature was assessed using an ingestible thermal sensor, which is subject to variability related to gastrointestinal transit. Body surface area and body fat percentage were estimated rather than directly measured, introducing additional uncertainty in the assessment of anthropometric influences on thermal responses.

Conclusions

We found that wetsuits and semi-drysuits between 5–8 mm thickness both provided sufficient thermal protection for male and female scuba divers in ~22.6°C water temperature performing recreational dives. Within these specific conditions, sex assigned at birth and individual anthropometrics when normalised for suit thickness were not associated with temperature loss or thermal sensation. Additional studies are needed in male and female scuba divers wearing different suit types across a wider range of water temperatures to determine the temperature and suit thickness thresholds for considering anthropometrics for the maintenance of core temperature.

References

1 Pendergast DR, Lundgren CEG. The underwater environment: cardiopulmonary, thermal, and energetic demands. J Appl Physiol (1985). 2009;106:276–83. doi: 10.1152/japplphysiol.90984.2008. PMID: 19036887.

2 Greenfield AM, Charkoudian N, Alba BK. Influences of ovarian hormones on physiological responses to cold in women. Temperature (Austin). 2022;9:23–45. doi: 10.1080/23328940.2021.1953688. PMID: 35655670. PMCID: PMC9154773.

3 Smith GB Jr, Hames EF. Estimation of tolerance times for cold water immersion. Aerosp Med. 1962;33:834–40. PMID: 13914228.

4 Castellani JW, Young AJ. Human physiological responses to cold exposure: Acute responses and acclimatization to prolonged exposure. Auton Neurosci. 2016;196:63–74. doi: 10.1016/j.autneu.2016.02.009. PMID: 26924539.

5 Gagge AP, Gonzalez RR. Mechanisms of heat exchange: biophysics and physiology. In: Terjung R, editor. Comprehensive Physiology. 1st ed. Wiley; 1996. p. 45–84. doi: 10.1002/cphy.cp040104.

6 Warner ME, Nessler JA, Newcomer SC. Skin temperatures in females wearing a 2 mm wetsuit during surfing. Sports (Basel). 2019;7:145. doi: 10.3390/sports7060145. PMID: 31197125. PMCID: PMC6628164.

7 Doubt TJ. Cardiovascular and thermal responses to SCUBA diving: Med Sci Sports Exerc. 1996;28:581–6. doi: 10.1097/00005768-199605000-00007. PMID: 9148087.

8 Kelly KR, Arrington LJ, Bernards JR, Jensen AE. Prolonged extreme cold water diving and the acute stress response during military dive training. Front Physiol. 2022;13:842612. doi: 10.3389/fphys.2022.842612. PMID: 35874531. PMCID: PMC9304957.

9 Lafère P, Guerrero F, Germonpré P, Balestra C. Comparison of insulation provided by dry or wetsuits among recreational divers during cold water immersion (< 5°C). Int Marit Health. 2021;72: 217–22. doi: 10.5603/IMH.2021.0040. PMID: 34604992.

10 Chapin AC, Arrington LJ, Bernards JR, Kelly KR. Thermoregulatory and metabolic demands of naval special warfare divers during a 6-h cold-water training dive. Front Physiol. 2021;12:674323. doi: 10.3389/fphys.2021.674323. PMID: 34658902. PMCID: PMC8511400.

11 Pollock NW. Scientific diving in Antarctica: history and current practice. Diving Hyperb Med. 2007;37:204–11. Available

from: https://dhmjournal.com/images/IndividArticles/37Dec/Pollock_dhm.37.4.204-211.pdf.
12 Bradbury KE, DiMarco KG, Futral JE, Lord RN, Edward JA, Barak O, et al. The maintenance of core temperature in SCUBA divers: contributions of anthropometrics, patent foramen ovale, and non-shivering thermogenesis. J Sci Med Sport. 2024;27:820–7. doi: 10.1016/j.jsams.2024.07.010. PMID: 39209689.
13 Orman T, Bradbury KE, Grosshennig T, Perez M, Möller FN, Dujić Ž, et al. Maintenance of core temperature in SCUBA divers in cold water: contributions of anthropometrics, suit type, and sex. Eur J Appl Physiol. 2025; 126(2):853–65. doi: 10.1007/s00421-025-05961-5. PMID: 40908408. PMCID: PMC12948925.
14 O'Connor P, Hyde D, Clarke J. The cognitive and physiological effects of heating the torso of a submerged diver. Proceedings of the Human Factors and Ergonomics Society Annual Meeting. 2008;52:738–42. doi: 10.1177/154193120805201109.
15 Young AJ, Sawka MN, Epstein Y, Decristofano B, Pandolf KB. Cooling different body surfaces during upper and lower body exercise. J Appl Physiol (1985). 1987;63:1218–23. doi: 10.1152/jappl.1987.63.3.1218. PMID: 3654466.
16 Möller FN, Jacobi E, Hoffmann U, Vogt T. Physiological and cognitive responses to hyperoxic exercise in full water submersion. Eur J Sport Sci. 2023;23:1647–57. doi: 10.1080/17461391.2023.2193942. PMID: 37009949.
17 Jackson AS, Stanforth PR, Gagnon J, Rankinen T, Leon AS, Rao DC, et al. The effect of sex, age and race on estimating percentage body fat from body mass index: The Heritage Family Study. Int J Obes Relat Metab Disord. 2002;26:789–96. doi: 10.1038/sj.ijo.0802006. PMID: 12037649.
18 Chapin AC, Arrington LJ, Bernards JR, Kelly KR. Thermoregulatory and metabolic demands of naval special warfare divers during a 6-h cold-water training dive. Front Physiol. 2021;12:674323. doi: 10.3389/fphys.2021.674323. PMID: 34658902. PMCID: PMC8511400.
19 Lundell RV, Wuorimaa T, Räisänen-Sokolowski A, Sundholm JK, Rintamäki H, Rissanen S, et al. Comparison of argon and air as thermal insulating gases in drysuit dives during military Arctic diving equipment development tests. Undersea Hyperb Med. 2019;46:429–35. doi: 10.22462/06.08.2019.5. PMID: 31509899.
20 Vrijdag XC, van Ooij PJA, van Hulst RA. Argon used as dry suit insulation gas for cold-water diving. Extreme Physiol Med. 2013;2:17. doi: 10.1186/2046-7648-2-17. PMID: 24438580. PMCID: PMC3710141.
21 Jeon YN, Ra SG, Kim CK. Effect of a bout of acute cold water immersion on circulating FGF21, Irisin and T3 hormones in SCUBA divers. Exerc Sci. 2015;24:283–9. doi: 10.15857/ksep.2015.24.3.283.
22 Klein Hazebroek M, Keipert S. Adapting to the cold: a role for endogenous fibroblast growth factor 21 in thermoregulation? Front Endocrinol (Lausanne). 2020;11:389. doi: 10.3389/fendo.2020.00389. PMID: 32714278. PMCID: PMC7343899.
23 Lee P, Linderman JD, Smith S, Brychta RJ, Wang J, Idelson C, et al. Irisin and FGF21 are cold-induced endocrine activators of brown fat function in humans. Cell Metab. 2014;19:302–9. doi: 10.1016/j.cmet.2013.12.017. PMID: 24506871. PMCID: PMC7647184.
24 Petrak S, Naglić MM, Geršak J. Sizing and fit for swimsuits and diving suits. In: Anthropometry, apparel sizing and design. 2nd ed. Elsevier; 2020. p. 255–87. doi: 10.1016/B978-0-08-102604-5.00010-X.

Acknowledgements

We thank all our divers for their participation, as well as Alenka Kokalj, the dive masters, and dive centres *Big Blue* (Croatia) and *Roses Sub* (Spain) for their support and collaborative spirit. We also thank Elena Jacobi for outstanding logistical support in Spain and help in collecting data.

Conflicts of interest and funding

The PADI Foundation and the University of Oregon funded this study. Temperature sensing equipment and consumables were provided by a Cooperative Research and Development Agreement between the Department of Defense and the University of Oregon. No conflicts of interest were declared

Submitted: 31 October 2025
Accepted after revision: 22 May 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

Microbiological monitoring of scuba cylinders under routine filling and seasonal diving conditions

Recep Özkan[1], Osman Turkmen[2], Elif Okumuş[3], Sukran Özhan[4], Hasan Sivrikaya[2], Ese Başbulut[5], Kübra Ozgok Kangal[6]

[1] *Department of Hyperbaric and Diving Medicine, Ataturk University Erzurum, Türkiye*
[2] *Department of Hyperbaric and Diving Medicine, Samsun Training and Research Hospital, Samsun, Türkiye*
[3] *Department of Medical Microbiology, University of Health Sciences, Van Training and Research Hospital, Van, Türkiye*
[4] *Department of Medical Microbiology, Erzurum City Hospital, Erzurum, Türkiye*
[5] *Department of Medical Microbiology, Samsun Training and Research Hospital, Samsun, Türkiye*
[6] *Department of Hyperbaric and Diving Medicine, University of Health Sciences, Gülhane Training and Research Hospital, Ankara, Türkiye*

Corresponding author: *Dr Recep Özkan, Department of Hyperbaric and Diving Medicine, Ataturk University, 25240 Erzurum, Türkiye*
ORCiD: *0000-0001-7342-6473*
recepzkn@hotmail.com

Keywords
Air; Bacteriology; Compressors; Contamination; Gas supply; Infection; Scuba diving

Abstract
(Özkan R, Turkmen O, Okumuş E, Özhan S, Sivrikaya H, Başbulut E, Ozgok Kangal K. Microbiological monitoring of scuba cylinders under routine filling and seasonal diving conditions. Diving and Hyperbaric Medicine. 2026;56(3):265–270. doi: 10.28920/dhm56.3.265-270. PMID: 42743565.)
Introduction: The aim of this study was to monitor the microbiological status of the inner surfaces of scuba cylinders under routine air filling conditions and to assess whether microbiological contamination emerges or persists over time.
Methods: A prospective longitudinal microbiological study was conducted using two complementary sampling models. Serial sampling of a steel scuba cylinder and its associated compressor air filter was performed at three-day intervals over 45 days. To assess reproducibility under operational conditions, additional microbiological sampling was performed from 27 scuba cylinders actively used during the diving season. Samples were analysed using standard culture methods.
Results: No microbial growth was detected in bacterial cultures obtained from any of the cylinder inner surface or compressor air filter samples throughout the 45-day monitoring period. No temporal changes or cumulative contamination trends were observed. Similarly, no microbial growth was detected in cultures obtained from the additional 27 scuba cylinders sampled during the active diving season.
Conclusions: Under routine operating conditions with standard maintenance and air filtration practices, the internal environment of scuba cylinders may remain microbiologically stable over time, suggesting a low risk of microbiological contamination in properly maintained compressed air systems.

Introduction

Infections associated with scuba diving have most commonly been attributed to direct exposure to aquatic environments, contact with moist surfaces, and variations in environmental hygiene. In particular, the inadvertent ingestion of water that may contain fecal contamination during diving has been linked to an increased risk of gastrointestinal infections, as well as infections involving the ear, skin, eyes, and respiratory tract.[1] Natural water environments, which may vary considerably in temperature, humidity, and microbial load, have also been reported to facilitate the development of infections during diving activities.

In contrast, the contribution of diving equipment to infection risk has received relatively limited attention. Although shared components such as regulators, mouthpieces, masks, and other parts of the breathing circuit are often considered potential vectors for microbial transmission, available data on their microbiological contamination remain scarce and inconsistent. Notably, the microbiological safety of compressed breathing air within scuba cylinders has not been adequately explored in the existing literature.

From a theoretical standpoint, both the internal surfaces of scuba cylinders and the compressor systems used for filling may be exposed to several factors that could influence microbiological contamination. Moisture introduction, ingress of ambient air during filling, filter performance, and maintenance practices are all variables that may affect the microbiological quality of breathing air. Despite these theoretical considerations, data regarding the microbiological stability of scuba cylinders and associated compressor systems over time remain limited.

Evaluating the microbiological stability of a scuba cylinder and its associated compressor filter over time is therefore relevant not only for diving safety but also for assessing the effectiveness of routine equipment maintenance protocols. A longitudinal monitoring approach allows for the detection of potential cumulative contamination and subtle microbiological changes that may otherwise go unnoticed.

Accordingly, the present study prospectively and longitudinally monitored a single steel scuba cylinder and the air filter of the compressor used to fill it under routine operating conditions. The primary objective was to generate data addressing this underexplored aspect of microbiological safety in compressed air systems used in scuba operations and to provide a methodological foundation for future studies involving larger sample sizes and multicentre designs.

In addition, to assess whether the findings observed in the longitudinal monitoring model were reproducible under real-world conditions, a single-time-point microbiological assessment was performed in 27 scuba cylinders actively used during the diving season at an independent diving centre.

Methods

A. EXPERIMENTAL SETUP

A scuba cylinder that had completed routine maintenance and cleaning protocols was chosen as the representative sample for the investigation. A single 15 L steel scuba cylinder (Aqualung, working pressure 230 bar) fitted with a Coltri Sub valve (EN 144-1, M25×2 thread) and a portable petrol-driven high-pressure breathing air compressor (CompTrade Kompressoren, Austria) were used throughout the study under routine operating conditions (Figure 1).

Figure 1

The steel scuba cylinder and the portable air compressor system used throughout the longitudinal monitoring period. The same cylinder and compressor were used for all filling procedures and microbiological sampling under routine operating conditions



At the beginning of the experimental period, a swab sample was obtained from the cylinder valve outlet using a sterile swab and placed in an appropriate transport medium. In addition, to ensure contact with the entire inner surface of the cylinder, approximately 50 mL of sterile physiological saline was introduced into the cylinder. The cylinder was gently rotated to allow the fluid to contact all internal surfaces, after which the lavage fluid was transferred into a sterile, capped container.

The swab and lavage samples were assessed for bacterial contamination through inoculation into 5% sheep blood agar and chocolate agar medium. The inoculated plates were incubated at 35 ± 1°C for 48 hours. No microbial growth was detected during the incubation time; hence, the scuba cylinder was considered suitable for inclusion in the experimental study.

B. CYLINDER FILLING AND TEMPERATURE MEASUREMENT

In the primary longitudinal monitoring model, microbiological sampling was performed at three-day intervals over a 45-day period. A total of 15 sampling events were conducted, during which samples were collected from both the scuba cylinder and the associated compressor air filter. During each filling procedure, temperature measurements were obtained using a non-contact infrared thermometer (Bosch, Germany) directed at the external surface of the cylinder near its midpoint. Measurements were recorded at 10-bar increments as cylinder pressure increased from 0 to 200 bar.

The 27 scuba cylinders included in the supplementary sampling had been filled during the active diving season at an air filling station in accordance with the requirements for breathable compressed air defined in the EN 12021:2014 standard (European Committee for Standardization. EN 12021:2014. Respiratory protective devices – Compressed gases for breathing apparatus. Brussels: CEN; 2014). These cylinders were subsequently depressurised prior to microbiological sampling.

C. THE EXPERIMENT

Sampling of the scuba cylinder inner surface was performed using a sterile lavage technique. Sampling of the scuba cylinder inner surface was performed using the sterile lavage procedure described above. The collected lavage fluid was subsequently used for microbiological analysis. Following each sampling procedure, the internal surface of the cylinder was rinsed with distilled water to remove residual sampling fluid and was allowed to dry completely at room temperature. No chemical sterilisation procedures were applied. After drying, the cylinder was refilled according to standard air-

filling procedures and stored under routine indoor conditions until the next scheduled sampling interval. This standardised handling procedure was applied throughout the study period.

Sampling of the compressor air filter was performed using sterile swabs. Microbiological sampling was carried out exclusively from the actively used compressor air filter (Figure 2, right), while the previously used filter (Figure 2, left) is presented solely for illustrative comparison. Swab samples were placed in Stuart transport medium to preserve microbial viability during transport and delivered to the microbiology laboratory without delay.

To exclude potential contamination originating from the physiological saline, negative control cultures were performed prior to sampling by inoculating the saline onto 5% sheep blood agar and chocolate agar and incubating under appropriate conditions; no microbial growth was observed.

Both the scuba cylinder inner surface lavage samples and the compressor air filter swab samples were inoculated onto 5% sheep blood agar and chocolate agar plates. All plates were incubated aerobically at 35 ± 1°C. Cultures were initially evaluated at 24 hours, and plates showing no growth or requiring further assessment were incubated for a total of 48 hours.

Supplementary microbiological sampling of the 27 scuba cylinders was performed as a single-time-point assessment during the active diving season. These cylinders were sampled following routine air filling procedures using the same microbiological culture methods to assess whether the findings obtained from the primary longitudinal model were consistent under real-world operational conditions. After sample collection, the internal surfaces of the cylinders were rinsed with distilled water and allowed to dry at room temperature. No repeated sampling was performed in these cylinders.

Results

During repeated air-filling procedures, temperature measurements were recorded at regular pressure intervals from 0 to 200 bar across a total of 15 separate filling events. In all filling cycles, the cylinder surface temperature increased progressively with rising pressure, reaching a maximum of approximately 35°C at 200 bar. The most pronounced temperature increase consistently occurred during the early phase of pressurisation, followed by a gradual reduction in the rate of temperature rise as filling continued.

Throughout the study period, no bacterial growth was detected in any of the 30 samples collected over 45 days, including 15 scuba cylinder inner surface lavage samples and 15 compressor air filter swab samples. Representative blood agar plates obtained from both sample types demonstrated no bacterial growth after 48 hours of incubation (Figure 3A–D).

Figure 2

Photographs of the compressor air filter elements. The filter shown on the right represents the actively used filter from which microbiological sampling was performed throughout the study period. The filter shown on the left represents a previously used and discarded filter and is included for illustrative comparison only; no microbiological samples were obtained from this filter



Similarly, no bacterial growth was observed on either 5% sheep blood agar or chocolate agar at 24 or 48 hours of incubation for any sample. In addition, sterile physiological saline used as a negative control showed no evidence of bacterial growth following incubation.

In addition, microbiological cultures obtained from 27 compressed air cylinders actively used during the diving season also demonstrated no bacterial growth following incubation under the same culture conditions.

Discussion

This longitudinal investigation found no microbial growth in serial samples collected over a 45-day period from the inner surface of a steel compressed air cylinder and from the air filter of the compressor used for air filling. Thus, under the conditions of the present study, bacterial contamination was not detected in the monitored scuba cylinder and compressor air filter throughout the monitoring period. The lack of observable microbial growth may be mainly linked to the utilisation of diving apparatus that undergoes proper maintenance and efficient filtration systems. In particular, efficient filtration in diving compressors can limit the passage of particulate matter and microorganisms, thereby reducing the risk of microbial contamination within compressed air systems.

Similarly, regular maintenance and cleaning practices play an important role in eliminating potential sources of contamination.

Temperature and pressure are established physical parameters that affect bacterial growth, survival, and viability.[2–4] Prior research indicates that increased pressure and temperature may exert inhibitory effects on specific microbes; however, the effectiveness of these factors is primarily dependent upon the intensity and length of exposure.[2–6] The present

study demonstrated that repeated air-filling methods led to a gradual rise in cylinder surface temperature, reaching a peak of around 35°C at 200 bar during 15 distinct filling events. Despite this increase, no bacterial colonisation was observed in any samples from the inner surfaces of scuba cylinders or compressor air filters collected throughout the 45-day monitoring period.

The temperature range recorded during normal filling stayed within ranges typically tolerated by several environmental and opportunistic bacteria.[2–4] These data indicate that the alterations in pressure and temperature experienced during routine scuba cylinder filling are inadequate to induce bacterial inactivation or inhibit potential microbial survival.[2,3,5] The obtained thermal profile aligns with the adiabatic compression of air, succeeded by partial heat transfer to the surrounding environment during the filling procedure.

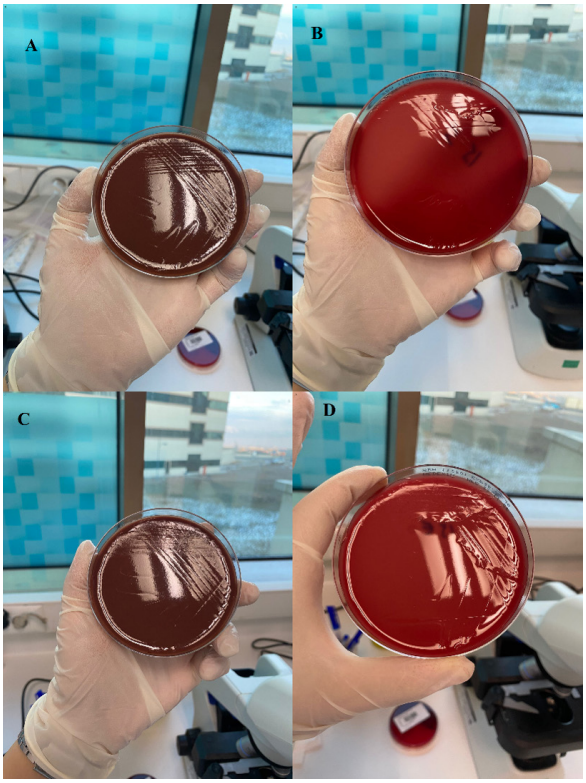
Importantly, the absence of bacterial colonisation throughout repeated sampling under natural operating conditions highlights the potential effectiveness of routine maintenance, proper cylinder handling, and standard air filtration practices in preventing microbial contamination of compressed air cylinder and filling systems. These findings indicate that, under appropriate operational conditions, the internal environment of a steel scuba cylinder can remain microbiologically stable over time. The absence of bacterial growth across all sampled time points, as illustrated in Figure 3A–D, supports the microbiological stability of the system.

Previous studies have emphasised microbial contamination risks associated primarily with external diving equipment and communal water sources. Washburn et al.[7] demonstrated that rinse tanks used communally by divers may harbor a wide range of microorganisms, including opportunistic pathogens, originating from both environmental and human sources. Similarly, investigations by the Divers Alert Network[8] reported that bacterial contamination in rinse tanks increased throughout the day due to repeated use and inadequate hygiene practices. While these investigations concentrated on external equipment surfaces, they highlighted the significance of appropriate handling and maintenance methods, as external pollution could potentially operate as a source of secondary contamination for internal air systems during filling or handling. Collectively, these findings suggest that contamination risks reported in diving environments are predominantly associated with external equipment surfaces and shared-use facilities rather than the internal environment of compressed air systems.

The inside environment of scuba cylinders often comprises a dry, high-pressure, and nutrient-deficient microenvironment. The physical and environmental circumstances are typically unfavorable to the survival and reproduction of numerous bacterial and fungal species. In comparison to ambient air, humidity levels within scuba cylinders are significantly

Figure 3

Representative blood agar culture plates obtained during the longitudinal monitoring period. Panels A and B show cultures from scuba cylinder inner surface lavage samples, and panels C and D show cultures from compressor air filter swab samples. All plates were incubated aerobically at 35 ± 1°C and demonstrated no bacterial growth after 48 hours. The images shown are representative of all samples collected during the study period



reduced. Low humidity facilitates evaporation, resulting in heightened solute concentrations and increased osmotic pressure, which can harm or inactivate microorganisms, hence diminishing microbial activity and growth.[9–11]

Moreover, elevated hydrostatic pressure has demonstrated efficacy in inactivating several vegetative bacteria, yeasts, and moulds.[12,13] The simultaneous occurrence of elevated pressure and diminished humidity constitutes a substantial stressor for microorganisms, hence decreasing the probability of successful colonisation. The physical characteristics of scuba cylinders, specifically low humidity and high pressure, produce an inhospitable habitat for microbial survival and sustained colonisation. Collectively, these physical attributes offer a credible rationale for the microbiological stability noted during the monitoring period and support the lack of detectable bacterial colonisation in this investigation.

The study by van Rijen et al. investigating MRSA (methicillin-resistant *Staphylococcus aureus*) transmission associated with shared scuba cylinder use suggests that the sharing of scuba diving equipment may be linked to community-acquired MRSA carriage, most likely through direct contact with contaminated equipment surfaces

rather than via airborne transmission originating from the tank itself.[14] In contrast, in the present study, no microbial growth was detected in any of the serial samples obtained from the inner surfaces of scuba cylinders or from compressor air filters. This finding supports the notion that contamination risks reported in the literature are predominantly associated with external equipment surfaces and that, under appropriate maintenance and filtration conditions, the internal environment of scuba cylinders can remain microbiologically stable.

In addition to the primary longitudinal monitoring model, supplementary microbiological cultures obtained from 27 compressed air cylinders actively used during the diving season also demonstrated no detectable microbial growth. The absence of bacterial colonisation in these real-world operational cylinders further supports the consistency of the longitudinal findings and suggests that microbiological stability of the internal cylinder environment may be maintained even under active underwater use and repeated refilling conditions.

This study has several limitations. The primary longitudinal monitoring component was conducted using a single compressed air cylinder and a single compressor system, while additional sampling was performed in multiple cylinders under real-world conditions; therefore, the generalisability of the findings to other cylinder materials, capacities, compressor types, or filling environments may be limited. The microbiological assessment exclusively utilised culture-based bacterial approaches, without the application of molecular detection techniques. Temperature data were recorded solely during filling activities, excluding storage and discharge phases. Furthermore, certain fastidious, slow-growing, or non-cultivable microorganisms may not have been detected using the culture-based methods employed in this study. In addition, temperature data were recorded solely from the external cylinder surface during filling activities and did not directly measure internal gas temperatures. Therefore, it cannot be excluded that transient internal temperatures may have exceeded the recorded surface temperatures and potentially influenced microbial viability.

Conclusions

This longitudinal investigation found no bacterial colonisation in serial samples obtained from the inner surfaces of a compressed air cylinder or from the associated compressor air filter over a 45-day monitoring period. In addition, no microbial growth was detected in samples collected from 27 compressed air cylinders actively used during the diving season, supporting the consistency of the primary longitudinal findings. Collectively, these results indicate that, under routine operating conditions and with appropriate maintenance and air filtration practices, the internal environment of scuba cylinders used in scuba systems can remain sterile and microbiologically stable over time.

Although the study employed a culture-based approach and a focused longitudinal model, it provides a structured methodological framework for future investigations involving larger sample sizes, different operational settings, and complementary molecular diagnostic techniques which would be necessary to achieve more thorough and generalisable understanding of the microbiological safety of scuba diving systems.

References

1 Schijven J, de Roda Husman AM. A survey of diving behaviour and accidental water ingestion among Dutch occupational and sport divers to assess the risk of infection with waterborne pathogenic microorganisms. Environ Health Perspect. 2006;114:712–7. doi: 10.1289/ehp.8523. PMID: 16675425. PMCID: PMC1459924.

2 Chen YY, Temelli F, Gänzle MG. Mechanisms of inactivation of dry Escherichia coli by high-pressure carbon dioxide. Appl Environ Microbiol. 2017;83(10):e00062–17. doi: 10.1128/AEM.00062-17. PMID: 28283526. PMCID: PMC5411503.

3 Chen YY, Gänzle MG. Influence of cyclopropane fatty acids on heat, high pressure, acid and oxidative resistance in Escherichia coli. Int J Food Microbiol. 2016;222:16–22. doi: 10.1016/j.ijfoodmicro.2016.01.017. PMID: 26828814.

4 Mallidis CG, Drizou D. Effect of simultaneous application of heat and pressure on the survival of bacterial spores. J Appl Bacteriol. 1991;71:285–8. doi: 10.1111/j.1365-2672.1991.tb04460.x.

5 Malinowska-Pańczyk E, Kołodziejska I, Dunajski E. Effect of high pressure on selected bacteria at subzero temperature. Pol J Food Nutr Sci. 2008;58:419–24.

6 Diels AM, Michiels CW. High-pressure homogenization as a non-thermal technique for the inactivation of microorganisms. Crit Rev Microbiol. 2006;32:201–16. doi: 10.1080/10408410601023516. PMID: 17123905.

7 Washburn BK, Levin AE, Hennessy K, Miller MR. Identification of bacteria in scuba divers' rinse tanks. Undersea Hyperb Med. 2010;37:233–40. PMID: 20737930.

8 Divers Alert Network. Microbial hazards. [cited 2026 Feb 6]. Available from: https://dan.org/alert-diver/article/microbial-hazards/.

9 Haines SR, Hall EC, Marciniak K, Misztal PK, Goldstein AH, Adams RI, et al. Microbial growth and volatile organic compound (VOC) emissions from carpet and drywall under elevated relative humidity conditions. Microbiome. 2021;9(1):209. doi: 10.1186/s40168-021-01158-y. Erratum in: Microbiome. 2021;9(1):219. doi: 10.1186/s40168-021-01179-7. PMID: 34666813. PMCID: PMC8524935.

10 Dedesko S, Siegel JA. Moisture parameters and fungal communities associated with gypsum drywall in buildings. Microbiome. 2015;3:71. doi: 10.1186/s40168-015-0137-y. PMID: 26642923. PMCID: PMC4672539.

11 Lin K, Marr LC. Humidity-dependent decay of viruses, but not bacteria, in aerosols and droplets follows disinfection kinetics. Environ Sci Technol. 2020;54(2):1024–32. doi: 10.1021/acs.est.9b04959. PMID: 31886650.

12 Lee YC, Tsai YH, Chen SL, Kung HF, Arakawa O, Wei CI. Inactivation and damage of histamine-forming bacteria by treatment with high hydrostatic pressure. Foods. 2020;9(3):266. doi: 10.3390/foods9030266. PMID: 32131391. PMCID: PMC7143767.

13 Syed QA, Buffa M, Guamis B, Saldo J. Factors affecting

bacterial inactivation during high hydrostatic pressure processing of foods: a review. Crit Rev Food Sci Nutr. 2016;56:474–83. doi: 10.1080/10408398.2013.779570. PMID: 25749439.

14 van Rijen MM, Kluytmans-van den Bergh MF, Verkade EJ, Ten Ham PB, Feingold BJ, Kluytmans JA, CAM Study Group. Lifestyle-associated risk factors for community-acquired methicillin-resistant Staphylococcus aureus carriage in the Netherlands: An exploratory hospital-based case-control study. PLoS One. 2013;8(6):e65594. doi: 10.1371/journal.pone.0065594. PMID: 23840344. PMCID: PMC3686778.

Conflicts of interest and funding: nil

Submitted: 7 February 2026
Accepted after revision: 14 June 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

The effect of drysuit diving in warm water on body temperature and post immersion orthostatic hypotension

Joshua T Murphey[1], Hayden W Hess[1], Morgan L Worley[1], Glenda Anderson[1], Jacqueline Schwob[1], Brian A Monaco[1,2], David Hostler[1,2]

[1] *Center for Research and Education in Special Environments, Exercise and Nutrition Sciences, University at Buffalo, Buffalo, NY, USA*
[2] *Department of Emergency Medicine, University at Buffalo, Buffalo, NY, USA*

Corresponding author: *Dr David Hostler, University at Buffalo, Exercise and Nutrition Sciences, 116 Sherman Hall, Buffalo NY 14020, USA*
ORCiD: *0000-0002-7711-4001*
dhostler@buffalo.edu

Keywords
Body temperature regulation; Cardiovascular physiology; Hyperthermia; Immersion; Orthostatic intolerance; Protective clothing

Abstract
(Murphey JT, Hess HW, Worley ML, Anderson G, Schwob J, Monaco BA, Hostler D. The effect of drysuit diving in warm water on body temperature and post immersion orthostatic hypotension. Diving and Hyperbaric Medicine. 2026 30 September;56(3):271–279. doi: 10.28920/dhm56.3.271-279. PMID: 42743566.)

Introduction: Warm-water diving can limit heat dissipation, particularly when performed in fully encapsulating protective gear, leading to substantial thermal and cardiovascular strain that may impair diver safety. Following immersion, removal of hydrostatic support combined with heat-induced vasodilation may reduce central blood volume and increase susceptibility to orthostatic intolerance during egress and recovery. The extent to which this thermal strain impairs post-immersion orthostatic tolerance remains unknown.

Methods: Four randomised, crossover immersion trials were conducted at 28°C, 33°C, 38°C without precooling (38°C), and 38°C with precooling (38°C + Cool), with subjects wearing fully encapsulating dive gear. Subjects walked for up to 60 minutes at approximately 50% of $\dot{V}O_{2max}$ heart rate (HR) or until core temperature (Tc) reached 38.5°C, or they voluntarily stopped. Tc, HR, and perceptual measures were recorded every 10 minutes. Orthostatic tolerance was assessed after immersion via a 70° head-up tilt test.

Results: Eight healthy adults completed all aspects of the study. Tc and HR were higher during both 38°C conditions compared with 28°C and 33°C (all $P < 0.01$) with no differences between 38°C and 38°C + Cool. Sweat loss exceeded 1.2 (SD 0.67) $L \cdot h^{-1}$ in both 38°C conditions compared with ≤ 0.3 (0.32) $L \cdot h^{-1}$ at 28°C and 33°C ($P < 0.01$). Survival analysis showed orthostatic tolerance decreased with increasing thermal stress (log-rank $P = 0.027$; trend $P = 0.003$). Precooling did not reduce peak Tc or HR, nor did it improve tolerance time in 38°C water.

Conclusions: Encapsulated warm-water diving causes heat stress and cardiovascular strain that persists after immersion, impairing orthostatic tolerance. Precooling does not significantly reduce these outcomes.

Introduction

Diving in contaminated water requires military and occupational divers to operate fully encapsulated in impermeable protective ensembles that impede heat dissipation. Although these systems provide essential protection against toxic exposures, they may promote body heat storage in warm water, increasing the risk of heat-related illness and limiting the duration of safe operations. Previous work identifies thermal strain as a primary limiting factor for endurance during warm-water diving.[1,2] This ultimately constrains performance and dive profiles. The water temperatures selected for the present study were intended to represent a range of operationally relevant warm-water exposures, from temperate or thermoneutral conditions to extreme warm water environments. The 28°C and 33°C conditions approximate water temperatures that may be encountered during prolonged operations in warm coastal, harbor, or industrial settings, whereas 38°C represents a high thermal load scenario relevant to enclosed, shallow, sun-heated, industrial, or contaminated-water environments where evaporative heat loss is unavailable and environmental heat gain may be substantial.

Increases in core body temperature during warm-water immersion reflect the combined effects of the environment and metabolic heat. Physical activities commonly performed during diving operations elevate metabolic heat production and further exacerbate heat storage. Increases in core body temperature during warm-water immersion reflect the combined effects of environmental heat load and metabolic heat production. During warm water drysuit diving,

heat exchange remains governed largely by conductive heat flux between the diver and the surrounding water, while encapsulation limits evaporative heat loss and may exacerbate internal heat storage during physical work. Laboratory and field studies in terrestrial environments show that precooling increases heat-storage capacity and delays the onset of life-threatening hyperthermia during exercise,[3–5] suggesting that similar strategies may mitigate thermal burden during warm-water diving.

In addition to thermoregulatory stress, warm-water diving imposes substantial cardio- and cerebrovascular challenges. Post-immersion weakness and pre-syncopal symptoms have been described following passive hot water immersion and exercise in the heat.[6–8] These responses are largely attributed to sustained peripheral vasodilation, venous pooling, and reduced central blood volume, all of which impair arterial pressure regulation.[7–9] During immersion, hydrostatic pressure promotes centralisation of blood volume and may transiently stabilise cardiovascular function. However, this protection is rapidly lost after exiting the water. Experimental evidence indicates that the interval following exercise and hot water immersion is characterised by persistent vasodilation and impaired orthostatic tolerance,[6,7] mechanisms that may be exacerbated by residual hyperthermia.

Diving in warm, contaminated water represents a complex physiological stress model characterised by the simultaneous exposure to heat stress, immersion-induced hydrostatic pressure, physical exertion, and respiratory loading from diving equipment. In addition to increasing work of breathing, helmet and breathing apparatus dead space may contribute to CO_2 retention, which can influence ventilatory drive, cerebral blood flow, and symptoms such as dizziness or lightheadedness. Each of these stressors independently alters systemic and cerebral regulation. Moderate-intensity exercise increases arterial pressure during activity, but is followed by post-exercise hypotension during recovery.[8] Similarly, hot-water immersion induces peripheral haemodynamic responses including vasodilation, reduced central blood volume, and altered arterial pressure profiles comparable to those observed during post exercise recovery.[9] Thermal strain during warm-water immersion induced marked peripheral vasodilation and reductions in systemic vascular resistance,[10] while cerebrovascular responses appear region and vessel specific. Increases in internal carotid artery blood flow and shear have been demonstrated during hot water immersion,[11] and similar elevations in vertebral artery blood flow have been reported,[12] suggesting that global cerebral perfusion is maintained or elevated during immersion despite systemic vasodilation. However, following immersion, the removal of hydrostatic support combined with persistent peripheral vasodilation may reduce arterial pressure and compromise cerebral perfusion. The convergence of hyperthermia, exercise cessation, and removal of hydrostatic support during egress may predispose divers to hypotension and cerebral hypoperfusion, increasing susceptibility to presyncope or syncope.

Despite the operational relevance of these mechanisms, data describing integrated thermoregulatory and cardiovascular responses during and after encapsulated warm-water diving remain limited. Much of the foundational work informing exposure guidance dates to investigations conducted more than two decades ago.[1,2] While precooling strategies are well established in terrestrial and occupational settings,[3–5] their efficacy in encapsulated warm-water diving has not been rigorously evaluated. The purpose of this study was to examine the effects of warm water immersion across a range of water temperatures on thermoregulatory and cardiovascular responses, and to determine whether a precooling intervention improves physiological stability and operational safety. We hypothesised that warm-water diving would increase internal heat storage and impair orthostatic tolerance compared with thermoneutral conditions, and that precooling would reduce heat storage, extend dive tolerance, and attenuate thermal and cardiovascular strain.

Methods

All subjects were recruited from the local community. All subjects completed six laboratory visits, including screening. The study was approved by the University at Buffalo Institutional Review Board (IRB# STUDY00007863). All subjects provided written informed consent prior to participation.

This study employed a randomised, repeated-measures crossover design. Each subject completed four immersion conditions: 28°C, 33°C, 38°C without precooling (38°C), and 38°C with precooling (38°C + Cool). Conditions were separated by a minimum of 72 hours to minimise carryover effects. The order of exposure was randomised and counterbalanced across subjects.

Subjects completed a health history questionnaire and a physical activity readiness questionnaire (PAR-Q+) and were screened for contraindications to ingestion of the telemetric temperature capsule used for continuous core temperature monitoring during experimental visits. Height was measured using a stadiometer (Health-o-meter Professional 402KL, Pelstar LLC, McCook, IL) and body mass was measured to the nearest 0.01 kg using a calibrated digital scale/platform scale (Midrics 2, Minebea Intec USA Inc., Holbrook, NY). The scale was calibrated according to manufacturer specifications before data collection. Body morphology and body composition were assessed using three-dimensional infrared scanning (SS20 Scanner, Size Stream, Cary, NC). Subjects underwent a physician-administered physical examination evaluating musculoskeletal, neurological, cardiovascular, and pulmonary systems. Individuals with a history of neurological, metabolic, pulmonary, or cardiovascular disease or illness were excluded. Maximal aerobic capacity ($\dot{V}O_{2max}$) was determined using an incremental treadmill protocol with open-circuit spirometry (TrueOne, PARVO Medics, Provo, UT, USA). Subjects with a $\dot{V}O_{2max}$ less than 30 $mL \cdot kg^{-1} \cdot min^{-1}$ were excluded.

Subjects reported to the laboratory for all experimental sessions. Experimental procedures were standardised across visits, with the exception of water temperature and the addition of a precooling intervention during one 38°C condition. Subjects were provided standardised pre-visit instructions to arrive normally hydrated, including guidance on fluid intake in the evening before and morning of each experimental visit. Upon arrival, subjects consumed a standardised meal of granola bars providing 4 kcal·kg^{-1} of body mass and then voided their bladder. This initial urine sample was used to measure urine specific gravity as a pre-exposure screening measure. Nude body mass was measured using the same calibrated scale. Urine specific gravity was measured upon arrival using a handheld refractometer (MASTER-SUR/Na, Atago USA Inc., Bellevue, WA, USA), and subjects were required to have a value < 1.020 before testing. Subjects ingested a telemetric temperature capsule (eCelsius Performance, Hérouville Saint-Clair, France) 6–8 hours prior to immersion or inserted the capsule rectally immediately before testing. Mean skin temperature[13] (T_{SK}) was recorded every minute throughout the protocol. iButton thermistors (Maxim Integrated, San Jose, CA) were secured to the skin over the triceps, pectoral region, thigh, and calf on the right side of the body to estimate regional skin temperature. An additional sensor was placed on the right temple to measure the temperature inside the dive helmet, and the data were analysed separately. Subjects were then fitted with a heart rate monitor (H10, Polar Electro, Kemple, FI) and donned lightweight base layers (32°Degrees Heat, David Peyser Sportswear Inc., Bay Shore, NY, USA), drysuit, and gloves (Viking HD, Ansell, Iselin, NJ, USA), and a commercial dive helmet (KM 37, Kirby Morgan Dive Systems, Inc., Santa Maria, California, USA). The drysuit was a fully encapsulating textile garment with vulcanised seams (material density 1550 ± 100 g·m^2). The helmet was equipped with a 350 SuperFlow regulator and represents the standard configuration for U.S. Navy operations. External weights were added as necessary to offset suit and helmet buoyancy and maintain an upright posture while immersed without impeding movement or unnecessarily increasing underwater workload.

Sweat loss was estimated from changes in nude body mass across each trial. Nude body mass was measured before and after immersion using the same calibrated scale. Any urine produced after the pre-immersion body mass measurement was collected and weighed. Because subjects did not consume fluid during the immersion protocol, precooling, or post-testing, estimated sweat loss was calculated as the reduction in nude body mass corrected for urine loss.

Subjects entered a temperature-controlled annular pool set to 27.9 (SD 0.6)°C, 32.7 (0.3)°C, 37.7 (0.3)°C, or 37.7 (0.5)°C and walked for a maximum of 60 minutes at an exercise intensity of approximately 50% of $\dot{V}O_{2max}$, heart rate (HR) until core temperature (Tc) reached 38.5°C, or until the subject requested to stop. The order of exposure was randomised and counterbalanced across subjects. Subjects were fully submerged throughout the experimental protocol. During immersion, HR and Tc were recorded every five minutes, and rate of perceived exertion (RPE), thermal comfort, thermal sensation, and sweating sensation were recorded every 10 minutes.

During the 38°C + Cool condition, prior to immersion, subjects donned a commercially available precooling vest (SICHNUL Summer Cooling Vest) that contained ice packs for 30-minutes. Heart rate and Tc were recorded every five minutes throughout the precooling period. Immediately following the 30-minute precooling period, subjects removed the cooling vest, donned dive gear and entered the water to begin the immersion protocol. For the protocols without a precooling intervention, subjects moved directly from the environmental chamber into dive gear and directly into the water.

Post-immersion testing was performed in a similar fashion in an environmental chamber set to 31.8 (0.1)°C and 71 (1.0)% relative humidity to preserve any heat gain from the water immersion during post-testing.

Following post-immersion measurements, subjects underwent a head-up tilt test (HUTT). Subjects were instrumented with equipment to measure HR (3-lead ECG), brachial blood pressure (Propaq CS, Welch Allyn, Skaneateles Falls, NY, USA), respiratory rate (RR), and partial pressure of end-tidal carbon dioxide ($EtCO_2$, nasal cannula; Repsense, Nonin Medical, Inc., Plymouth, MN, USA). Mean arterial pressure (MAP) was calculated. Following a two-minute supine baseline, subjects were tilted to 70° for up to 20 minutes, until subjects showed physiological or subjective signs of pre-syncope (e.g., nausea, tunnel vision, sweating, dizziness), or the subject requested to stop. Pre-syncope was identified by the onset of rapid, progressive decreases in blood pressure, resulting in sustained systolic blood pressure < 80 mmHg and/or relative bradycardia. Subjects were then returned to a supine position for recovery. The main outcome measure of the HUTT was total time.

STATISTICAL ANALYSIS

Data are presented as mean (SD). Changes in physiological variables were analysed using repeated-measures analysis of variance with factors for condition and time. Data were examined for outliers using a robust nonlinear regression. Linear mixed-effects models were used when data were missing intermittently to preserve within-subject comparisons. Model residuals were assessed using Q-Q plots to confirm normalcy, and Greenhouse–Geisser corrections were applied when sphericity was violated. Post hoc comparisons were performed using Tukey-adjusted multiple comparisons. Paired Student's *t*-tests were used for selected pairwise comparisons where appropriate. Orthostatic

tolerance during HUTT was analysed using Kaplan–Meier survival analysis with log-rank and Gehan–Breslow–Wilcoxon tests. Statistical significance was set at $P \leq 0.05$.

Results

Characteristics for all subjects ($n = 8$, four men, four women) are presented in Table 1. Core temperature increased over time in both 38°C conditions ($P < 0.001$). Mean Tc differed by condition, with higher values in 38°C + Cool compared with 28°C and 33°C ($P = 0.009$ and $P = 0.003$, respectively) and in 38°C compared with 28°C and 33°C ($P = 0.04$ and $P = 0.03$, respectively). No difference was observed between 38°C and 38°C + Cool. Maximum Tc was higher in both 38°C conditions compared with 28°C and 33°C ($P < 0.01$), with no differences between 28°C and 33°C or between 38°C and 38°C + Cool (Figure 1). One subject exceeded the Tc safety threshold (38.5°C) during 38°C, and four subjects terminated early due to heat-related symptoms. During 38°C + Cool, three subjects requested to stop early without exceeding the Tc threshold of 38.5°C. Mean time to termination was 47 min 56 s (38°C) and 40 min 02 s (38°C + Cool) (Figure 1).

Heart rate increased over time in the 38°C and 38°C + Cool conditions (time, $P < 0.001$). Mean HR differed by condition ($P < 0.01$) with higher values in 38°C and 38°C + Cool compared to 28°C (both $P < 0.001$). Heart rate was higher in 38°C compared with 38°C + Cool ($P < 0.001$). Heart rate during 33°C was also higher than 28°C ($P < 0.001$). (Figure 1)

Mean T_{SK} increased over time in 33°C, 38°C, and 38°C + Cool ($P < 0.01$). Mean T_{SK} was higher in 38°C than 38°C + Cool ($P = 0.02$). Maximum T_{SK} was greater in both 38°C conditions compared with 28°C and 33°C ($P < 0.001$), with no difference between 38°C and 38°C + Cool. Helmet temperature increased over time in all conditions ($P < 0.001$). Mean helmet temperature differed across conditions ($P < 0.001$). Maximum helmet temperature was higher in 38°C and 38°C + Cool than 28°C and 33°C ($P < 0.001$), with no difference between 38°C conditions. (Figure 2)

Rate of perceived exertion increased over time ($P = 0.02$). Mean rate of perceived exertion was higher in 38°C + Cool than 33°C ($P = 0.04$). Thermal comfort decreased over time in 33°C, 38°C, and 38°C + Cool ($P < 0.001$). Comfort ratings were higher in 38°C and 38°C + Cool than in 28°C and 33°C ($P < 0.01$), with no difference between the 38°C conditions (Figure 3). Thermal sensation increased over time in all conditions ($P < 0.001$) and was higher in 38°C and 38°C + Cool compared with 28°C and 33°C ($P < 0.01$). Thermal sensation was also higher in 33°C versus 28°C ($P = 0.01$). Sweating sensation increased over time in 38°C and 38°C + Cool ($P < 0.001$) (Figure 3). Across conditions, mean sweating sensation was higher in 33°C, 38°C, and 38°C + Cool compared to 28°C ($P = 0.04$), with the greatest differences observed in 38°C ($P = 0.03$) and 38°C + Cool ($P = 0.04$) (Figure 4).

Table 1

Subject characteristics; BMI – body mass index; bpm – beats per minute; HR – heart rate; SD – standard deviation

Characteristic $n = 8$	Mean (SD)	Range
Age (y)	25 (4)	20–31
Height (cm)	176 (8)	165–188.5
Mass (kg)	79.9 (14.4)	59.2–97.7
BMI ($kg \cdot m^{-2}$)	25.6 (3.3)	21.8–30.9
Body Fat (%)	24 (10)	14–38
Resting HR (bpm)	66 (10)	53–82
Resting respiratory rate (bpm)	10 (2)	9–16
Resting diastolic blood pressure (mmHg)	112 (8)	100–122
Resting systolic blood pressure (mmHg)	70 (7)	60–76
VO_2max ($mL \cdot kg^{-1} \cdot min^{-1}$)	46.6 (9.2)	31.8–60.5

Figure 1

Heart rate (HR) in beats per minute (bpm) and core temperature (Tc) over time (A1 and B1, respectively); mean heart rate and core temperature between conditions (A2 and B2, respectively) (see text for explanation of conditions); data presented as mean with whiskers for SD

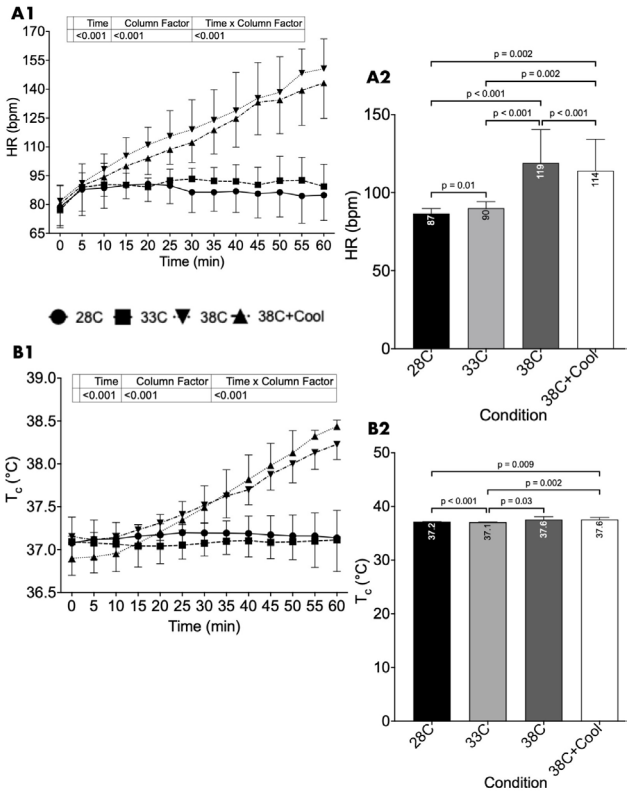


Figure 2

Mean skin temperature (T_{SK}) and helmet temperature (T_{Helmet}) over time (A1 and B1, respectively) and by condition (A2 and B2, respectively) (see text for explanation of conditions); data presented as mean with whiskers for SD

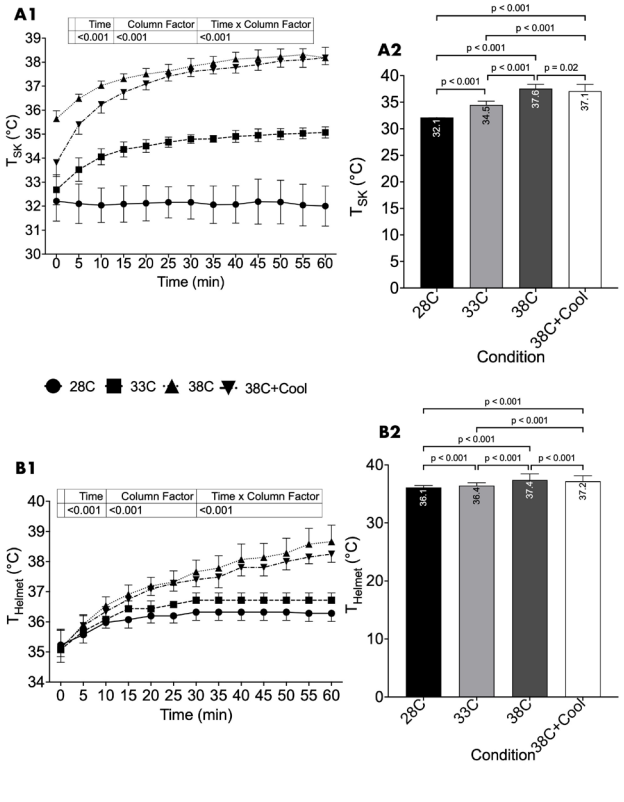
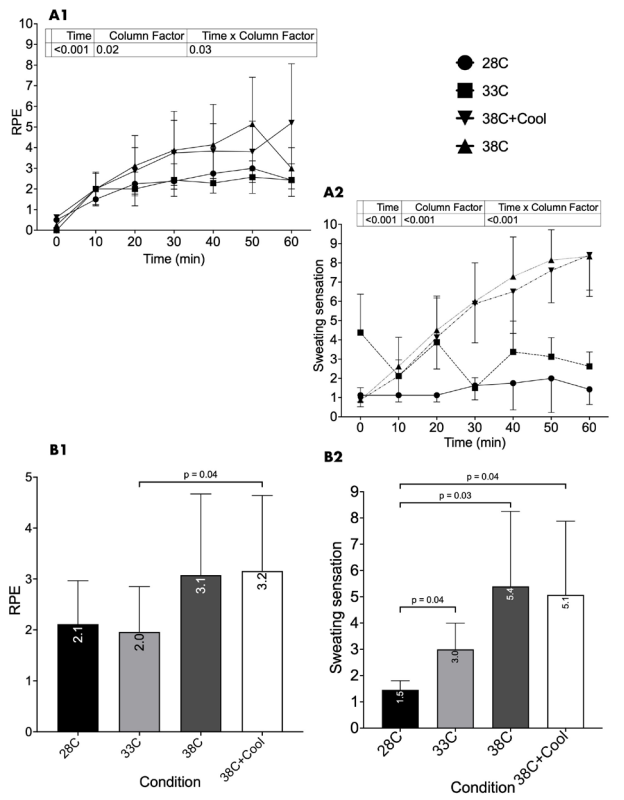


Figure 3

Sweating sensation, and rate of perceived exertion (RPE) over time (A1 and A2, respectively) and by condition (B1 and B2, respectively); (see text for explanation of conditions); data presented as mean with whiskers for SD



Core temperature differed across conditions during HUTT ($P < 0.001$), remaining higher in both 38°C conditions compared with 28°C and 33°C. Tc did not change over time during HUTT. Skin temperature decreased over time during HUTT in 33°C, 38°C, and 38°C + Cool ($P < 0.001$) and remained stable in 28°C. Mean T_{SK} during HUTT was higher in both 38°C conditions compared with 28°C and 33°C ($P < 0.001$), and higher in 33°C versus 28°C ($P = 0.005$), with no difference between 38°C conditions. Ambient temperature during HUTT did not differ between conditions. (22.1 (1.6)°C, $P = 0.98$). Heart rate increased over time in all conditions ($P < 0.001$). Mean HR was higher in 38°C and 38°C + Cool compared with 28°C and 33°C ($P < 0.001$), and higher in 33°C versus 28°C ($P < 0.001$), with no difference between 38°C conditions. Respiratory rate changed over time ($P < 0.001$). Mean RR was higher in 38°C + Cool and 28°C compared with 38°C and 33°C (all $P \leq 0.02$). End-tidal CO_2 did not change over time. Mean $EtCO_2$ was higher in 28°C compared with all other conditions ($P < 0.001$) and higher in 38°C + Cool compared with 38°C ($P < 0.001$) (Figure 5). Mean arterial pressure did not differ over time during the HUTT. However, MAP was lower during 38°C + Cool compared to 28°C ($P = 0.008$) and 33°C ($P = 0.02$). No other differences in MAP were found.

Survival analysis demonstrated differences in orthostatic tolerance across conditions (log-rank $P = 0.027$), with a significant monotonic trend across increasing thermal stress ($P = 0.003$). Early-phase differences between conditions were also observed (Gehan–Breslow–Wilcoxon $P = 0.035$), indicating reduced tolerance with increasing water temperature (Figure 5). Estimated sweat loss differed across conditions and was higher in 38°C and 38°C + Cool compared with 28°C and 33°C ($P < 0.01$). Body mass decreased from pre- to post-exposure in all conditions ($P < 0.001$), with no differences between conditions (Figure 6).

Discussion

Diving in warm water elicited substantial thermal and cardiovascular strain, characterised by progressive increases in core and skin temperature, elevated heart rate, and reduced orthostatic tolerance following immersion. These responses developed rapidly during 38°C exposures, were not meaningfully attenuated by 30 min of precooling, and persisted into the post-immersion period. Together, these findings indicate that encapsulated warm-water diving can produce uncompensable heat stress that compromises physiological stability during egress and recovery.[14]

The progressive rise in core temperature observed during 38°C conditions is consistent with uncompensable heat stress, in which environmental heat gain exceeds the capacity

Figure 4

Thermal comfort and thermal sensation over time (A1 and B1, respectively) and by condition (A2 and B2, respectively); (see text for explanation of conditions); data presented as mean with whiskers for SD

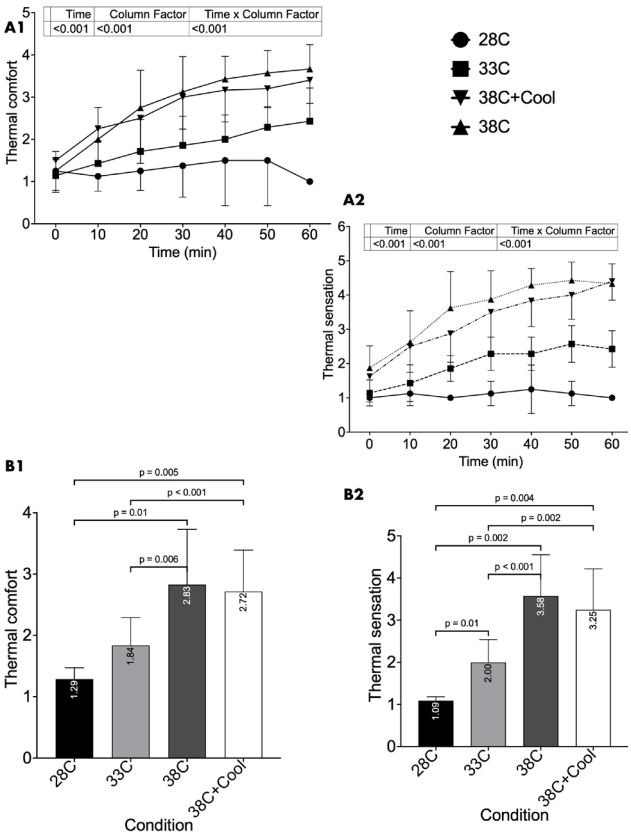
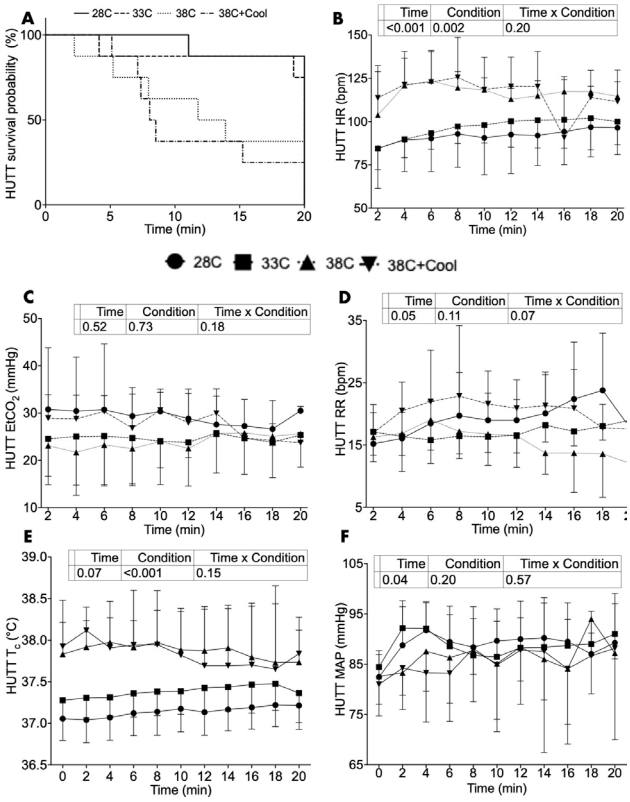


Figure 5

Survival analysis (A), heart rate (HR, B), end-tidal carbon dioxide ($EtCO_2$, C), respiratory rate (RR, D), core temperature (Tc, E), and mean brachial arterial pressure (MAP, F) during the head-up tilt test (HUTT) over time; data presented as mean with whiskers for SD

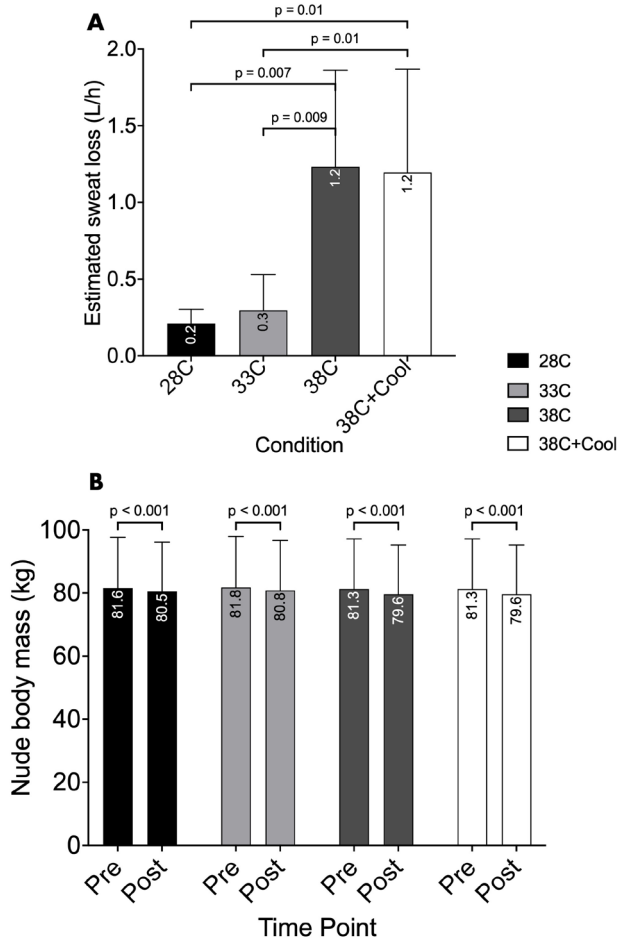


for heat dissipation. In encapsulated systems, evaporative heat loss is unavailable as a thermoregulatory pathway, leaving conductive heat exchange as the primary avenue for heat transfer.[15,16] The effectiveness of this pathway depends on whether the surrounding water provides a gradient for heat loss or instead imposes an external heat load. These findings extend established terrestrial models of heat stress to immersed encapsulated conditions, demonstrating that even moderate-intensity work is sufficient to produce clinically relevant hyperthermia when environmental heat flux is sustained.[3,4] Comparable core temperature elevations during warm-water immersion have been reported in both military and occupational diving contexts, consistent with known physiological responses to immersion in which combined environmental and equipment-related stressors alter thermoregulation and cardiovascular function.[17]

Heat exchange during immersion depends on the direction and magnitude of the temperature gradient between the body, protective ensemble, and surrounding water. In cooler water, conductive heat transfer favors heat loss from the body to the environment. As water temperature approaches skin temperature, this gradient narrows and conductive heat loss becomes progressively limited.[10] When water temperature exceeds that of skin or peripheral tissue, the temperature gradient may reverse, promoting environmental heat gain rather than heat dissipation. Under these conditions, increases in cutaneous blood flow will not improve heat loss and may instead facilitate heat transfer from the periphery toward the core.[18] In the present study, the 38°C water conditions likely approached or exceeded effective skin/peripheral temperatures, while encapsulation limited evaporative cooling, producing a thermal environment favoring progressive internal heat storage.[19,20]

A key finding of this study was the reduction in orthostatic tolerance following warm-water immersion, which worsened with increasing thermal strain, indicating a direct relationship between heat load and cardiovascular instability. This response is consistent with established mechanisms of heat-induced orthostatic intolerance, including sustained vasodilation, venous pooling, and reduced central blood volume.[21] The transition from immersion to upright posture represents a critical physiological stressor: hydrostatic pressure support for venous return is abruptly removed while peripheral vasodilation persists. Similar impairments in orthostatic tolerance have been observed following both passive heat exposure and exercise in the heat.[7,22] This combination likely exacerbates reductions in arterial pressure regulation and increases susceptibility to presyncope.[14] From an operational perspective, these

Figure 6
Estimated sweat loss during submersion (A) and change in body mass over the entire experimental visit (B) by condition; (see text for explanation of conditions); data presented as mean with whiskers for SD



findings identify the post-immersion period is a high-risk window for cardiovascular instability.

The head-up tilt test was included as a standardised post-immersion orthostatic challenge to assess cardiovascular stability during recovery. Total tilt tolerance time provides an integrated index of the ability to maintain arterial pressure under upright stress. This outcome is operationally relevant because divers must transition rapidly from hydrostatic support during immersion to unsupported upright posture during egress and post-dive recovery. In heat-stressed individuals, persistent cutaneous vasodilation, reduced central blood volume, and impaired cardiac filling can compromise arterial pressure regulation during orthostatic stress. The observed reduction in tilt-test tolerance following warmer water exposures supports the interpretation that residual thermal strain increases vulnerability to post-immersion cardiovascular instability.

Estimated sweat loss increased during warm-water exposure, exceeding 1.2 $L \cdot h^{-1}$ during the 38°C conditions compared with < 0.4 $L \cdot h^{-1}$ at cooler temperatures. Because sweat loss was estimated from changes in nude body mass, these values should be interpreted as estimates of whole-body fluid loss rather than direct measurements of sweat production. Despite this sustained increase in fluid loss, sweating likely contributed minimally to effective heat dissipation because the encapsulating suit created a high-humidity microenvironment at the skin surface, limiting evaporation and allowing sweat to accumulate within the suit rather than facilitating latent heat loss.[23] As a result, evaporative heat loss was effectively minimised, decoupling sweat production from heat dissipation.[24,25] Consistent with this interpretation, core temperature increased progressively throughout the 38°C conditions despite greater estimated sweat loss. Under these conditions, sweating represents a physiologically costly and functionally ineffective thermoregulatory response. Although plasma volume was not directly measured, greater fluid loss may have contributed to cardiovascular strain when combined with peripheral vasodilation during egress and recovery.

Perceptual responses paralleled the physiological changes observed during immersion. Ratings of perceived exertion, thermal sensation, and thermal discomfort increased with thermal strain and tracked closely with rising core temperature. Subjective measures of thermal stress are often closely associated with physiological heat storage and may provide important operational indicators of diver tolerance.[26] Several subjects approached the predetermined safety cutoff of 38.5°C under 38°C conditions. One subject exceeded the cutoff during the 38°C exposure, and four additional subjects terminated exercise early due to nausea, lightheadedness, or general discomfort. Similar symptoms were reported during the precooling condition, in which three subjects voluntarily terminated the exercise early despite Tc remaining below the cutoff. These findings emphasise the operational relevance of heat strain during submersion, which may limit performance prior to critical hyperthermia and may serve as practical indicators of declining physiological tolerance and increase the chances of syncope upon egress.

Precooling with the cooling vest used in the present study did not meaningfully attenuate thermal strain during 38°C immersion. Although precooling can increase heat-storage capacity in terrestrial environments, the magnitude and duration of cooling provided by this intervention may have been insufficient for the uncompensable heat load imposed by encapsulated warm-water diving. These findings should not be interpreted as evidence that all precooling strategies are ineffective. More aggressive, longer-duration, or combined cooling strategies may provide greater benefit and should be evaluated in future studies.

Collectively, these findings support an integrated model of physiological stress during encapsulated warm-water diving. Progressive hyperthermia and vasodilation increase cardiovascular strain during immersion, while hydrostatic pressure partially preserves central blood volume. Upon egress, the loss of hydrostatic support, combined with

persistent vasodilation and post-immersion hypotension, reduces orthostatic tolerance and elevates the risk of syncope.[27,28] This sequence provides a mechanistic explanation for operational observations of post-dive weakness and collapse, highlighting the transition from immersion to upright posture as a critical vulnerability. Operationally, these findings have direct implications for diver safety and exposure management. Thermal strain developed rapidly in warm-water conditions and is not fully mitigated by protective equipment or precooling. Importantly, risk extends beyond immersion into the immediate recovery period. Exposure guidelines should incorporate both in-water thermal limits and post-immersion recovery strategies. Interventions aimed at reducing heat storage, enhancing post-dive cooling, or mitigating venous pooling may be necessary to improve safety during warm-water operations.

LIMITATIONS

Several limitations should be acknowledged. The laboratory environment may not fully replicate operational diving conditions, including variable workloads, mission duration, and environmental exposures such as current, equipment variability, and task complexity. Additionally, direct measurements of stroke volume, central blood volume, and cerebral perfusion were not obtained, limiting mechanistic resolution. As a result, interpretations of cardiovascular strain and post-immersion orthostatic tolerance are based on indirect indices of haemodynamic function rather than direct quantification of cardiac preload, cerebral blood flow, or regional perfusion. The absence of these measures precludes definitive identification of the relative contributions of reduced central blood volume, altered cardiac function, and cerebrovascular regulation to the observed responses.

Conditions were separated by at least 72 h to reduce carryover effects. However, this interval may not have fully eliminated residual physiological or perceptual effects from prior exposures. Although trial order was randomised and counterbalanced, the possibility of carryover effects cannot be excluded. Hydration status was addressed through standardised pre-visit instructions and pre-exposure urine specific gravity screening but was not directly controlled through prescribed fluid intake or plasma volume assessment. Because hydration status can influence thermoregulatory strain, cardiovascular responses, blood volume, and orthostatic tolerance, residual differences in hydration status may have contributed to interindividual variability in the observed responses.

Mean skin temperature was estimated using a four-site model and iButton thermistors, which may not fully capture regional variation in skin temperature under an encapsulating drysuit. In addition, iButtons are larger than thin surface thermistors and may have locally altered conductive heat exchange between the skin, base layer, and suit. Therefore, skin temperature data should be interpreted as estimates of regional thermal responses rather than direct measures of skin heat flux. Core temperature was measured using telemetric capsules that were either ingested before testing or inserted rectally immediately before exposure. Although this approach allowed continuous temperature monitoring during immersion, differences in capsule location may have introduced variability in the measured core temperature response.

Conclusions

Diving in warm water induces uncompensable heat stress characterised by progressive hyperthermia, peripheral vasodilation, and increased cardiovascular strain that are not mitigated by precooling. These responses persist beyond immersion and reduce orthostatic tolerance, indicating impaired central haemodynamic stability. The transition to upright represents a period of increased physiological vulnerability and risk of syncope. Thermal load is a primary limitation to diver safety and performance in warm-water environments. Mitigation strategies should address both in-water heat strain and post-immersion orthostatic tolerance.

References

1 Long ET, O'Connor PE, Liberatore TC. Development of exposure guidance for warm water diving: volume 1—physiology and endurance. Research Report NEDU TR 03-11. Panama City (FL): Navy Experimental Diving Unit; 2003.
2 Long ET, O'Connor PE, Liberatore TC. Development of exposure guidance for warm water diving: volume 2—system for investigation of divers' behavior at depth (SINDBAD) and Special Operations Forces (SOF) mission-related performance measures (MRPM). Research Report NEDU TR 03-15. Panama City (FL): Navy Experimental Diving Unit; 2003.
3 González-Alonso J, Teller C, Andersen SL, Jensen FB, Hyldig T, Nielsen B. Influence of body temperature on the development of fatigue during prolonged exercise in the heat. J Appl Physiol (1985). 1999;86:1032–9. doi: 10.1152/jappl.1999.86.3.1032. PMID: 10066720.
4 Nybo L, Nielsen B. Hyperthermia and central fatigue during prolonged exercise in humans. J Appl Physiol (1985). 2001;91:1055–60. doi: 10.1152/jappl.2001.91.3.1055. PMID: 11509498.
5 Tyler CJ, Sunderland C. Cooling the neck region during exercise in the heat. J Athl Train. 2011;46:61–8. doi: 10.4085/1062-6050-46.1.61. PMID: 21214352. PMCID: PMC3017491.
6 Seeley AD, Giersch GEW, Charkoudian N. Post-exercise body cooling: skin blood flow, venous pooling, and orthostatic intolerance. Front Sports Act Living. 2021;3:658410. doi: 10.3389/fspor.2021.658410. PMID: 34079934. PMCID: PMC8165173.
7 Francisco MA, Colbert C, Larson EA, Sieck DC, Halliwill JR, Minson CT. Hemodynamics of postexercise versus post-hot water immersion recovery. J Appl Physiol (1985). 2021;130:1362–72. doi: 10.1152/japplphysiol.00260.2020. PMID: 33630675. PMCID: PMC8354820.
8 Halliwill JR. Mechanisms and clinical implications of post-exercise hypotension in humans. Exerc Sport Sci Rev.

2001;29:65–70. doi: 10.1097/00003677-200104000-00005. PMID: 11337825.
9 Amin SB, Hansen AB, Mugele H, Simpson LL, Marume K, Moore JP, et al. High-intensity exercise and passive hot water immersion cause similar postintervention changes in peripheral and cerebral shear. J Appl Physiol (1985). 2022;133:390–402. doi: 10.1152/japplphysiol.00780.2021. PMID: 35708700.
10 Crandall CG, González-Alonso J. Cardiovascular function in the heat-stressed human. Acta Physiol (Oxf). 2010;199:407–23. doi: 10.1111/j.1748-1716.2010.02119.x. PMID: 20345414. PMCID: PMC3496876.
11 Worley ML, Wheelock CE, Stooks J, Schwob JR, Bratta B, Leddy JJ, et al. Hot water immersion increases internal carotid artery shear rate but does not alter intracranial vascular reactivity to carbon dioxide. Appl Physiol Nutr Metab. 2025;50:1–17. doi: 10.1139/apnm-2024-0048. PMID 40116297.
12 Gibbons TD, Ainslie PN, Thomas KN, Wilson LC, Akerman AP, Donnelly J, et al. Influence of the mode of heating on cerebral blood flow, non-invasive intracranial pressure and thermal tolerance in humans. J Physiol. 2021;599:1977–96. doi: 10.1113/JP280970. PMID: 33586133.
13 Ramanathan NL. A new weighting system for mean surface temperature of the human body. J Appl Physiol. 1964;19:531–3. doi: 10.1152/jappl.1964.19.3.531. PMID: 14173555.
14 Pollock NW, Lippmann J, Pearn J, Hayman J. Arterial dissection in scuba divers: a potential adverse manifestation of the physiological effects of immersion. Diving Hyperb Med. 2024;54:188–95. doi: 10.28920/dhm54.3.188-195. PMID: 39288923. PMCID: PMC11659074.
15 American College of Sports Medicine; Sawka MN, Burke LM, Eichner ER, Maughan RJ, Montain SJ, Stachenfeld NS. American college of sports medicine position stand. Exercise and fluid replacement. Med Sci Sports Exerc. 2007;39:377–90. doi: 10.1249/mss.0b013e31802ca597. PMID: 17277604.
16 Proulx CI, Ducharme MB, Kenny GP. Effect of water temperature on cooling efficiency during hyperthermia in humans. J Appl Physiol (1985). 2003;94:1317–23. doi: 10.1152/japplphysiol.00541.2002. PMID: 12626467.
17 Tetzlaff K. Pulmonary physiology and medicine of diving. Semin Respir Crit Care Med. 2023;44:705–18. doi: 10.1055/s-0043-1770065. PMID: 37369217.
18 Coyle EF, González-Alonso J. Cardiovascular drift during prolonged exercise: new perspectives. Exerc Sport Sci Rev. 2001;29:88–92. doi: 10.1097/00003677-200104000-00009. PMID: 11337829.
19 Wilson TE, Crandall CG. Effect of thermal stress on cardiac function. Exerc Sport Sci Rev. 2011;39:12–7. doi: 10.1097/JES.0b013e318201eed6. PMID: 21088607. PMCID: PMC3076691.
20 Tso JV, Powers JM, Kim JH. Cardiovascular considerations for scuba divers. Heart. 2022;108:1084–9. doi: 10.1136/heartjnl-2021-319601. PMID: 34670825. PMCID: PMC9018859.
21 Schlader ZJ, Wilson TE, Crandall CG. Mechanisms of orthostatic intolerance during heat stress. Auton Neurosci. 2016;196:37–46. doi: 10.1016/j.autneu.2015.12.005. PMID: 26723547. PMCID: PMC5607624.
22 Halliwill JR, Buck TM, Lacewell AN, Romero SA. Postexercise hypotension and sustained postexercise vasodilatation: what happens after we exercise? Exp Physiol. 2013;98:7–18. doi: 10.1113/expphysiol.2011.058065. PMID: 22872658.
23 Gagnon D, Crandall CG. Sweating as a heat loss thermoeffector. Handb Clin Neurol. 2018;156:211–32. doi: 10.1016/B978-0-444-63912-7.00013-8. PMID: 30454591.
24 Beigtan M, Gonçalves M, Weon BM. Heat transfer by sweat droplet evaporation. Environ Sci Technol. 2024;58:6532–9. doi: 10.1021/acs.est.4c00850. PMID: 38538556. PMCID: PMC11025549.
25 Candas V, Libert JP, Vogt JJ. Sweating and sweat decline of resting men in hot humid environments. Eur J Appl Physiol Occup Physiol. 1983;50:223–34. doi: 10.1007/BF00422161. PMID: 6681754.
26 Flouris AD, Schlader ZJ. Human behavioral thermoregulation during exercise in the heat. Scand J Med Sci Sports. 2015;25 Suppl 1:52–64. doi: 10.1111/sms.12349. PMID: 25943656.
27 Keller DM, Low DA, Wingo JE, Brothers RM, Hastings J, Davis SL, et al. Acute volume expansion preserves orthostatic tolerance during whole-body heat stress in humans. J Physiol. 2009;587:1131–9. doi: 10.1113/jphysiol.2008.165118. PMID: 19139044. PMCID: PMC2673780.
28 Wilson TE, Brothers RM, Tollund C, Dawson EA, Nissen P, Yoshiga CC, et al. Effect of thermal stress on Frank-Starling relations in humans. J Physiol. 2009;587:3383–92. doi: 10.1113/jphysiol.2009.170381. PMID: 19417092. PMCID: PMC2727045.

Data availability statement

Deidentified data are available upon reasonable request to the corresponding author and after approval by the Institutional Review Board.

Conflicts of interest and funding

This work was supported by the Naval Sea Systems Command's (NAVSEA) Diving Systems and Buoyancy Development (DSBD) under contract [N00024-24C-4302]. No conflicts of interest were declared.

Submitted: 23 April 2026
Accepted after revision: 25 July 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

Maintained critical flicker fusion frequency after acute hyperbaric hyperoxia at 140 kPa

Jochen D Schipke[1], Thomas Muth[2], Anne-Kathrin Brebeck[3], Dominik von Hertlein[4], Fabian N Möller[5,6,7]

[1] *Research Group Experimental Surgery, University Hospital Düsseldorf, Düsseldorf, Germany*
[2] *Institute of Occupational, Social, Environmental Medicine, Faculty of Medicine, Heinrich-Heine-University Düsseldorf, Düsseldorf, Germany*
[3] *Artemis MVZ Frankfurt, Frankfurt/Main, Germany*
[4] *German Sport University Cologne, Department of Psychology, Cologne, Germany*
[5] *University of Oregon, Department of Human Physiology, Eugene, OR, USA*
[6] *Massachusetts Institute of Technology, Cambridge, MA, USA*
[7] *German Sport University Cologne, Department of Exercise Physiology, Cologne, Germany*

Corresponding author: *Professor Jochen Schipke, Research Group Experimental Surgery, University Hospital, Moorenstrasse 5, D-40545 Düsseldorf, Germany*
ORCiD: *0000-0002-1747-5657*
j.schipke@gmx.org

Keywords
Central nervous system; Diving; HRV; Hyperbaric oxygen; Narcosis; Oxygen; Scuba diving

Abstract
(Schipke JD, Muth T, Brebeck A-K, von Hertlein D, Möller FN. Maintained critical flicker fusion frequency after acute hyperbaric hyperoxia at 140 kPa. Diving and Hyperbaric Medicine. 2026 30 September;56(3):280–285. doi: 10.28920/dhm56.3.280-285. PMID: 42743567.)

Introduction: In diving medicine, critical flicker fusion frequency (cFFF) has been employed to assess alertness under varying ambient pressures and gas compositions, yet the effects of elevated oxygen partial pressure on cFFF performance underwater remain inconsistently characterised. This investigation aimed to assess the impact of breathing 140 kPa oxygen on underwater cFFF performance, a pressure level corresponding to established safety limits and common closed-circuit rebreather setpoints during operational diving.

Methods: Nineteen experienced divers (10 females; mean age 23.8 [SD 3.0] years) completed cFFF assessments under three conditions: breathing air at poolside (21 kPa O_2), breathing air at 4 m depth (29 kPa O_2), and breathing 100% oxygen at 4 m depth (140 kPa O_2). Concurrent measurements included heart rate, respiratory rate, and heart rate variability indices representing autonomic function; specifically the LF/HF ratio indicating sympathetic activity and RMSSD reflecting parasympathetic activity.

Results: Mean cFFF breathing air at poolside was 37.9 (SD 2.9) Hz and breathing 100% oxygen at 4 m was 37.2 (SD 2.5) Hz, indicating that mild hyperoxia at 140 kPa does not impair cortical processing speed or visual temporal resolution. However, oxygen breathing produced notable cardiovascular and respiratory effects: heart rate decreased from 70.9 (10) to 67.8 (11.6) beats per minute, respiratory rate declined from 15.2 (2.7) to 13.4 (1.7) breaths per minute, the LF/HF ratio decreased from 5.9 (1.4) to 3.1 (2.0), and RMSSD increased from 73.7 (10.8) to 97.0 (6.6) milliseconds. These physiological changes indicate reduced sympathetic activity and enhanced parasympathetic tone during oxygen breathing.

Conclusions: The findings suggest that breathing 140 kPa oxygen during shallow water immersion maintains neural integrity as measured by cFFF while beneficially modulating autonomic regulation toward parasympathetic dominance. This autonomic shift resembles known effects of both submersion and moderate hyperoxia, supporting the safety profile of current oxygen exposure protocols in recreational and technical diving.

Introduction

Critical flicker fusion frequency (cFFF) is the frequency at which an intermittently illuminated stimulus is perceived as steady and has been discussed as an indicator of cortical arousal and visual-cognitive processing,[1,2] with a higher cFFF associated with better performance. Conceptually, flicker fusion occurs when temporal modulation exceeds the visual system's temporal resolution, yielding a perception of continuous light above this threshold.[2,3] Previous studies reported significant variation in individual cFFF thresholds for healthy adults, i.e., 35–90 Hz, reflecting methodological and stimulus-dependent differences,[2,4] respectively.

In diving and hyperbaric research, cFFF has been applied to assess the effects of elevated nitrogen and oxygen gas pressures on arousal and cognition,[5–8] though its suitability without additional markers remains debated.[9] Furthermore,

findings on the effects of increased inspiratory oxygen pressures, from increased ambient pressure or oxygen fraction in the breathing gas, remain equivocal. In chamber studies, oxygen produced dose-dependent changes in cFFF with no effects at 70 kPaO_2, lower cFFF at 140 kPa (i.e., indicating a negative effect), and a recovery to baseline at 280 kPa. This response is similar to the dose-dependent physiological effects of O_2, from the benefits of increased oxygenation to oxygen toxicity, consistent with overlapping mechanisms of cFFF modulation and O_2 toxicity.[10] When high O_2 and N_2 pressures are combined, like when breathing air at 600 kPa ambient pressure (pO_2 ≈120 kPa), reaction times were worse, but cFFF values increased by 2.5 (SD 2.8)%, indicating solely impaired performance.[11] In contrast, Lafère et al. reported a transient positive effect with increased cFFF values after breathing 160 kPa O_2 at 400 kPa ambient pressure.[12] Values returned to baseline within 20 min, suggesting an early O_2-linked enhancement of cFFF that dissipates over time. Other work found that 100% O_2 at 100 and 280 kPa decreased cFFF, i.e., had a negative effect on it, with no change at 600 kPa, leading to questions about cFFF's sensitivity to gas narcosis.[9]

The equivocal results from chamber experiments are echoed in field studies. After no-decompression, depth-matched dives to 21 m with 32% O_2 in the breathing gas (i.e., Nx32), divers reported less fatigue than after air dives. Furthermore, cFFF increased by 4% with Nx32 and decreased by 6% with air compared to pre-dive, indicating gas-dependent neural modulation [5]. Breathing gases with increased pO_2 have also been associated with benefits for sustaining alertness and short-term memory when compared to breathing air, but cFFF was not assessed in these studies.[13] During a 20-min air dive to 33 m (pO_2 ≈90 kPa), cFFF acutely increased from the 100% control to 104 (5)% at depth, then decreased to 94 (4)% over ten minutes at depth, with deficits persisting 30 min post-surfacing.[14] Data from closed-circuit rebreather (CCR) dives remain limited, with one study reporting slight increases in alertness/arousal during 50 m dives on air, trimix, or heliox, implicating the roles of nitrogen and oxygen – alone or combined – in modulating neuronal excitability.[6]

Given divergent reports on oxygen's neurophysiological effects under pressure, and the potential for dose-dependent excitatory versus depressive responses, we aimed to isolate the specific impact of oxygen on CFFF by using 100% oxygen during immersion to 140 kPa, replicating an established O_2-exposure limit and rebreather setpoint, while minimising the influence of N_2.

Methods

This study was approved by the Ethics Committee of the German Sport University Cologne (100/2023), following the Declaration of Helsinki and its amendments.[15]

PARTICIPANTS

The study included 19 healthy participants (mean age of 23.8 (SD 3.2) years; 10 females), an average height of 175 (22) cm, and an average body mass of 72 (14) kg. Calculated body mass index was 23.4 (1.4) kg·m^{-2}. All participants were certified divers, medically cleared to dive, and had normal vision. Participants were asked to refrain from alcohol and caffeine intake on the day of testing; however, compliance was self-reported and was not formally controlled.

EQUIPMENT AND MEASUREMENTS

Data collection was conducted in a well-lit indoor pool (20 m × 20 m × 5 m) with water and air temperatures at ~27 to 30°C. The diving gear was provided and consisted of a 3 mm wetsuit, a 10 L steel cylinder, a commercially available buoyancy control device (BCD), a breathing regulator, mask, and fins. Oxygen was supplied with a separate system at depth. Some participants used personal masks, and clear vision was ensured for all trials. The participants were equipped with a Polar V800 watch and H7 chest belt (Polar Electro Oy, Kempele, FI).

A manually operated flicker device was employed in this study due to its feasibility for underwater testing (Scaleo flicker, RP-Engineering GmbH, D-Esslingen). The device allowed manual adjustment of flicker frequency. The light source was an LED with a color temperature of 8,000 K (i.e., blueish light). The individual cFFF value was displayed to one decimal place on a readout facing the test conductor only. The frequency display was inactive during testing and activated exclusively by a button press directed toward the conductor, ensuring that participants were never exposed to their frequency scores at any point during the experiment.

PROTOCOL

All participants received a comprehensive explanation of the experimental protocol prior to the binocular testing. Here, they were presented with demonstrations of both flickering and steady light conditions. The protocol consisted of 10 min at the pool bottom, breathing either air or 100% O_2 via an open-circuit underwater breathing apparatus.

All measurements were conducted with participants kneeling on the pool bottom. The stimulus frequency was increased from 20 Hz to the individual cFFF threshold, signaled by the participant. All measures were repeated three times by a single test conductor. In cases where participants were uncertain about their decision, or when a clear outlier was observed, all three measurements were repeated. As the distance to the light source was reported to affect cFFF,[16] all experiments maintained a fixed distance of 1.5 m between the CFFF device and the participants' eyes.

DATA PREPARATION

Cardiac parameters were analysed using Kubios 4.03 (Kubios Oy, HRV Scientific 4.1.0, Kuopio, FI). From the HR signal, the respiratory rate (RespR) and two established measures of heart rate variability (HRV) were derived: low frequency:high frequency ratio (LF/HF),[17] representing sympathetic activity, and root mean square of successive differences (RMSSD), representing parasympathetic activity.

The cFFF values from the baseline session (surface, non-immersed, 21 kPa O_2), at 4 m water depth (29 kPa O_2), and breathing 100% O_2 at 4 m (140 kPa O_2) are presented as means (standard deviation) from three consecutive measurements conducted at each time point.

STATISTICS

Differences between the three experimental conditions were analysed using Wilcoxon signed-rank tests for paired samples (SPSS 29.0.1). Pairwise comparisons of variables were conducted between the 21 kPa O_2 and 29 kPa O_2 conditions, and between the 29 kPa O_2 and 140 kPa O_2 conditions. All tests were two-tailed, with the significance level set at $P < 0.05$. In accordance with the American Statistical Association's guidance on careful interpretation of *P*-values and statistical significance,[18] effect sizes are presented using Cohen's d [19]. Effect size estimates were reported to aid interpretation of non-significant findings, particularly where physiological relevance may exist despite limited statistical power, and to aid comparisons to the literature.

Results

BASELINE MEASUREMENTS (21 kPa O_2, dry)

During this step (no masks worn), critical flicker fusion frequency (cFFF) was 37.1 (2.2) Hz and was thus within the range described for a similar age group [4]. The participants had a mean heart rate (HR) of 76 (11), a respiratory rate (RespR) of 14 (2), LF/HF equalled 11.0 (7.1), and RMSSD was 53.5 (39.7) ms.

MAIN SUBMERSION SESSION (29 vs. 140 kPa O_2)

No significant differences for cFFF thresholds were detected after 10 min breathing either air or oxygen at 4 m water depth (Δ -0.7 Hz). However, heart rate was lower after breathing oxygen compared to air (Δ -3 bpm). For respiration rate, no statistically significant difference was detected based on *P*-value analysis; however, effect size estimates suggested a substantial reduction during oxygen breathing (Δ -2 breaths·min^{-1}), indicating a potentially meaningful physiological change that may not have reached statistical significance. The LF/HF ratio was markedly decreased under oxygen, reflecting a decreased sympathetic dominance. RMSSD did not differ significantly according to *P*-value criteria, but effect size estimates indicated a moderate increase by O_2-breathing (Δ -23 ms), suggesting enhanced parasympathetic activity (see Table 1 and Figure 1).

Discussion

The critical flicker fusion frequency (cFFF), originally described by Braunstein (1903),[20] defines the temporal resolution of the visual system, with higher cFFF thresholds reflecting greater cortical processing speed and alertness. Under conditions of elevated breathing gas pressures, such as during diving or hyperbaric exposure, cFFF has been widely applied as a functional index of cortical activity and vigilance.[21] However, results regarding the influence of hyperbaric oxygen on cFFF remain inconsistent.[9,10,12,22,23]

Similarly, studies investigating oxygen's neural effects have produced divergent findings. Hyperbaric oxygen can act either depressively or excitatorily, producing narcotic[24,25] or stimulating[26] effects. Reports vary from cognitive impairment[27,28] to increased neuronal excitability.[8,29,30] Some authors have suggested that these opposite outcomes may represent different phases of oxygen toxicity, from an initial hyperexcitability to depression,[10] while others observed no measurable changes.[31,32]

In our previous normobaric study using the same cFFF measurement system, 5 min of oxygen breathing at the poolside (i.e., 100 kPa pO_2) had no impact on cFFF compared with air.[21] The present investigation extends this finding to underwater mild hyperbaric conditions and the frequently cited limit and rebreather setpoint for oxygen of 140 kPa. However, no effects for breathing 140 kPa O_2 on cFFF were observed relative to air breathing at identical pressure and time.

The absence of a cFFF change at 140 kPa O_2 agrees with Vrijdag et al. (2020), who reported similarly unchanged cFFF in a hyperbaric chamber breathing oxygen at 142 kPa.[9] Yet, other research demonstrated dose-dependent alterations in neuronal excitability, where cFFF was reduced at 140 kPa.[10] The reasons for these discrepancies remain unclear. Psychological stress and sleep deprivation, which are known to decrease cFFF,[2,33,34] are unlikely, as the study participants were well-rested and tested under conditions with minimal stress. Likewise, temperature differences between chamber and aquatic environments (~27 to 30°C) are unlikely confounders, as an increase in ambient temperature has been shown to increase, not lower, cFFF.[35,36] Responses to elevated pO_2 and their potential effects on CFFF appear to be highly individual, as reflected by the pronounced inter-individual variability in CFFF observed in the present dataset (33.2–43.4 Hz). Comparisons across studies are further complicated by differences in exposure duration, which ranges from as little as five minutes to 30 minutes in the existing literature, and by the heterogeneity of study populations, spanning recreational divers, technical

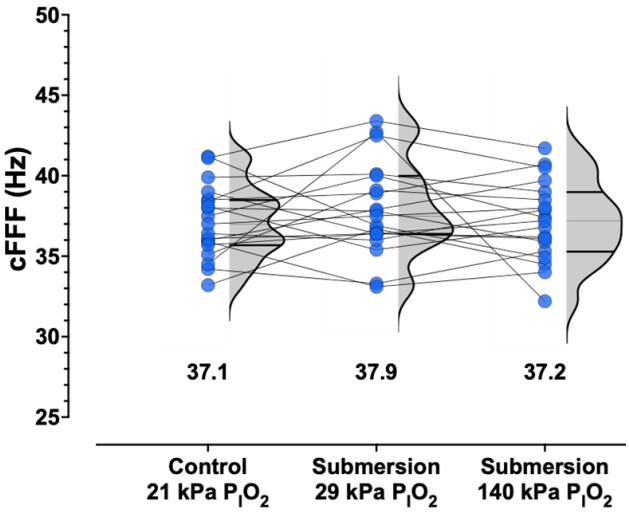
Table 1

Critical flicker fusion frequencies (cFFF), heart rate (HR), respiratory rate (RespR), root mean square of successive differences (RMSSD), and low frequency:high frequency ratio (LF/HF) during breathing air at the pool side (21 kPa O_2), at 4 m (29 kPa O_2) or breathing 100% O_2 (140 kPa O_2); data are presented as mean (SD). Cohen's effects are: small (0.2), medium (0.5), and large (0.8 or greater)

Parameter	cFFF (Hz)	HR (min^{-1})	RespR (min^{-1})	LF/HF	RMSSD (ms)
21 kPa O_2	37.1 (2.2)	75 (11)	15 (2)	11.0 (7.1)	53.3 (39.7)
29 kPa O_2	37.9 (2.9)	71 (10)	15 (3)	5.9 (1.4)	73.7 (10.8)
140 kPa O_2	37.2 (2.5)	68 (12)	13 (2)	3.1 (2.0)	97.0 (6.6)
P	0.59	0.03	0.13	0.03	0.95
Cohen's d	0.13	0.77	0.89	1.40	0.28

Figure 1

Critical flicker fusion frequencies (cFFF) during air breathing at the pool side (21 kPa O_2) and at 4 m water depth breathing air (29 kPa O_2) or 100% O_2 (140 kPa O_2); data are depicted as individual mean values with connecting lines and violin plots with median and interquartile ranges to show data distribution. Group means are indicated in bold within the figure



divers, and military or hyperbaric personnel with regular oxygen exposure. Both factors likely contribute to the variability in reported cFFF responses and should be considered when interpreting divergent findings.

Apart from cFFF, oxygen breathing at 4 m depth produced notable physiological effects, with a decreased heart rate and respiratory rate compared with air. This likely reflects enhanced autonomic balance, as parasympathetic activity (RMSSD) increased and sympathetic modulation (LF/HF ratio) decreased, consistent with the diving response. These findings align with data from professional divers breathing normobaric oxygen, where similar shifts in autonomic tone were observed.[37] It is therefore worth emphasising that both submersion[38,39] and moderate hyperoxia at 140 kPa pO_2 appear to promote parasympathetic dominance.

Our chosen protocol mirrors setpoints commonly used in closed-circuit rebreather diving during the bottom phase[40,41] and in decompression procedures, where 100% oxygen at ~140 kPa improves nitrogen offgasing driving pressure.[42] This pO_2 also corresponds to the widely accepted safety threshold for operational diving.[43,44]

Taken together, our findings show no change in cFFF from mild hyperbaric hyperoxia at 140 kPa over 10 min duration, while parasympathetic activity is increased. Future research should aim to disentangle potential interactions between oxygen and nitrogen under hyperbaric conditions and clarify the thresholds at which oxygen-induced effects transition from neurofunctional neutrality to excitatory or narcotic effects.

LIMITATIONS

The sample comprised young, well-rested certified divers, limiting generalisability to broader diving populations, including older, less experienced, or fatigued individuals.

Only a single oxygen partial pressure (140 kPa) and exposure duration (10 min) were tested, precluding assessment of dose-response relationships or effects during longer exposures. However, these parameters closely mimic the real-world diving conditions of decompression stops at the tested ambient and inspiratory pressure. The controlled pool environment with stationary participants does differ from actual diving scenarios involving navigation, task performance, and variable conditions, though this approach enabled precise physiological measurements under standardised conditions.

Finally, we cannot fully explain the discrepancy between our maintained cFFF thresholds at 4 m depth breathing air or O_2 and the decreased cFFF thresholds reported by others at the same ambient pressure.[10] Environmental differences between the aquatic and dry hyperbaric chamber settings – particularly concerning posture and temperature – may have contributed to these divergent findings.

Conclusions

Breathing 140 kPa O_2 at 4 m depth maintains cFFF stability, suggesting no cortical depression, while a lower heart rate suggests parasympathetic upregulation. A trend toward reduced respiratory rate was also observed, though this did not reach statistical significance and should be interpreted with caution: reduced ventilation under hyperoxia may promote hypercapnia, which is a recognised potentiator of CNS oxygen toxicity. This validates standard setpoints for bottom time and decompression without neurofunctional risk, but calls for more pooled data on O_2 / N_2 interactions at depth to refine protocols.

References

1 Mankowska ND, Marcinkowska AB, Waskow M, Sharma RI, Kot J, Winklewski PJ. Critical flicker fusion frequency: A narrative review. Medicina (Kaunas). 2021;57(10):1096. doi: 10.3390/medicina57101096. PMID: 34684133. PMCID: PMC8537539.

2 Muth T, Schipke JD, Brebeck AK, Dreyer S. Assessing critical flicker fusion frequency: Which confounders? A narrative review. Medicina (Kaunas). 2023;59(4):800. doi: 10.3390/medicina59040800. PMID: 37109758. PMCID: PMC10141404.

3 Seitz AR, Nanez JE Sr, Holloway SR, Watanabe T. Perceptual learning of motion leads to faster flicker perception. PLoS ONE. 2006;1(1):e28. doi: 10.1371/journal.pone.0000028. PMID: 17183655. PMCID: PMC1762365.

4 Gautam D, Vinay D. A study of critical flicker fusion threshold among smartphone users. International Journal of Current Microbiology and Applied Sciences. 2020;9(3):2381–6. doi: 10.20546/ijcmas.2020.903.271.

5 Lafère P, Balestra C, Hemelryck W, Donda N, Sakr A, Taher A, et al. Evaluation of critical flicker fusion frequency and perceived fatigue in divers after air and enriched air nitrox diving. Diving Hyperb Med. 2010;40:114–8. PMID: 23111908. [cited 2026 Feb 1]. Available from: https://dhmjournal.com/images/IndividArticles/40Sept/Lafere_dhm.40.3.114-118.pdf.

6 Rocco M, Pelaia P, Di Benedetto P, Conte G, Maggi L, Fiorelli S, et al. Inert gas narcosis in scuba diving, different gases different reactions. Eur J Appl Physiol. 2019;119:247–55. doi: 10.1007/s00421-018-4020-y. PMID: 30350155.

7 Piispanen WW, Lundell RV, Tuominen LJ, Räisänen-Sokolowski AK. Assessment of alertness and cognitive performance of closed circuit rebreather divers with the critical flicker fusion frequency test in Arctic diving conditions. Front Physiol. 2021;12:722915. doi: 10.3389/fphys.2021.722915. PMID: 34447319. PMCID: PMC8384076.

8 Sharma RI, Mankowska ND, Marcinkowska AB, Winklewski PJ, Kot J. Critical flicker fusion frequency results during oxygen decompression in standard HBOT session - observational study. Int Marit Health. 2024;75:167–76. doi: 10.5603/imh.99568. PMID: 39411987.

9 Vrijdag XC, van Waart H, Sleigh JW, Balestra C, Mitchell SJ. Investigating critical flicker fusion frequency for monitoring gas narcosis in divers. Diving Hyperb Med. 2020;50:377–85. doi: 10.28920/dhm50.4.377-385. PMID: 33325019. PMCID: PMC7872789.

10 Kot J, Winklewski PJ, Sicko Z, Tkachenko Y. Effect of oxygen on neuronal excitability measured by critical flicker fusion frequency is dose dependent. J Clin Exp Neuropsychol. 2015;37:276–84. doi: 10.1080/13803395.2015.1007118. PMID: 25715640.

11 Tikkinen J, Parkkola K, Siimes MA. Reaction test revealed impaired performance at 6.0 atm abs but not at 1.9 atm abs in professional divers. Undersea Hyperb Med. 2013;40:33–9. PMID: 23397866.

12 Lafère P, Hemelryck W, Germonpré P, Matity L, Guerrero F, Balestra C. Early detection of diving-related cognitive impairment of different nitrogen-oxygen gas mixtures using critical flicker fusion frequency. Diving Hyperb Med. 2019;49:119–26. doi: 10.28920/dhm49.2.119-126. PMID: 31177518. PMCID: PMC6704008.

13 Brebeck AK, Deussen A, Schmitz-Peiffer H, Range U, Balestra C, Cleveland S, et al. Effects of oxygen-enriched air on cognitive performance during SCUBA-diving - an open-water study. Res Sports Med. 2017;25:345–56. doi: 10.1080/15438627.2017.1314289. PMID: 28397524.

14 Balestra C, Lafère P, Germonpré P. Persistence of critical flicker fusion frequency impairment after a 33 mfw SCUBA dive: evidence of prolonged nitrogen narcosis? Eur J Appl Physiol. 2012;112:4063–8. doi: 10.1007/s00421-012-2391-z. PMID: 22476770.

15 Harriss DJ, Macsween A, Atkinson G. Standards for ethics in sport and exercise science research: 2018 Update. Int J Sports Med. 2017;38:1126–31. doi: 10.1055/s-0043-124001. PMID: 29258155.

16 Hosokawa T, Mikami K, Saito K. Basic study of the portable fatigue meter: effects of illumination, distance from eyes and age. Ergonomics. 1997;40:887–94. doi: 10.1080/001401397187720. PMID: 9306740.

17 Task force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Eur Heart J. 1996;17:354–81. PMID: 8737210.

18 Wasserstein RL, Lazar NA. The ASA Statement on *p* -values: Context, process, and purpose. The Am Stat. 2016;70:129–33. doi: 10.1080/00031305.2016.1154108.

19 Cohen J. Statistical power analysis for the behavioral sciences [Internet]. 2nd ed. Hillsdale (NJ): L. Erlbaum Associates; 1988. [cited 2020 May 20]. Available from: https://www.taylorfrancis.com/books/mono/10.4324/9780203771587/statistical-power-analysis-behavioral-sciences-jacob-cohen.

20 Braunstein EP. Beitrag zur Lehre des intermittierenden Lichtreizes der gesunden und kranken Retina. Zeitschrift für Psychologie und Physiologie der Sinnesorgane. 1903;33:171–206, 241–88.

21 Muth T, Schipke JD, Brebeck AK, Dreyer S. Critical flicker fusion frequency: confounders and caveats. Eur J Appl Physiol. 2026;126:725–34. doi: 10.1007/s00421-025-05935-7. PMID: 40864284. PMCID: PMC12948893.

22 Hemelryck W, Rozloznik M, Germonpré P, Balestra C, Lafère P. Functional comparison between critical flicker fusion frequency and simple cognitive tests in subjects breathing air or oxygen in normobaria. Diving Hyperb Med. 2013;43:138–42. PMID: 24122188. [cited 2026 Feb 1]. Available from: https://dhmjournal.com/images/IndividArticles/43Sept/Hemelryck_dhm.43.3.138-142.pdf.

23 Tikkinen J, Wuorimaa T, Siimes MA. A comparison of simple reaction time, visual discrimination and critical flicker fusion frequency in professional divers at elevated pressure. Diving Hyperb Med. 2016;46:82–6. PMID: 27334995. [cited 2026 Feb 1]. Available from: https://dhmjournal.com/images/IndividArticles/46June/Tikkinen_dhm46.2.82-86.pdf.

24 Lang M. The state of oxygen-enriched air (nitrox). Diving Hyperb Med. 2006;36(2):87–93. [cited 2026 Feb 1]. Available from: https://dhmjournal.com/images/IndividArticles/36June/Lang_dhm.36.2.87-93.pdf.

25 Linnarsson D, Ostlund A, Sporrong A, Lind F, Hesser CM, Hamilton RW. Does oxygen contribute to the narcotic action of hyperbaric air? In: Proceedings of the XVIth meeting of the European undersea biomedical society. Amsterdam: EUBS; 1990. p. 339–41.

26 Ciarlone GE, Hinojo CM, Stavitzski NM, Dean JB. CNS function and dysfunction during exposure to hyperbaric oxygen in operational and clinical settings. Redox Biol. 2019;27:101159. doi: 10.1016/j.redox.2019.101159. PMID: 30902504. PMCID: PMC6859559.

27 Frankenhaeuser M, Graff-Lonnevig V, Hesser CM. Effects on psychomotor functions of different nitrogen-oxygen gas mixtures at increased ambient pressures. Acta Physiol Scand. 1963;59:400–9. doi: 10.1111/j.1748-1716.1963.tb02756.x. PMID: 14082611.

28 Freiberger JJ, Derrick BJ, Natoli MJ, Akushevich I, Schinazi EA, Parker C, et al. Assessment of the interaction of hyperbaric N_2, CO_2, and O_2 on psychomotor performance in divers. J Appl Physiol (1985). 2016;121:953–64. doi: 10.1152/japplphysiol.00534.2016. PMID: 27633739.

29 Scholey AB, Moss MC, Wesnes K. Oxygen and cognitive performance: the temporal relationship between hyperoxia and enhanced memory. Psychopharmacology (Berl). 1998;140:123–6. doi: 10.1007/s002130050748. PMID: 9862412.

30 Germonpre P, Balestra C, Hemelryck W, Buzzacott P, Lafere P. Objective vs. subjective evaluation of cognitive performance during 0.4-MPA dives breathing air or nitrox. Aerosp Med Hum Perform. 2017;88:469–75. doi: 10.3357/AMHP.4608.2017. PMID: 28417835.

31 Frankenhaeuser M, Graff-Lonnevig V, Hesser CM. Psychomotor performance in man as affected by high oxygen pressure (3 atmospheres). Acta Physiol Scand. 1960;50:1–7. doi: 10.1111/j.1748-1716.1960.tb02067.x. PMID: 13701420.

32 Gill M, Natoli MJ, Vacchiano C, MacLeod DB, Ikeda K, Qin M, et al. Effects of elevated oxygen and carbon dioxide partial pressures on respiratory function and cognitive performance. J Appl Physiol (1985). 2014;117:406–12. doi: 10.1152/japplphysiol.00995.2013. PMID: 24947022.

33 Buhler RA. Stress and flicker fusion. In: Ittelson WH, Kutash SB, Abramson L, Seidenberg B, editors. Perceptual changes in psychopathology. [Internet]. Piscataway: Rutgers University Press; 1961. p. 105–252. doi: 10.1037/14348-007. [cited 2022 Oct 6]. Available from: http://content.apa.org/books/14348-007.

34 Kaur V, Walia L, Singh R. Critical flicker fusion frequency: Effect of age, gender, sleep and display screens. International Journal of Clinical Medicine Research. 2020;7(6). doi: 10.21276/ijcmr.2020.7.6.20.

35 Lockhart JM. Ambient temperature and the flicker-fusion threshold. J Exp Psychol. 1971;87:314–9. doi: 10.1037/h0030513. PMID: 5547292.

36 Accornero N, Vito GD, Rotunno A, Perugino U, Manfredi M. Critical fusion frequency in MS during mild induced hyperthermia. Acta Neurol Scand. 1989;79(6):510–4. doi: 10.1111/j.1600-0404.1989.tb03823.x. PMID: 2782032.

37 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.

38 Gooden BA. Mechanism of the human diving response. Integr Physiol Behav Sci. 1994;29:6–16. PMID: 8018553.

39 Schipke JD, Pelzer M. Effect of immersion, submersion, and scuba diving on heart rate variability. Br J Sports Med. 2001;35:174–80. doi: 10.1136/bjsm.35.3.174. PMID: 11375876. PMCID: PMC1724326.

40 Edge CJ. Recreational diving medicine. Curr Anaesth Crit Care. 2008;19:235–46. doi: 10.1016/j.cacc.2008.06.001.

41 Hann NA, Hoyt JC. NOAA diving standards & safety manual [Manual]. NOAA Diving [Internet]; 2023. [cited 2025 Aug 25]. Available from: https://omao.noaa.gov/sites/default/files/2023-05/NDSSM%20MAY%202023%20FINAL.pdf.

42 Bosco G, Vezzani G, Mrakic Sposta S, Rizzato A, Enten G, Abou-Samra A, et al. Hyperbaric oxygen therapy ameliorates osteonecrosis in patients by modulating inflammation and oxidative stress. J Enzyme Inhib Med Chem. 2018;33(1):1501–5. doi: 10.1080/14756366.2018.1485149. PMID: 30274530. PMCID: PMC6171420.

43 Pollock NW. Oxygen partial pressure - hazards and safety. In: McKenna SA, Prezant RS, editors. Proceedings of the American Academy of Underwater Sciences 38th Symposium. Dauphin Island (AL): American Academy of Underwater Sciences; 2019.

44 Fock A, Millar I. Oxygen toxicity in recreational and technical diving. Diving Hyperb Med. 2008;38:86–90. PMID: 22692690. [cited 2026 Feb 1]. Available from: https://dhmjournal.com/images/IndividArticles/38June/Fock_dhm.38.2.86-90.pdf.

Conflicts of interest and funding: nil

Submitted: 6 February 2026
Accepted after revision: 30 May 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

Early nitrogen wash-out for inside attendants during hyperbaric oxygen therapy: a novel oxygen distribution regimen

Mårten Silvanius[1,2], Oscar Plogmark[3], Måns Forsberg[1,2,4], Oskar Frånberg[1,3]

[1] *Department of Mathematics and Natural Sciences, Blekinge Institute of Technology, Karlskrona Sweden*
[2] *Swedish Armed Forces Diving and Naval Medicine Centre, Karlskrona Sweden*
[3] *Department of Respiratory Medicine and Allergology, Institution for Clinical Sciences, Lund University, Lund, Sweden*
[4] *Regional Hospital of Helsingborg, Helsingborg, Sweden*

Corresponding author: *Mårten Silvanius, Swedish Armed Forces, Karlskrona Sweden*
marten.silvanius@gmail.com

Keywords
Decompression sickness; Decompression tables; Hyperbaric oxygen treatment; Hyperbaric research; Pressure chambers

Abstract
(Silvanius M, Plogmark O, Forsberg M, Frånberg O. Early nitrogen wash-out for inside attendants during hyperbaric oxygen therapy: a novel oxygen distribution regimen. Diving and Hyperbaric Medicine. 2026 30 September;56(3):286–293. doi: 10.28920/dhm56.3.286-293. PMID: 42743568.)
Introduction: Hyperbaric oxygen therapy (HBOT) is widely used as treatment of decompression sickness (DCS). The patient breathes oxygen at an elevated oxygen partial pressure of up to 280 kPa, whereas any inside attendant (IA) normally breathes air. This results in nitrogen uptake and tissue loading in the IA, which must be considered during decompression. The US Navy Treatment Table 6, and its equivalent used by the Swedish Armed Forces, prescribe periods of oxygen breathing prior to and during decompression to reduce the risk of DCS for the IA. However, since these oxygen periods are concentrated toward the latter part of the hyperbaric treatment, they do not account for the possibility of rapid decompression in the event of an earlier emergency.
Methods: We suggest rescheduling the distribution of oxygen for the IA to an earlier stage of the HBOT, which would allow early emergency decompression to a greater extent than the traditional IA oxygen regimen. To verify the duration of oxygen periods and their timing, we used the exponential linear Thalmann decompression algorithm with SWEN21B parameters to analyse compartmental gas load and perform a comparison between the original and our newly proposed regimens.
Results: We identified an alternative oxygen regimen for the inside attendant (IA) that remains within the permissible limits of the decompression algorithm yet allows for emergency decompression to be performed over an extended section of the period of compression.
Conclusions: Based on the decompression model used herein, we can conclude that greater flexibility in emergency decompression is possible if the oxygen regimen for the inside attendant (IA) is administered at an earlier stage of the therapy session.

Introduction

Hyperbaric oxygen therapy (HBOT) is widely recognised as the gold standard for treating decompression sickness (DCS). This treatment has been known for over a century and has shown good results in mitigating symptoms resulting from rapid decompression or arterial gas emboli.[1] HBOT is typically performed at hospitals, military facilities, offshore facilities, ships, or private facilities.

The typical pressure used in HBOT ranges from 240–280 kPa, which corresponds to a depth of 14–18 metres of seawater (msw). The pressurising gas is typically air and the oxygen is normally distributed through a built-in breathing system (BIBS) or a hood. The inside attendants (IA) breathe the chamber air and therefore absorb nitrogen into their tissues in the same way as they would during a dive, but without any submersion, thermal or workload-related issues.[2–4]

DECOMPRESSION PROTOCOLS: SWAF B6 AND USN TT6

In the Swedish Armed Forces (SwAF), decompression sickness is normally treated using Behandlingstabell 6 (B6), which closely resembles the U.S. Navy Treatment Table 6 (TT6).[4,5] Both tables involve prolonged oxygen breathing during compression and during decompression, with specific oxygen-breathing periods prescribed for the IA to limit inert gas uptake. While effective, these oxygen periods are largely concentrated in the latter phase of the treatment, limiting the possibility for earlier emergency decompression should the need arise.

USN TT5 AND ALTERNATIVE PROTOCOLS

According to the U.S. Navy Diving Manual, if the patient achieves full recovery after 10 minutes at 280 kPa, it is acceptable to transition to the shorter Treatment Table 5

(TT5).[4] This table has been evaluated with favourable results.[6,7] Alternative protocols resembling TT5 have also been proposed for the treatment of DCS and have demonstrated promising outcomes.[8] Alternative gases such as nitrox with fractions of oxygen (F_{O2}) 50% and 60.5% have been suggested for the IA.[9,10]

INSIDE ATTENDANT SAFETY IN EMERGENCY SCENARIOS

The maximum pressure of the TT6 and B6 tables is 280 kPa (2.8 bar, 18 msw equivalent), a pressure/depth at which the maximum allowable duration of air breathing before decompression stops would become mandatory varies between 50–60 minutes, depending on the decompression table used. For instance, the 'no-decompression time' for SWEN21-dive table at 18 msw is 59 minutes.[11] Emergencies requiring premature termination of HBOT may arise due to patient deterioration or external hazards, necessitating consideration of the IA's decompression safety.

The safety for IA must always be remembered regardless of situation.[12] According to multiple studies, the risk of DCS for IAs during HBOT is considered low and acceptable, with a reported incidence below 1%.[13,14] Severe cases of central nervous system (CNS) bends have nevertheless occurred, even when IAs are within limits of compartmental gas load.[2,15,16] This highlights a critical limitation of 'decompression models' as they provide a structured and predictive framework, but cannot fully capture the complex physical, physiological and individual factors involved in DCS.

In most scenarios, chamber operators have limited support and flexibility apart from standard decompression tables, even though there are strategies described in US Navy diving manual such as "*Recompression treatment abort procedures*", however the IA might be confined until the completion of the therapy session.[4]

Methods

The primary aim of this study was to evaluate the oxygen administration regimen for the IA and to assess the feasibility of facilitating emergency decompression over an extended portion of the HBOT session, focusing on the SwAF B6 recompression table.

The secondary aim was to compare the IA inert gas load across different HBOT protocols by introducing the Nitrogen Tension Index (NTI), a normalised measure of compartmental nitrogen tension relative to the maximum permissible tissue tension (MPTT).

We hypothesised that redistributing oxygen periods can reduce the proportion of the treatment during which the IA is unable to perform immediate emergency decompression. An analytical approach was chosen to calculate expected tissue tension or compartmental gas load, according to the Thalmann decompression algorithm (EL-DCM Thalmann), and to compare these values to other standard exposures for IA's that are described as safe or associated with a low risk of DCS.

SUGGESTED STRATEGY

The objective of the proposed new oxygen regimen is to start administering oxygen earlier in order to maintain the nitrogen load below the MPTT defined by SWEN21B parameters.[11] Breathing oxygen at greater pressures than 2.5 bar (equivalent to ~15 msw depth) carries a risk of CNS oxygen toxicity, including seizures. While the risk is generally low in resting individuals at 2.5 bar (~15 msw) in a hyperbaric chamber, and very unlikely at 1.9 bar (~9 msw), patients with severe Type II DCS or arterial gas embolism may be unusually sensitive and thus at higher risk.[4] This must be considered when selecting an oxygen administration regimen.

INTRODUCING NITROGEN TENSION INDEX (NTI)

To enable standardised comparison of inert gas loading across tissue compartments and between profiles with differing dive characteristics, we introduce the Nitrogen Tension Index (NTI). This index expresses the current partial pressure of nitrogen (PtN_2) in a given compartment as a normalised percentage of the chosen MPTT. Lower NTI values indicate a greater margin to the maximum permissible tissue tension (MPTT) and should therefore be interpreted as reflecting lower decompression risk for the inside attendant, whereas higher NTI values indicate reduced safety margins. NTI may assume negative values during hyperoxic exposure, when tissue nitrogen tension is reduced below surface equilibrium pressure.

This methodology follows Witucki et al, who employed a similar normalisation approach to evaluate nitrogen load as a fraction of allowable tissue tension limits in the US Navy no-stop diving tables.[3] NTI is not intended as a dynamic model of decompression stress during descent or bottom time. It is designed as a surface-referenced metric, most relevant for assessing and comparing nitrogen load after surfacing.

The advantage of this method is that it facilitates comparison between compartments with different MPTT and enables inter-profile comparisons based on a surface-referenced nitrogen pressure ($P_{surf}N_2$), defined as the tissue nitrogen partial pressure corresponding to equilibrium with inspired air at the local surface ambient pressure.

CALCULATION OF NTI

NTI for each compartment is calculated using a piecewise formulation, depending on whether tissue nitrogen tension is above or below surface equilibrium.

For $PtN_2 \geq P_{surf}N_2$:

$$NTI_{pos} = \frac{PtN_2 - P_{surfN2}}{MPTT - P_{surfN2}} \quad (1)$$

For $PtN_2 < P_{surf}N_2$:

$$NTI_{neg} = \left(\frac{PtN_2}{P_{surfN2}} - 1\right) \quad (2)$$

This yields NTI = 0% when PtN_2 equals $P_{surf}N_2$. NTI = 100% when PtN_2 equals MPTT. NTI < 0% when PtN_2 is below $P_{surf}N_2$, indicating nitrogen elimination due to for example hyperoxic exposure. NTI > 100% represents overloading beyond safe decompression limits.

Both the US Navy and our implementation of the Thalmann algorithm involves a deliberate limitation as it does not account for inhaled F_{O2} greater than 80%. As a result, when faced with F_{O2} greater than 80%, the lowest possible value of NTI is -80%.[11,17]

COMPARING NTI VALUES AND GAS LOADS ACROSS HBOT PROTOCOLS

To contextualise and compare the derived NTI values, established hyperbaric treatment tables were used as reference, with three HBOT protocols from the University of California San Diego (UCSD) and two from Haukeland, analysed using the Thalmann decompression algorithm.[3,18] These protocols are used clinically and have demonstrated a low incidence of decompression sickness in IAs. The UCSD and Haukeland reference protocols involve chamber exposures of comparable depth but shorter overall duration than the SwAF B6, and include either no oxygen breathing for the inside attendant or only brief oxygen periods at pressure and/or during decompression.

DECOMPRESSION CEILING

For each time point during the HBOT, tissue nitrogen tension was evaluated across all compartments. The decompression ceiling was determined by identifying the compartment with the greatest exceedance of its maximum permissible tissue tension (MPTT) at surface pressure, expressed as $PtN_2 - MPTT$. This compartment represents the leading tissue with respect to ascent limitation at that time point. The red field shown in Figures 1–4 therefore represents the decompression ceiling imposed by the compartment that most restricts ascent, and may shift between compartments over the course of the exposure.

ANALYSIS

Oxygen administration strategies for the inside attendant were evaluated using iterative computer-based simulations of inert gas uptake and elimination. All simulations were performed under the following desired boundary conditions: (i) no alteration of the treatment table pressure–time profile; (ii) no oxygen administration deeper than 2.5 bar (15 msw); (iii) oxygen administration between 1.9–2.5 bar (9–15 msw) permitted only during ascent; (iv) oxygen administration initiated as early as possible; (v) suppression of nitrogen load during the decompression phase; and (vi) equal or fewer oxygen periods compared with the original schedule, while maintaining synchronisation with the patient's oxygen administration and air breaks. Compartment-level nitrogen tissue tensions relative to the MPTT were calculated for each strategy. The analysis focused on the leading tissue, defined as the compartment with the smallest remaining margin to its MPTT, and only the optimal oxygen strategy for each case is presented in the results.

Results

In Figures 1–4, the red field represents the decompression ceiling for the leading tissue and is used as indicator of nitrogen load suppression. When present, ascent must not pass the pressure indicated by this ceiling. If a ceiling is absent, a direct ascent to the surface is permissible. Oxygen distribution protocol is shown as a dashed line, and each period is 20 minutes long followed by a 5-minute air-break. No oxygen administration for the IA is performed at greater pressure than 2.5 bar (15 msw).

The oxygen distribution for our proposed regimen can clearly be seen to suppress the nitrogen gas load for leading compartments indicated by a reduction of the red area in those alternatives.

Figure 5 shows the NTI for the IA at surfacing for the TT6 compared to our proposed regimen. Each of the nine compartments are presented along the x-axis and NTI on the y-axis. For reference, 'UCSD 1', 'Haukeland new', and a simulated direct decompression from 2.8 bar (18 msw) after 59 minutes are included as standard protocol comparisons.

In Figure 5 it is apparent that the half-time (HT) 40 min compartment is the leading tissue for the 2.8 bar (18 msw) / 59 min profile as it has reached its MPTT revealing an NTI of 100%. It can also be observed that the NTI when decompressing is higher in the slower compartments with half-time HT ≥ 120 min for TT6 and our proposal compared to 'UCSD 1', 'Haukeland new' and 18 msw / 59 min.

While NTI is typically interpreted based on the maximum value per compartment, the average NTI for all compartments at completion of decompression is presented in Figure 6 as a descriptive metric to illustrate overall nitrogen load. This is not intended as a decision metric in a deterministic model, but rather as a comparative tool to highlight differences in decompression stress distribution among treatment strategies.

Figure 1

The decompression ceiling defined by the leading tissue compartment is shown in red for the original B6 (A), compared with the proposed oxygen regimen for the inside attendant (IA) (B)

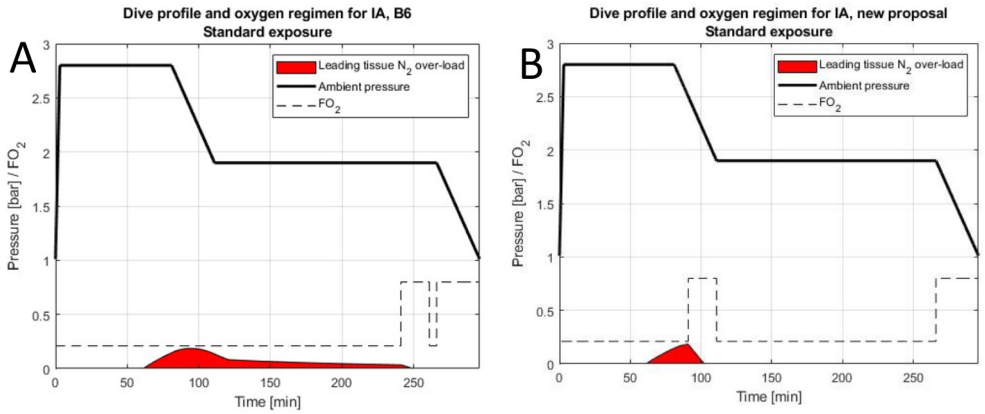


Figure 2

The decompression ceiling defined by the leading tissue compartment is shown in red for the original B6 (A) and the proposed oxygen regimen for the inside attendant (IA) (B), following a previous exposure of 18 msw for 59 minutes

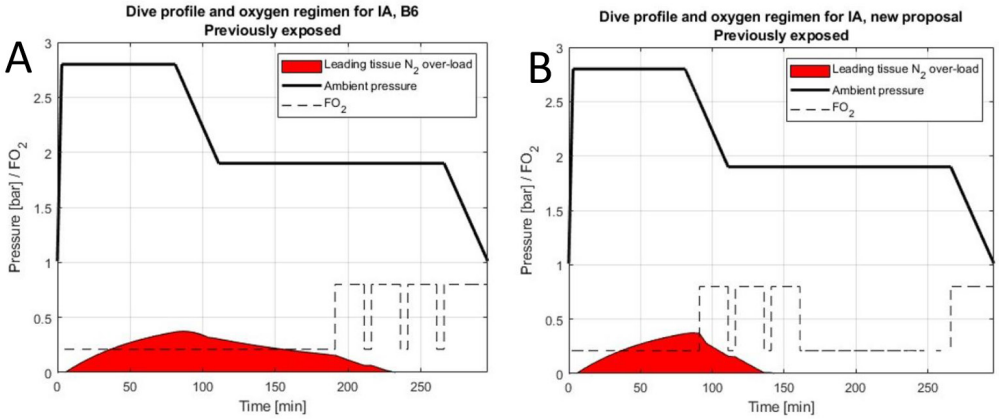


Figure 3

The red field represents the decompression ceiling imposed by the leading tissue compartment for the original extended B6 (A) and the proposed oxygen regimen for the inside attendant (IA) (B). This scenario requires additional oxygen periods

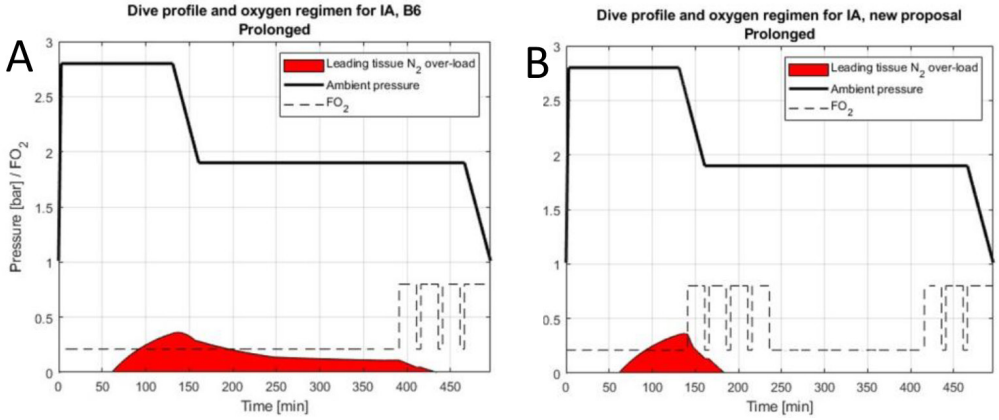


Figure 4

The red field represents the decompression ceiling imposed by the leading tissue compartment for the original extended B6 (A) and the proposed oxygen regimen for the inside attendant (IA) (B), following a previous exposure of 18 msw for 59 minutes. This scenario requires additional oxygen periods

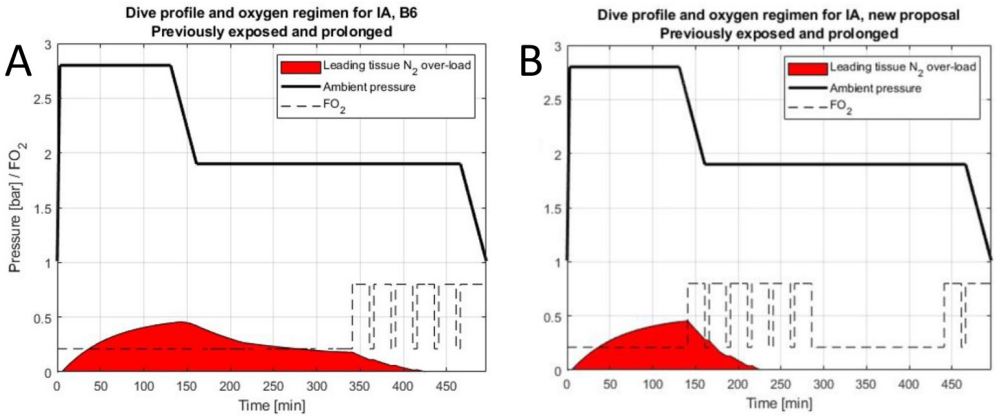


Figure 5

A comparison of different treatment tables and dive profiles with correlating Nitrogen Tension Index NTI when surfacing/completion of decompression for each of the nine compartments presented as half-time between 5 and 240 min. Panel 1 includes the B6 and our proposal, 'UCSD 1', 'Haukeland new' and the direct ascent dive profile 18 msw 59 min. Panel 2–4 includes a comparison for the cases where previously exposed IA and extended treatment tables are present

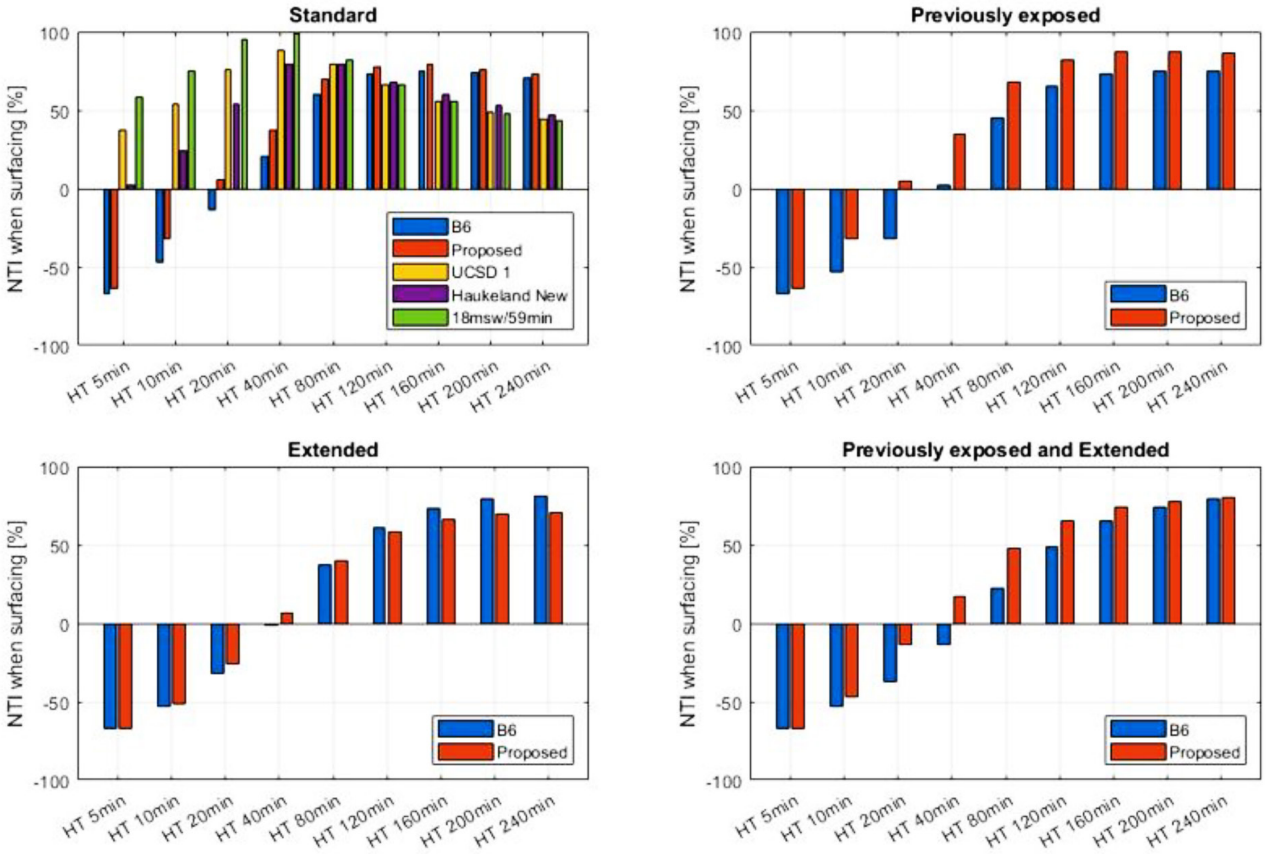
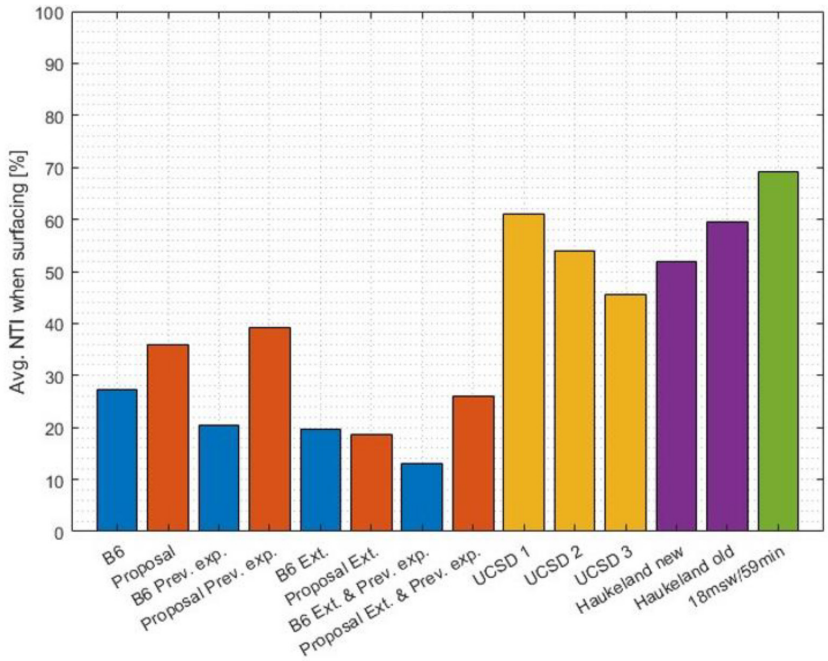


Figure 6
The average nitrogen tissue index (NTI) when surfacing in comparison of the analysed HBOT regimens and an 18 msw / 59 min dive profile



Our analysis indicates that the average NTI at completion of decompression is lower with our proposed regimen compared to the UCSD case, the Haukeland profile, and the 2.8 bar (18 msw) / 59 min dive profile. However, it remains higher or similar than that of the original B6, whether extended or not, and regardless of prior exposure.

Discussion

This study proposes a modification to the current SwAF Treatment Table B6 protocol for the IA. In the revised protocol, oxygen breathing is initiated earlier during the decompression from 2.5 to 1.9 bar (15 to 9 msw). This contrasts with the original B6, where the IA begins oxygen breathing only during the final part at 1.9 bar (9 msw). The goal of this modification is to reduce the IA decompression obligation early during the treatment table, thereby increasing operational flexibility and enabling safe emergency decompression if required.

In high-risk situations or environments such as on a warship in a conflict zone, or any situation where the treatment may need to be abruptly terminated, it can be more appropriate to adopt this alternative oxygen regimen for the attendant during HBOT. Moreover, in the event of an unexpected deterioration in the patient's condition, it may be imperative to promptly evacuate them from the chamber while avoiding DCS in the attendant. This may also be an important property if chamber is small and transportable.

While this alternative oxygen regimen reduces the amount of time the chamber attendant must undergo a decompression stop, it is important to consider some potential criticisms. Firstly, instances of emergencies during this type of treatment are rare. Secondly, the attendant could instead undergo recompression using a surface decompression strategy if considered necessary. Thirdly, the increased partial pressure of oxygen (PO_2) during the initial oxygen period (2.5 to 1.9 bar [15 msw to 9 msw] decompression) could pose a risk for oxygen convulsions. Fourthly, the attendant must sometimes assist the patient and equipment during decompression, potentially compromising their ability to administer oxygen to themselves. Fifthly, the oxygen regimen for the chamber attendant outlined in TT6 has been widely recognised as effective in reducing DCS. Lastly, an alternative solution to achieve the desired ability to decompress the IA at any time, could be to have several IAs alternating prior to their no-decompression time expiring.

To address the potential drawbacks previously described, we recommend separating a traditional hospital setting from a military environment where this new oxygen regimen may be more appropriate. In an emergency there may not be access to a pressure chamber for surface decompression for the IA. It is uncommon for convulsions to occur in a dry hyperbaric environment with pure oxygen breathing at pressures less than 2.5 bar (~15 msw), especially if there has been no prior oxygen exposure or if the workload is low.[4] Any concern for oxygen convulsions during decompression between 2.5 to 1.9 bar (15 to 9 msw) can be mitigated by allowing the IA to hold the oxygen mask rather than having it securely fastened, as this will avoid continued breathing from the mask if unexpected convulsions occur. This certain period still poses a low risk, as the time and decompression are

favorable to avoid convulsions and, referring to experiences from UCSD and Haukeland, no cases of oxygen convulsions have been reported during > 20,000 of treatments with up to 30 minutes of oxygen exposure at a pressure of 2.4 bar (14 msw).[2,3] During decompression, there may be multiple tasks to attend to, which is similar to the situation during decompression from 1.9 bar (9 msw) to the surface. According to the decompression algorithm used, there are similarly low NTI with this oxygen decompression regimen compared to the TT6 regimen. Lastly, it may not always be possible to have multiple available and unexposed IAs.

When deciding the best treatment option for the patient, it is relevant to highlight the advantages of shorter treatment tables, such as the USN Treatment Table 5 or the Fremantle Hospital Hyperbaric Medicine Unit FH01. They have demonstrated equal or better resolution than the longer TT6 for a range of conditions, including mild neurological symptoms, pain, lymphatic/skin and constitutional/non-specific symptoms which often resolved within 10 minutes at treatment pressure.[6–8] By using a shorter treatment table it may lower the threshold for initiating HBOT, especially in the case where only a smaller (transportable) hyperbaric chamber is available. For these treatments it is also possible to decompress the IA at any time avoiding DCS, according to our mathematical analysis.

Among the few reported cases of central nervous system decompression sickness in IA referenced in this study, one case occurred in the absence of a patent foramen ovale (PFO), one was associated with a large PFO, and in one case PFO status was not reported.[2,15,16] As PFO is a recognised risk factor for decompression sickness, particularly for neurological manifestations, this variability may contribute to outcome heterogeneity in reported cases.[19,20] However, this does not alter the compartmental gas-load analysis presented herein. Furthermore, hyperbaric chamber operation at altitude has not been shown to increase decompression risk for IAs.[21]

Conclusions

Our main finding is that an alternative oxygen regimen for the IA, compared to the Swedish Armed Forces Treatment Table 6 (B6) with extensions and previous exposure, is feasible and allows for the option of emergency decompression over a longer period. For example, the total time fraction of the IA's decompression obligation decreased from 75% with the original B6 to just 17% with our newly proposed protocol. This analysis is based on the compartmental gas load according to the exponential-linear Thalmann algorithm with SWEN21B parameters. We also found that the average Nitrogen Tension Index (NTI) on completion of decompression is slightly higher or similar for our protocol compared to the standard B6, however lower compared to other uneventful HBOT regimens and the diving profile 18 msw/59 min. The slower compartment's half-time ≥ 120 min has a higher NTI when surfacing for B6 and our proposal compared to reference dive profile and HBOTs.

Our proposed oxygen regimen for IA is believed to be particularly beneficial for handling unforeseen situations such as emergency decompressions. The traditional oxygen regimen, however, has been extensively verified and does not pose any increased risk of oxygen convulsions due to high partial pressures of oxygen or substantial risk for decompression sickness. After human evaluations it can be warranted to switch to this new protocol as the standard method for the distribution of IA oxygen breathing time.

References

1 Moon RE, Mitchell SJ. Hyperbaric oxygen for decompression sickness. Undersea Hyperb Med. 2021;48:195–203. doi: 10.22462/03.04.2021.11. PMID: 33975411.

2 Risberg J, Englund M, Aanderud L, Eftedal O, Flook V, Thorsen E. Venous gas embolism in chamber attendants after hyperbaric exposure. Undersea Hyperb Med. 2004;31:417–29. PMID: 15686273.

3 Witucki P, Duchnick J, Neuman T, Grover I. Incidence of decompression sickness and oxygen toxicity in chamber attendants: a 28-year experience. Undersea Hyperb Med. 2013;40:345–50. PMID: 23957205.

4 U.S. Navy Diving Manual. Revision 7. Washington (DC): Naval Sea Systems Command; 2016.

5 Regler för militär sjöfart RMS-Dyk [Swedish Armed Forces diving regulations]. Stockholm: Högkvarteret; 2013.

6 Goodman MW, Workman RD. Minimal-recompression, oxygen-breathing approach to treatment of decompression sickness in divers and aviators. Res Rep US Nav Exp Diving Unit. 1965;5-65:1–40. PMID: 5295232.

7 Green JW, Tichenor J, Curley MD. Treatment of type I decompression sickness using the U.S. Navy treatment algorithm. Undersea Biomed Res. 1989;16:465–70. PMID: 2603243.

8 Banham N, Hawkings P, Gawthrope I. A prospective single-blind randomised clinical trial comparing two treatment tables for the initial management of mild decompression sickness. Diving Hyperb Med. 2022;52:85–91. doi: 10.28920/dhm52.2.85-91. PMID: 35732279. PMCID: PMC9522606.

9 Larsson AC, Uusijärvi J, Frånberg O, Eksborg S, Lindholm P. Nitrox permits direct exit for attendants during extended hyperbaric oxygen treatment. Undersea Hyperb Med. 2012;39:605–12. PMID: 22400451.

10 Hansen MB, Jansen T, Sifakis MB, Hyldegaard O, Jansen EC. Chamber personnel's use of Nitrox 50 during hyperbaric oxygen treatment: a quality study. Undersea Hyperb Med. 2013;40:395–402. PMID: 24224283.

11 Silvanius M, Rullgård H, Ekström M, Frånberg O. Proposed Thalmann algorithm air diving decompression table for the Swedish Armed Forces. Undersea Hyperb Med. 2023;50:67–83. doi: 10.22462/01.01.2023.37. PMID: 37302072.

12 Clarke R. Health care worker decompression sickness: incidence, risk and mitigation. Undersea Hyperb Med. 2017;44:509–19. PMID: 29281188.

13 Sheffield PJ, Pirone CJ. Decompression sickness in inside attendants. In: Sheffield PJ, Desautels DA, editors. Hyperbaric

facility safety: A practical guide. Flagstaff (AZ): Best Publishing Company; 1999. p. 643–63.
14 Uzun G, Mutluoğlu M, Ay H, Yildiz S. Decompression sickness in hyperbaric nurses: retrospective analysis of 4500 treatments. J Clin Nurs. 2011;20(11–12):1784–7. doi: 10.1111/j.1365-2702.2010.03623.x. PMID: 21554460.
15 Kot J, Lenkiewicz E, Lizak E, Góralczyk P, Chreptowicz U. Spinal cord decompression sickness in an inside attendant after a standard hyperbaric oxygen treatment session. Diving Hyperb Med. 2021;51:103–6. doi: 10.28920/dhm51.1.103-106. PMID: 33761550. PMCID: PMC8313770.
16 Johnson-Arbor K. Type II decompression sickness in a hyperbaric inside attendant. Undersea Hyperb Med. 2012;39:915–9. PMID: 23045920.
17 Gerth WA, Doolette DJ. VVal-79 maximum permissible tissue tension table for Thalmann algorithm support of air diving. NEDU Research Report No.: TR-12-01. Panama City (FL): Naval Experimental Diving Unit; 2012.
18 Brattebo G, Aanderud L, Risberg J, Thorsen E, Forland M. Incidence of decompression illness among HBO_2 nurses [abstract]. Zdrav Vestn. 1997;66:17A.
19 Bove AA. Risk of decompression sickness with patent foramen ovale. Undersea Hyperb Med. 1998;25:175–8. PMID: 9789338.
20 Germonpré P, Lafère P, Portier W, Germonpré FL, Marroni A, Balestra C. Increased risk of decompression sickness when diving with a right-to-left shunt: results of a prospective single-blinded observational study (Carotid Doppler Study). Front Physiol. 2021;12:763408. doi: 10.3389/fphys.2021.763408. PMID: 34777020. PMCID: PMC8586212.
21 Bell J, Thombs PA, Davison WJ, Weaver LK. Decompression tables for inside chamber attendants working at altitude. Undersea Hyperb Med. 2014;41:505–13. PMID: 25562942.

Conflicts of interest and funding

This work was partially supported by the Swedish Armed Forces through, Swedish Defense Material Administration under Grant [430919-LB967593]. No conflicts of interest were declared.

Submitted: 18 March 2023
Accepted after revision: 6 June 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

The effect of hyperoxic environment on lung function and myopisation in an extended closed-circuit rebreather diving expedition

Sofia A Sokolowski-Wojnicki[1,2], Mikolaj J Wojnicki[3], Anne K Räisänen-Sokolowski[1,2,4]

[1] *Department of Pathology, University of Helsinki, Finland*
[2] *DAN Europe Research Division (Roseto-Helsinki), Helsinki, Finland*
[3] *Institute of Clinical Medicine, University of Eastern Finland, Kuopio, Finland*
[4] *Department of Pathology, Helsinki University Hospital, Helsinki, Finland*

Corresponding author: *Sofia A Sokolowski-Wojnicki, Department of Pathology, University of Helsinki, Finland*
ORCiD: *0000-0002-7936-1436*
sofia.sokolowski@helsinki.fi

Keywords
Myopia; Oxygen toxicity; Scientific diving; Side effects; Spirometry; Technical diving; Vision

Abstract
(Sokolowski-Wojnicki SA, Wojnicki MJ, Räisänen-Sokolowski AK. The effect of hyperoxic environment on lung function and myopisation in an extended closed-circuit rebreather diving expedition. Diving and Hyperbaric Medicine. 2026 30 September;56(3):294–302. doi: 10.28920/dhm56.3.294-302. PMID: 42743569.)
Introduction: Repetitive closed-circuit rebreather (CCR) dives subject the human body to a hyperoxic environment. Prolonged oxygen exposure can lead to lung abnormalities and myopic shift. The aim of this study was to analyse the effects of a 31-day period of repeated CCR dives on divers' lung function and visual acuity.
Methods: The CCR divers recruited for the study were participating in a research project in Chuuk Lagoon, Micronesia. Weekly measurements included lung function using spirometry and visual acuity with a Snellen chart. Oxygen exposure was calculated using multiple models. The impact on spirometry was tested by comparing the values before and after the expedition and by running regression analysis against accumulated oxygen exposure.
Results: The divers (n = 12) performed a total of 173 CCR dives with mean (SD) maximum depth of 38 (13.9) msw and dive time 101 (38.8) min. The daily oxygen exposure limits for unit pulmonary toxic dose and central nervous system toxicity were respected. One diver had a decrease in multiple lung function parameters. Another developed spirometric evidence of mild obstruction by the end of the expedition. However, there were no statistically significant changes in spirometry results. Visual acuity showed fluctuations throughout the expedition, but no overall trend.
Conclusions: Diving expeditions can be executed safely when respecting the guidelines for oxygen exposure. Reasonable oxygen exposure does not seem to significantly affect lung function or visual acuity.

Introduction

Scuba diving can be used as a tool to collect scientific data underwater, especially in the fields of marine biology and archaeology. Scientific diving is a form of occupational diving requiring specific data collection techniques underwater. It is often practised during expeditions with limited time, where the goal is to maximise data collection through consecutive and intensive diving sessions. Closed-circuit rebreathers (CCRs) have become increasingly popular in scientific diving as they allow for longer and deeper dives.

Consecutive CCR diving regularly exposes the human body to hyperbaric and hyperoxic environments. Oxygen toxicity primarily affects three target organs: the central nervous system (CNS), the lungs, and the eyes. Hyperoxic exposure can also have systemic effects, such as the decrease of haemoglobin concentration and increased ferritin.[1] More than 35 years ago, the National Oceanic and Atmospheric Administration (NOAA) established oxygen exposure standards that remain widely used today.[2] These guidelines were designed to prevent the onset of acute CNS oxygen toxicity symptoms. While the unit pulmonary toxic dose (UPTD) was initially developed over half a century ago to quantify the pulmonary risk related to repetitive exposure,[3] it is still widely used by divers, with few alterations to the formula over the years.[4] However, the old UPTD model has been replaced by models that consider physiological recovery, such as the Arieli K index.[5] This has been further improved into equivalent surface oxygen time (ESOT), which is simpler to use when planning diving operations.[6] Additionally, the residual oxygen time (ROT) model can be used when the partial pressure of oxygen (PO_2) is near 130kPa.[7]

A constant high PO_2, as often used in CCR diving, can have toxic effects on the lungs. In the early stages of pulmonary toxicity, alveolar exudate forms due to endothelial dysfunction, followed by low-grade airway inflammation. If the exposure continues, small-airway fibrosis will develop. The clinical symptoms of pulmonary oxygen toxicity include dry cough, tracheal irritation, shortness of breath and chest

discomfort.[8] Spirometry is a commonly used method to measure lung function when investigating pulmonary oxygen toxicity. Although the evidence remains contradictory, it is believed that oxygen toxicity can cause a decrease in vital capacity (VC) and possibly other parameters, such as forced expiratory flows (FEF).[9]

Prolonged exposure to a hyperoxic environment can cause changes in vision, more commonly the development of myopia. An accumulation of high PO_2 damages the structure of the lens increasing its refractive power.[10] However, this is typically reversible after the exposure ends.[11] This phenomenon is more common in hyperbaric oxygen treatment (HBOT) patients, and the evidence in divers is scarce.[12] As CCR diving maintains a consistently high PO_2, divers are at a higher risk of developing myopia during periods of intensive diving.[13,14]

Anecdotally, divers report having an 'end of expedition cough' or 'blurry vision' after prolonged diving expeditions. However, to our knowledge, there is little real-life data to show such changes. Thus, the aim of this study was to analyse the effects of a 31-day period of repeated CCR dives on divers' lung function and visual acuity, the two principal targets of long-term oxygen toxicity.

Methods

This study was conducted according to the Declaration of Helsinki 2024. The ethical review of the study (authorisation number 37/17) and research permission were granted by the University of Milan, Milan, Italy.

STUDY SUBJECTS AND DIVING

The technical divers (n = 12) recruited for the study were participating in a nautical archaeological research project in Chuuk Lagoon, the Federated States of Micronesia. All divers gave their written informed consent to participate in this study. The divers used back-mount CCR for research dives and open circuit scuba (OC) for safety dives. They completed 1–2 dives per day with regular days off during the 31-day expedition. For CCR diving, PO_2 setpoints were 70 kPa at surface and during descent, and130 kPa for the remainder of the dive. Dive time was restricted to a maximum of 180 minutes for a single dive. The divers used their own gear and dive computer for dive planning and decompression. Liberty (n = 5), JJ-CCR (n = 5), XCCR (n = 1) and AP Inspiration (n = 1) electronic rebreathers were used. Buhlmann ZHL-16C decompression software with gradient factors of 50/70 were used for decompression planning. The investigators did not have access to the divers' gear. Hence, the dive profiles were collected from additional dive computers in the gauge-only mode (iX3M 2 Tech+, Ratio Computers, Valpiana, Italy). Dive logs were analysed in the application Dana Health (DAN Europe, Roseto, Italy). The retrieved data included maximum depth, dive time, decompression profile and gradient factors.

OXYGEN TOXICITY

Oxygen exposure was calculated separately for CNS%, pulmonary oxygen toxicity (POT) and accumulated exposure. The CNS% was calculated as a percentage of the duration of the maximum single exposure at a constant PO_2 of 130 kPa according to NOAA, which is equal to 180 minutes.[2] POT was estimated using UPTD,[15] ESOT[6] and ROT.[7] The ESOT value, and its recovery formula, were considered to reset after 12 hours, if the ESOT load was less than 500.[16] Additionally, the probability of toxicity was derived from ROT.[7] A constant partial pressure of 130 kPa was assumed for the whole duration of the dive. The accumulated exposure throughout the expedition was calculated as hours at 101 kPa breathing 100% O_2.[12]

LUNG FUNCTION

To evaluate lung function, spirometry was performed before the first dive, and thereafter weekly until the end of the expedition. The measurements were performed by a single operator at least three hours after the previous dive by a Medikro Pro Spirometer (Kuopio, Finland). The device fulfilled the ISO 26782 and ATS/ERS criteria for diagnostic use and calibration was performed by the manufacturer. Spirometry was performed, according to the European Respiratory Society's guidelines in sitting position at least three times with less than 5% difference, and the highest values were recorded.[17] The following parameters were analysed as predicted percentages for the reference group: forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), FEV_1/FVC ratio, forced expiratory flow 75 (FEF_{75}), and forced expiratory flow 25–75 ($FEF_{25–75}$). GLI 2017 reference equations for Caucasians were used as reference values.

VISUAL ACUITY

Visual acuity was measured using a Snellen chart. The baseline measurement was taken before any diving activities. If a diver wore glasses, measurements were done both with and without them. Throughout the expedition visual acuity was measured once a week and the last measurement was performed after the last dive. Subjects were standing 6 m from the chart on a covered terrace. Measurements were taken during daylight; however, the exact time could not be standardised due to logistic issues. Both eyes were measured first separately and then together.

STATISTICAL ANALYSIS

The Shapiro-Wilk test was used to test for normality of the spirometry data. To check if the expedition affected spirometry results, paired Student t-tests were used on the first and last measurement of five parameters (predicted FVC, FEV_1, FEV_1/FVC, FEF_{75} and $FEF_{25–75}$). To evaluate the effect of accumulated physiological load on spirometry results, linear mixed-effects regression models were used.

The analysis was performed using the lme4 R package,[18] with random intercepts for the divers. As fixed effects, we included the number of CCR dives, accumulated UPTD, or ROT probability of toxicity from the previous dive, to form three models. The normality and homoscadiscity of regression residuals were checked using the Shapiro-Wilk test and visual inspection. A two-sided significance level of 0.05 was used for all statistical tests to account for possible training effect, recovery, or adaptation. To account for multiple testing, Bonferroni correction was applied separately to t-tests and to regression models. All analyses were performed using R version 4.5.3. Visualisation was done using base R graphics[19] or ggplot.[20] The data are presented as means (standard deviation). Additionally, for oxygen exposure, maximum values are also reported.

Table 1
Diver demographic data presented as mean (standard deviation); BMI – body mass index CCR – closed-circuit rebreather; msw – metres of seawater

Parameter (*n* = 12)	Mean (SD)
Age	41.6 (10.6)
BMI (kg·m^{-2})	28.9 (4.2)
Years of diving	20.7 (10.6)
Number of dives	2,846 (1,757)
Years of CCR	10.2 (6.1)
Hours of CCR	1,072.0 (969.6)
Max. depth (msw)	106.8 (33.0)

Results

POPULATION AND DIVING

The demographics of the divers participating in the study are shown in Table 1. Pre-existing medical conditions included hypothyroidism treated with thyroxine (*n* = 1), medication treated hypertension (*n* = 1), previous decompression sickness (DCS) (*n* = 2), patent foramen ovale (PFO) closure (*n* = 1), back surgery (*n* = 1), and previous cataract surgery (*n* = 1). The latter was excluded from the myopisation part of the study. The rest of the divers (*n* = 7) had no medical conditions or medications.

The divers (*n* = 12) performed a total of 173 CCR dives during a 31-day period, with no diving occurring on three of those days. The divers took additional days off for gear maintenance, administrative tasks, illness, or rest. However, none of these were due to symptoms of pulmonary or visual oxygen toxicity or decompression sickness. The divers participated in the project for two to four weeks resulting in 5–26 dives per diver. Additionally, six divers performed a total of 26 OC safety dives. One research dive was performed using OC due to CCR malfunction. The OC dives were excluded from the analysis. The summary of the descriptive statistics for each diver is shown in Table 2. The mean maximum diving depth was 38 (13.9) msw and mean dive time was 101 (38.8) min. Diver E had a high number of OC dives (nine) compared to the number of CCR dives (nine). Thus, the diver was excluded from spirometry analysis due to OC diving being a remarkable confounding factor.

OXYGEN TOXICITY

Oxygen exposure was estimated using the dive time and constant PO_2 of 130 kPa, as there was no access to the divers' own computers. The daily exposure for diving days was summarised in Table 3. It contains the mean and maximum CNS%, UPTD, ESOT, ROT and ROT probability of toxicity along with established daily limits. Two divers were contacted retrospectively and the real CNS% and UPTD were received for 35 dives to compare and assess the extent of approximation error. For a single dive, the median (IQR) percentage error of CNS% was -1.2 (-4.2–2.7) % and for UPTD 2.3 (0.17–4.2) %. The cumulative error was estimated using the bootstrap method. Depending on the number of dives (5–26) that a diver completed, the mean and 95% confidence interval for the percentage error was between -1.04 (-6.51–6.55)% and -1.09 (-3.49–1.75)% for cumulative CNS% and between 3.20 (0.32–9.62)% and 3.04 (1.44–5.34)% for cumulative UPTD. This means the cumulative UPTD from CCR dives is likely to be overestimated for most divers. Some identified sources of error included descent at PO_2 of 70 kPa, a manual increase of PO_2 at safety or decompression stops and alternating the PO_2 manually on shallow dives. Unfortunately, the oxygen exposure was not recorded for all the safety dives. Out of the 26 OC dives, the data were available for 11. Their mean (SD) CNS% was 3.71 (2.38) and UPTD 5.39 (4.94). The mean accumulated oxygen exposure was 32.0 (20.5) hours at 101 kPa breathing 100% O_2. The OC dives were excluded from the analysis of accumulated oxygen exposure.

LUNG FUNCTION

Lung function was measured by weekly spirometry during the expedition. The result of the paired *t*-test is shown in Table 4. There was no significant difference between the first and last measurements. For the regression models, spirometry data from all weeks was used, however, we decided to exclude six measurements from the ROT predicted toxicity model as the spirometry was performed more than one day after diving. The final dataset for the linear mixed-effects models is visualised in Figure 1, with detailed model results shown in Table 5. These models showed no significant association of FVC Pred% with accumulated UPTD, number of CCR dives or ROT probability of toxicity.

When looking at individual results, there was one diver with substantially reduced absolute values for lung function parameters at four weeks compared to baseline as follows: FVC -7.0%, FEV_1 -16.1%, FEV_1/FVC -9.7%, PEF -9.6%, $FEF_{25–75}$ -44.8%. However, the diver did not have any clinical symptoms of pulmonary oxygen toxicity, such as

Table 2

Summary of the dives for each diver; total days off refers to the number of days the diver was not diving, diving period describes the length of the expedition in days, and longest consecutive refers to the longest streak of consecutive diving days. *Diver E was excluded from further analysis due to a high number of OC dives. CC – closed-circuit; OC – open-circuit; EAN – enriched air nitrox

Diver	Total CC	Total off	Diving period (days)	Longest consecutive (days)	Max depth (msw)	Dive time (min)	Total OC dives	OC max depth (msw)	OC dive time (min)	OC gas O_2 (EAN)
A	5	7	11	4	38 (3.1)	98 (19.2)	0	0 (0)	0 (0)	0 (0)
B	6	7	11	4	34 (5.9)	90 (8.6)	0	0 (0)	0 (0)	0 (0)
C	8	13	21	4	27 (9.9)	93 (12.8)	0	0 (0)	0 (0)	0 (0)
D	9	0	13	10	34 (12.1)	110 (31.2)	4	22 (15.4)	28 (13.1)	30 (0.1)
E*	9	1	13	10	25 (9.7)	88 (27.2)	9	20 (10.2)	35 (16.8)	30.1 (0.3)
F	12	6	20	4	28 (5.7)	73 (15.7)	5	14 (6.5)	20 (19.8)	30.9 (1.3)
G	14	1	12	10	34 (8.8)	66 (22.2)	5	15 (6.0)	23 (12.3)	30.1 (0.1)
H	17	7	24	6	35 (12.9)	100 (36.6)	0	0 (0)	0 (0)	0 (0)
I	19	3	9	6	50 (13.7)	130 (25.1)	1	23 (0)	35 (0)	30 (0)
J	23	6	30	9	35 (11.8)	99 (32.2)	3	20 (2.9)	54 (14.2)	30 (0)
K	25	4	27	9	49 (11.7)	130 (34.6)	0	0 (0)	0 (0)	0 (0)
L	26	5	28	9	43 (11.6)	100 (44.7)	0	0 (0)	0 (0)	0 (0)

chest discomfort or dry cough. One diver developed mild obstruction (FEV_1/FVC z-score declined from -1.16 to -1.93) by the end of the expedition. Another diver was already mildly obstructive at the beginning of the expedition. All parameters were still within the normal range, and both were asymptomatic.

VISUAL ACUITY

Thirty-six measurements of visual acuity were performed during the expedition. The number of measurements per diver varied between two and five depending on the length of their expedition. One diver was excluded from the analysis due to previous cataract operation. None of the included divers (n = 11) had any previous eye surgeries, trauma or infections within two months prior to the expedition. The measurements were conducted approximately weekly; however, for two divers they were performed less frequently due to logistical constraints. Two divers had no changes in their eyesight. The remaining nine divers had fluctuations throughout the expedition, mostly a one-line improvement or worsening in one eye. When the baseline measurements were compared to the last measurement, two divers had an improvement of one line with both eyes, whereas the rest had no changes. Thus, there was no change of visual acuity throughout the expedition.

Discussion

In this extended, 31-day field study, research divers participated in marine archaeological work consisting of CCR dives. It was found that some divers may develop mild, asymptomatic pulmonary obstruction during extended oxygen exposure. There were no significant changes in visual acuity observed. Thus, most divers completed the expedition safely without any major adverse effects.

OXYGEN EXPOSURE AND TOXICITY

Conservative dive planning and a limited number of dives per diver kept the oxygen exposure within an acceptable range. A new guideline suggests that the acceptable exposure time limit to manage risk of cerebral oxygen toxicity when diving at a constant PO_2 of 130 kPa is four hours of working dive and four hours of rest.[21] As the older and more conservative NOAA guidelines of CNS% were respected during this expedition,[2] it is not surprising that there were no major adverse effects related to oxygen toxicity. Both UPTD and ESOT had mean and maximum values below accepted limits. The current recommendations only consider up to seven days of consecutive diving due to limited evidence. In this dataset, the longest consecutive diving period was 10 days and there were no pulmonary adverse effects. ROT showed that by diving mostly once per day, divers did not accumulate oxygen exposure, as there was enough time for recovery. This can also be seen in the low probability of toxicity. In a recently published review, the total oxygen exposure in divers leading to myopic changes was higher (68 or 110

Table 3

Summary of daily oxygen exposure (based on a constant inspired PO_2 of 130 kPa) for different models presented as mean (standard deviation) and maximum; the daily limit recommendation is displayed if there is one. CNS (%) – percentage of the maximum allowed exposure for central nervous system toxicity; ESOT – equivalent surface oxygen time; ROT – residual oxygen time; UPTD – unit pulmonary toxic dose

Exposure model	Mean (SD)	Maximum	Daily limit	Ref no.
CNS (%)	58.2 (19.5)	97.8	100	2
UPTD	156.9 (52.7)	272.2	300	15
ESOT	190.9 (63.7)	320.6	450	6
ROT	1.8 (0.6)	3.1	–	7
ROT Probability of toxicity (%)	6.5 (1.9)	11.3	–	7

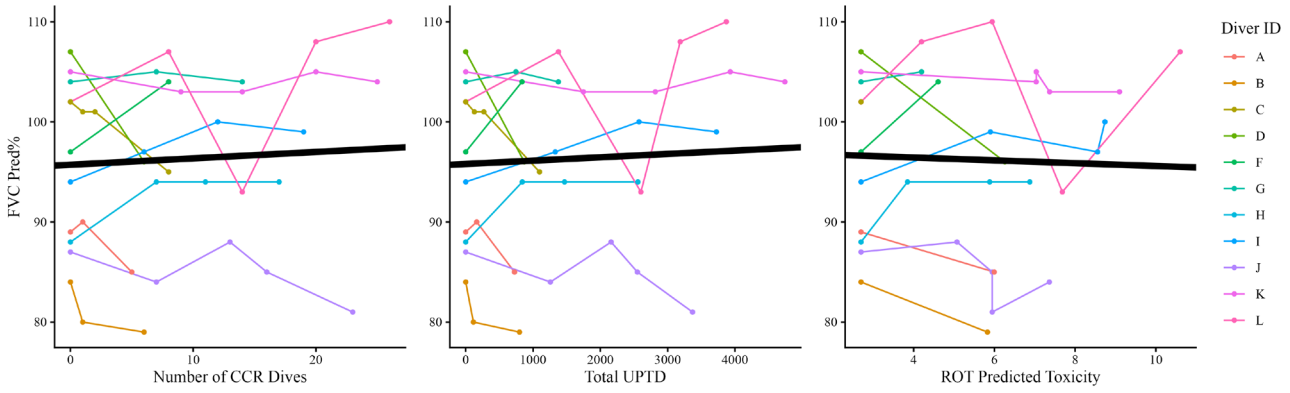
Table 4

Paired *t*-test results for the change in predicted spirometry values between the start and end of the diving periods of 11 divers. The *P*-value was adjusted using Bonferroni correction. CI – confidence interval, FEF_{75}% – forced expiratory flow of 75% of lung volume; $FEF_{25–75}$% – forced expiratory flow of 25–75% of lung volume; FVC – forced vital capacity; FEV_1 – forced expiratory volume during the first second, FEV_1/FVC – ratio of FEV_1/FVC

Variable	Estimate (95% CI)	*P*-value	Adjusted *P*-value
FVC Pred%	-0.72 (-5.06–3.61)	0.71	1
FEV_1 Pred%	-2.46 (-7.07–2.16)	0.26	1
FEV_1/FVC Pred%	-2.09 (-5.29–1.11)	0.18	0.88
FEF_{75} Pred%	-11.73 (-34.8–11.3)	0.28	1
$FEF_{25–75}$% Pred%	-7.55 (-20.0–4.89)	0.21	1

Figure 1

Predicted forced vital capacity (FVC Pred%) measurements plotted against three measures of accumulated oxygen exposure: number of closed-circuit rebreather (CCR) dives, total unit pulmonary toxic dose (UPTD) and residual oxygen time (ROT) predicted toxicity. The thick line shows the linear mixed model trendline for an average diver



hours of 100% O_2 at 101 kPa) compared to the mean of this group (32.0 [SD 20.5] hours of 100% O_2 at 101 kPa).[12]

LUNG FUNCTION

In this study, most of the divers did not have changes in their lung function, although one diver had a 7–44.8% decrease in multiple parameters. In addition, one diver developed mild obstruction, though it was clinically insignificant. It could be argued whether using general reference z-score -1.64 for the lung impairment is correct in this population. Wingelaar et al. demonstrated that, in annual fit-to-dive evaluations for military divers, applying a lower normal limit of 2.5% (z-score -1.96) was sufficient without compromising diver health. More extensive lung function testing did not identify any additional contraindications to diving when combined with detailed medical history taking.[22]

Table 5

Results of linear mixed-effects models with random intercepts for the divers and fixed effects specified in the 'Independent variable' column; the *P*-values were estimated using the Kenward-Roger method. CCR – closed-circuit rebreather; FVC Pred% – percentage of predicted forced vital capacity; ROT – residual oxygen time; UPTD – unit pulmonary toxic dose

Dependent variable	Independent variable	Estimate (95% CI)	*P*-value	Adjusted *P*-value	Marginal R^2 / Contitional R^2	*n* observations (*n* divers)
FVC Pred %	CCR *n* dives	0.064 (-0.12–0.25)	0.50	1	0.003 / 0.81	40 (11)
	Total UPTD	0.00034 (-0.00077–0.0015)	0.56	1	0.003 / 0.81	40 (11)
	ROT toxicity	-0.14 (-0.85–0.58)	0.67	1	0.001 / 0.80	34 (11)

The comparison of the first and last spirometry measurements of each diver using paired *t*-tests did not show any statistically significant changes. However, the confidence intervals were quite wide, especially for FEF_{75} Pred% and $FEF_{25–75}$ Pred%. Therefore, more evidence is needed to draw reliable conclusions. There were also no significant associations found between accumulated oxygen exposure and spirometry changes tested using regression models. This is not unexpected, as the pulmonary oxygen exposure was kept within safe limits, minimising the likelihood of large spirometry changes. Simultaneously, spirometry variation introduced by the long observation time, combined with a modest sample size, limits the statistical power for finding small changes in spirometry. Nevertheless, the regression model results and associated confidence intervals imply that similar diving expeditions are unlikely to result in clinically significant FVC Pred% changes in an average diver. This is most evident in the regression on the number of CCR dives were the estimated change in FVC Pred% was only 0.064 (95% CI -0.117–0.250) per dive.

Table 6 summarises studies using spirometry in evaluating lung function in divers.[14,23–29] The results shown were contradictory. It is argued that spirometry is not the most sensitive test to detect early signs of pulmonary toxicity.[30] Some studies measured diffusion capacity for carbon monoxide (D_LCO), which seems to be a more sensitive measurement for pulmonary oxygen toxicity.[28,30] Studies involved either a single bounce dive or 4–14 consecutive days of diving. Diver's subjective symptoms like coughing or chest discomfort, did not correlate with abnormal spirometry results.[14,28] Conversely, some individual divers showed deteriorated lung function - as was the case for one diver in our study – but no subjective symptoms.

The question of how to recognise divers that are at risk of developing lung dysfunction after extended diving expeditions remains. Increasing the surface time between long hyperoxic exposures was shown to decrease both subjective symptoms and the diffusion capacity for the carbon monoxide (D_LCO) regression slope.[28] Although these experiments were done in a pool with resting divers, the findings can be applied to working divers who are more prone to hyperoxic injury.[21,28] In working oxygen CCR divers, smaller airways (maximal expiratory flow at 25% [MEF_{25}] and maximal expiratory flow at 50% [MEF_{50}]) seemed more affected, suggesting long-term hyperoxic pulmonary injury.[31] Recent revised guidelines strongly recommend that if pulmonary oxygen toxicity symptoms develop, the diver should wait until fully recovered before resuming diving activity.[21]

VISUAL ACUITY

There were no significant changes in the visual acuity of divers during this expedition. Some fluctuation in the measurements was observed. However, this was most likely due to the natural fluctuation of eyesight. On some

Table 6

Summary of spirometry results after single or several days of diving; *the study had 14 divers providing 20 data sets collected in several expeditions in 2007–2010 and pooled together. CCR – closed-circuit rebreather; $FEF_{25–75}$% – forced expiratory flow at 25–75% of lung volume; FEV_1 – forced expiratory volume in one second; FVC – forced vital capacity; MEF_{25} – maximum expiratory flow when 25% of lung volume is left; O_2 -CCR – oxygen rebreather; OC – open-circuit; n.a. – not available; ssOC – surface supply OC; Unpub – unpublished data previously recorded by the authorship team

Ref	Divers (*n*)	Dives/diver (*n*)	Length (days)	Place and temperature	Depth (m)	Dive Time	Gas	OC/ CCR	Spirometry results
14	14/20*	n.a.	13	n.a.	69	112 min	PO_2 130–140 kPa	CCR	no change
23	26	1	1	n.a., 23–24°C	80–120	243–516 min	heliox	ssOC	FEV_1/FVC, MEF_{25} decreased to 89.2–87.1% 30 min after the dive, returned to baseline in 24 h
24	7	1–4	4	Tahiti, 27°C	90, 120	160, 180 min	trimix	CCR	FVC 84% vs. 91%, FEV_1/FVC 98% vs. 95%, other no change
25	8	8–9	5	Baltic, 16.5/8.5°C	19.7–64.7	54–99 min	air/trimix	CCR	no change
26	6	1	1	pool, 31–32°C	15	20 min	air/PO_2 140 kPa	OC/CCR	no change
27	11	2	2	n.a.	5	180 min	pO_2 150 kPa	O_2-CCR	no change
28	9	10	14	pool, 29–35°C	4.6	4 h / 20 h break	130–140 kPa	ssOC	resting, no change
28	16	5	5	pool, 29–35°C	4.6	6 h / 18 h break	130–140 kPa	ssOC	resting, regression line for FVC -0.4%/day
29	10	5	5	pool, 29–35°C	4.6	6 h / 18 h break	130 kPa	ssOC	exercising vs. resting incidence of changes in flow-volume parameters 17–30% vs. 10–20%
Present article	12	14 (5–26)	31	Micronesia, 30–31°C	36 (14–68)	95 (27–176) min	PO_2 70/130 kPa	CCR	FEV_1 decreased in two weeks, no other changes in the group, one diver had decline in $FEF_{25–75}$%
Unpub	9	1	1	Finland, 8/4°C	103	167 min	trimix	CCR	no change

days, the divers might have been more tired, or the lighting differed, as the tests were performed outside in natural light. Baseline measurements were taken after the long journey to Micronesia, when divers were fatigued, which might explain the improvement seen. The development of hyperoxic myopia has previously been documented in technical divers during rigorous diving expeditions.[13,14] Additionally, there has been a case of a recreational diver developing myopia on a diving vacation.[12]

There are a few possible explanations for no changes in the visual acuity. Firstly, the oxygen load remained low compared to HBOT patients, for whom long treatment protocols may cause transient myopisation. Additionally, breaks in the exposure (i.e., weekends), have been shown to allow partial recovery of vision before the next HBOT session begins.[32] As divers on this expedition predominantly dove once a day, there was recovery time between exposures. Furthermore, due to the low sensitivity and subjectivity of the Snellen chart, small changes may have been missed. An autorefractor could have provided more precise measurements but it was not available during the expedition. Thus, confounding factors may explain the fluctuations seen in the results. However, had there been major changes in vision, they would likely have been detected.

STRENGTHS AND LIMITATIONS

This study presents real-life data of working scientific CCR divers, collected over 31 days, in a remote location. There is currently little real-world evidence collected systematically for such a long period of time. However, the real-world setting comes at the expense of data quality.

The primary goal of the expedition was to collect archaeological, not physiological, data. Thus, data collection could not always be optimised: the time for spirometry and visual acuity measurements varied and spirometry could not be collected systematically eight hours post dive. A month of data collection includes a number of confounding factors that further added noise to the data: gear malfunctions, divers getting ill and plans changing. For example, test divers suddenly had to perform as OC safety divers due lack of support divers introducing mixed modality diving that we did not have the resources to account for. Finally, the expedition team was international, making follow-up for the affected divers impossible.

Moving forward, we hope to encourage the scientific community to continue data collection in a real-life setting. This type of evidence is needed to further solidify the oxygen exposure limits and hence, make diving safer.

Conclusions

This study demonstrates that diving expeditions can be executed safely, even in remote locations, when planned carefully and conservatively. Diving once a day and adhering to consensus guidelines for oxygen exposure led to a reasonable total accumulation of oxygen exposure that does not appear to have a substantial effect on lung function or visual acuity. However, more real-life, long-term evidence is required to confirm these conclusions and to detect more subtle changes.

References

1 Thorsen E, Haave H, Hofsø D, Ulvik RJ. Exposure to hyperoxia in diving and hyperbaric medicine--effects on blood cell counts and serum ferritin. Undersea Hyperb Med. 2001;28:57–62. PMID: 11908696.

2 NOAA Diving Manual: Diving for Science and Technology. U.S. Department of Commerce, National Oceanic and Atmospheric Administration; 1991. [cited 2026 Jan 20]. Available from: https://books.google.fi/books?id=_yGjgZGfMfEC.

3 Bardin H, Lambertsen CJA. A quantitative method for calculating cumulative pulmonary oxygen toxicity. Use of the “Unit Pulmonary Toxic Dose” (UPTD). University of Pennsylvania; 1970.

4 Arieli R. The pulmonary oxygen toxicity index. Respir Physiol Neurobiol. 2023;315:104114. doi: 10.1016/j.resp.2023.104114. PMID: 37460079.

5 Risberg J, van Ooij PJ. Hyperoxic exposure monitoring in diving: A farewell to the UPTD. Undersea Hyperb Med. 2022;49:395–413. doi: 10.22462/07.08.2022.1. PMID: 36446287.

6 Risberg J, van Ooij PJ, Mátity L. Recovery from pulmonary oxygen toxicity: a new (ESOT) model. Undersea Hyperb Med. 2024;51:407–23. PMID: 39821770.

7 Shykoff BE. Residual oxygen time model for oxygen partial pressure near 130 kPa (1.3 atm). Undersea Hyperb Med. 2015;42:547–64. PMID: 26742255.

8 van Ooij PJAM, Hollmann MW, van Hulst RA, Sterk PJ. Assessment of pulmonary oxygen toxicity: relevance to professional diving; a review. Respir Physiol Neurobiol. 2013;189:117–28. doi: 10.1016/j.resp.2013.07.014. PMID: 23886638.

9 Clark JM, Lambertsen CJ, Gelfand R, Flores ND, Pisarello JB, Rossman MD, et al. Effects of prolonged oxygen exposure at 1.5, 2.0, or 2.5 ATA on pulmonary function in men (predictive studies V). J Appl Physiol (1985). 1999;86:243–59. doi: 10.1152/jappl.1999.86.1.243. PMID: 9887137.

10 Schaal S, Beiran I, Rubinstein I, Miller B, Dovrat A. Lenticular oxygen toxicity. Invest Ophthalmol Vis Sci. 2003;44:3476–84. doi: 10.1167/iovs.03-0122. PMID: 12882797.

11 Cooper JS, Hendriksen S, Launico MV. Hyperbaric related myopia and cataract formation. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 Jan. PMID: 28613605.

12 Sokolowski SA, Räisänen-Sokolowski AK, Lundell RV. Development of myopia in scuba diving and hyperbaric oxygen treatment: a case report and systematic review. Diving Hyperb Med. 2024;54:328–37. doi: 10.28920/dhm54.4.328-337. PMID: 39675741. PMCID: PMC12018702.

13 Butler FK Jr, White E, Twa M. Hyperoxic myopia in a closed-circuit mixed-gas scuba diver. Undersea Hyperb Med. 1999;26:41–5. PMID: 10353183.

14 Fock A, Harris R, Slade M. Oxygen exposure and toxicity in recreational technical divers. Diving Hyperb Med. 2013;43:67–71. PMID: 23813459. [cited 2026 Jan 20]. Available from:

https://dhmjournal.com/images/IndividArticles/43June/Fock_dhm.43.2.67-71.pdf.
15 Hamilton R, Kenyon D, Peterson R. REPEX Habitat diving procedures: Repetitive vertical excursions, oxygen limits, and surfacing techniques. In: National Undersea Research Program Technical Report 88-1B. NOAA and U.S. Department of Commerce; 1988.
16 The Diving Medical Advisory Committee. Exposure index for pulmonary oxygen toxicity in surface-oriented diving. Published online February 2025. [cited 2026 Jun 3]. Available from: https://www.dmac-diving.org/guidance/.
17 Stanojevic S, Kaminsky DA, Miller MR, Thompson B, Aliverti A, Barjaktarevic I, et al. ERS/ATS technical standard on interpretive strategies for routine lung function tests. Eur Respir J. 2022;60(1):2101499. doi: 10.1183/13993003.01499-2021. PMID: 34949706.
18 Bates D, Mächler M, Bolker B, Walker S. Fitting linear mixed-effects models using lme4. Journal of Statistical Software. 2015;67(1):1–48. doi: 10.18637/jss.v067.i01.
19 R Core Team. A language and environment for statistical computing. Published online 2026. [cited 2026 Mar 15]. Available from: https://www.R-project.org.
20 Wickham H. ggplot2: Elegant graphics for data analysis. Published online 2016. [cited 2026 Apr 17]. Available from: https://ggplot2.tidyverse.org.
21 Hoyt JT, Murphy FG, Pollock NW, Kernagis D, Bird N, Menduno M, et al. Revised guideline for central nervous system oxygen toxicity exposure limits when using an inspired PO_2 of 1.3 atmospheres. Diving Hyperb Med. 2025;55:262–70. doi: 10.28920/dhm55.3.262-270. PMID: 40986922. PMCID: PMC12263281.
22 Wingelaar TT, van Ooij PJA, Endert EL. The lower limit for FEV_1/FVC in dive medical assessments: a retrospective study. Diving Hyperb Med. 2021;51:368–72. doi: 10.28920/dhm51.4.368-372. PMID: 34897602. PMCID: PMC8920896.
23 Bao XC, Yang T, Fang YQ, Sun YJ, Wang N. Lung function changes in divers after a single deep helium-oxygen dive. Diving Hyperb Med. 2022;52:183–90. doi: 10.28920/dhm52.3.183-190. PMID: 36100929. PMCID: PMC9731142.
24 Dugrenot E, Balestra C, Gouin E, L'Her E, Guerrero F. Physiological effects of mixed-gas deep sea dives using a closed-circuit rebreather: a field pilot study. Eur J Appl Physiol. 2021;121:3323–31. doi: 10.1007/s00421-021-04798-y. PMID: 34435274.
25 Gouin E, Balestra C, Orsat J, Dugrenot E, L'Her E. Pulmonary effects of one week of repeated recreational closed-circuit rebreather dives in cold water. Medicina (Kaunas). 2023;59(1):81. doi: 10.3390/medicina59010081. PMID: 36676705. PMCID: PMC9864030.
26 Bosco G, Rizzato A, Quartesan S, Camporesi E, Mrakic-Sposta S, Moretti S, et al. Spirometry and oxidative stress after rebreather diving in warm water. Undersea Hyperb Med. 2018;45:191–8. PMID: 29734571.
27 van Ooij PJAM, van Hulst RA, Houtkooper A, van der Weide TJS, Sterk PJ. Lung function before and after oxygen diving: a randomized crossover study. Undersea Hyperb Med. 2012;39:699–7. PMID: 22670550.
28 Shykoff BE. Pulmonary effects of submerged oxygen breathing in resting divers: repeated exposures to 140 kPa. Undersea Hyperb Med. 2008;35:131–43. PMID: 18500077.
29 Shykoff BE, Florian JP. Pulmonary effects of repeated six-hour normoxic and hyperoxic dives. PLoS One. 2018;13(9):e0202892. doi: 10.1371/journal.pone.0202892. PMID: 30192774. PMCID: PMC6128531.
30 van Ooij PJAM, van Hulst RA, Houtkooper A, Sterk PJ. Differences in spirometry and diffusing capacity after a 3-h wet or dry oxygen dive with a PO(2) of 150 kPa. Clin Physiol Funct Imaging. 2011;31:405–10. doi: 10.1111/j.1475-097X.2011.01034.x. PMID: 21771262.
31 Tetzlaff K, Friege L, Reuter M, Haber J, Mutzbauer T, Neubauer B. Expiratory flow limitation in compressed air divers and oxygen divers. Eur Respir J. 1998;12:895–9. doi: 10.1183/09031936.98.12040895. PMID: 9817165.
32 Evanger K, Vaagbø G, Haugen OH. Short-term effects on ocular variables immediately after hyperbaric oxygen exposures. Undersea Hyperb Med. 2018;45:395–402. PMID: 30241118.

Acknowledgements

The authors thank the participating divers of Major Projects Foundation, Australia, and Ms Anjé Clarke of Inkfish for organisational support of the study project. Morag Tolvi is thanked for reviewing the English language.

Conflicts of interest and funding

No conflicts of interest were declared. The study was funded by InkFish LLC, a non-profit organisation, which also provided staff support during data collection. Sofia Sokolowski-Wojnicki received a personal grant from The Finnish Society of Diving and Hyperbaric Medicine.

Submitted: 21 January 2026
Accepted after revision: 5 June 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

Scuba-related injuries presenting to United States emergency departments: paediatric and adult patterns

Andy Tom[1], Sam Lindberg[1], Sergio Navarro[2], Daniel Stephens[2]

[1] *Mayo Clinic Alix School of Medicine, Mayo Clinic, Rochester, Minnesota, USA*
[2] *Division of Trauma, Critical Care, and General Surgery, Department of Surgery, Mayo Clinic, Rochester, Minnesota, USA*

Corresponding author: *Dr Daniel Stephens, Mayo Clinic Division of Trauma, Critical Care, and General Surgery, Mail Station: RONT02800-TR, 200 First Street SW, Rochester, MN, 55905, USA*
ORCiD: *0000-0002-6980-0001*
stephens.daniel@mayo.edu

Keywords
Arterial gas embolism; Barotrauma; Children; Decompression sickness; Drowning; Epidemiology; Pediatric divers

Abstract
(Tom A, Lindberg S, Navarro S, Stephens D. Scuba-related injuries presenting to United States emergency departments: paediatric and adult patterns. Diving and Hyperbaric Medicine. 2026 30 September;56(3):303–308. doi: 10.28920/dhm56.3.303-308. PMID: 42743570.)

Introduction: Scuba diving is a popular sport that carries risks unique to the underwater environment. Paediatric emergency department presentations for scuba diving injuries are incompletely characterised, particularly in comparison with adults.

Methods: We retrospectively analysed scuba-related injuries recorded in the National Electronic Injury Surveillance System (NEISS) from 2005–2024. We estimated national paediatric emergency department presentations using NEISS weights and compared injury categories and hospitalisation rates between paediatric and adult patients.

Results: We identified 68 paediatric cases, corresponding to an estimated 122 paediatric scuba-related emergency department presentations annually. Trauma was the most common injury category (41.2%), followed by otitis (17.6%) and ear, sinus, ocular, or dental barotrauma (16.2%). Hospitalisation was most common among patients with possible decompression sickness or arterial gas embolism (DCS/AGE) (42.9%) and drowning/aspiration (25.0%). Compared with adults, paediatric patients were less often hospitalised (10.3% vs 21.4%, $P = 0.038$), more likely to present with traumatic injuries (41.2% vs 26.1%, $P = 0.010$), and less likely to present with possible DCS/AGE (10.3% vs 25.0%, $P = 0.006$).

Conclusions: Among patients presenting to United States emergency departments with scuba-related injuries, paediatric patients most commonly had trauma, otitis, or ear, sinus, ocular, or dental barotrauma. Paediatric divers were also less likely than adults to experience DCS/AGE injuries and were less likely than adults to require hospitalisation. Our data describes emergency department presentation patterns rather than overall injury risk among paediatric scuba divers.

Introduction

Scuba diving is a popular sport with inherent hazards. Historical surveys estimated that approximately three million people in the United States participate annually, although contemporary participation may differ from these pre-COVID estimates.[1] Most were casual participants completing one to seven dives per year.[1] In addition to trauma and musculoskeletal injuries common to many sports, scuba diving carries risks related to changes in ambient pressure, inert gas bubble formation, and environmental hazards.

Diving injuries may occur during descent, at depth, or during ascent. During descent, rising ambient pressure requires equalisation of gas-filled spaces; failure to equalise the middle ear can cause pain, haemorrhage, or tympanic membrane rupture. Middle-ear barotrauma is among the most common diving complications and may be especially relevant in paediatric divers.[2–3] During ascent, trapped gas expands as ambient pressure decreases according to Boyle's law. Breath-holding or impaired gas escape can cause pulmonary overinflation syndromes, including pneumothorax, pneumomediastinum, subcutaneous emphysema, and arterial gas embolism.[4]

Decompression sickness (DCS) occurs when inert gas comes out of solution during or after decompression, forming bubbles in tissues or the circulation. Manifestations may be musculoskeletal, cutaneous, lymphatic, neurologic, vestibular, cardiopulmonary, or constitutional. Although traditionally divided into Type I and Type II disease, this classification may oversimplify the clinical spectrum.[5] Treatment often requires recompression in a hyperbaric chamber.[6]

In addition to pressure-related injuries, the diving environment itself poses significant hazards. Equipment failure or injury underwater can lead to hypoxia, aspiration, drowning, and death. Marine animal encounters, while uncommon, may also result in serious injury.

Adult scuba-related morbidity and mortality in the United States and Canada have been described, but paediatric data remain limited.[7] Prior paediatric studies have used different data sources, shorter study periods, or lacked direct comparison with adult emergency department presentations.[8]

The Professional Association of Diving Instructors (PADI) offers diving experiences for paediatric divers as young as eight years of age; however, even younger divers may participate in 'backyard' scuba diving outside formal training offered by professional associations.[9] Serious injuries, including arterial gas embolism, can occur even during shallow-water pool dives when appropriate safety measures are not in place.[10–11] Paediatric divers may be more likely to sustain diving injuries if they do not fully understand or appreciate the hazards of scuba diving and its unique physiological risks.

Paediatric divers may also differ from adults in supervision, training pathway, dive depth, medical screening, experience, and exposure patterns. We therefore hypothesised that paediatric and adult patients presenting to emergency departments after scuba-related incidents would differ in injury patterns and hospitalisation rates. This study characterised paediatric scuba-related emergency department presentations from 2005–2024, estimated national paediatric presentation counts using NEISS weights, and compared paediatric and adult injury patterns and hospitalisation rates.

Methods

This study met criteria for institutional review board exemption because it used publicly available, deidentified data without patient contact or protected health information. Exemption (26-003659) was received on April 3, 2026.

We performed a retrospective analysis of scuba-related injuries presenting to emergency departments (EDs) in the United States. We extracted data from the National Electronic Injury Surveillance System (NEISS).[12] The NEISS is a publicly available database that collects statistics on consumer product-related injuries. At participating hospitals, a professional research coordinator records information including patient demographics, injury setting, body part injured, and the product or activity associated with an injury. Reports also include a short narrative describing the events leading to the injury and, in some cases, an emergency department diagnosis. Approximately 100 hospitals across the United States form a representative sample from which national estimates can be calculated. The participating hospitals are selected and maintained by the Consumer Product Safety Commission as part of the NEISS sampling frame; they were not selected by the study authors.

Each NEISS case is assigned a sampling weight based on hospital stratum and emergency department volume. We summed these weights to estimate national emergency department presentations during the study period. We calculated mean annual presentations by dividing the national estimate by the length of the study period in years.[13]

We queried the NEISS database for all records assigned the scuba diving product code (product code 1275) from January 1, 2005, through December 31, 2024. Case identification was based solely on the NEISS product code. We divided the cohort into paediatric (age ≤ 18 years) and adult (age > 18 years) sub-cohorts. Narratives and diagnosis fields were manually reviewed, and each case was assigned to a prespecified injury category (Table 1).

We categorised injuries using the NEISS narrative, diagnosis field when available, body part, disposition, and contextual details such as reported depth, ascent history, or aspiration. Categories included trauma; ear, sinus, ocular, or dental barotrauma; pulmonary barotrauma; possible DCS or arterial gas embolism (AGE); otitis; marine animal injury; drowning/aspiration; immersion pulmonary oedema; other medical presentations; and undifferentiated post-dive symptoms. Otitis included otitis externa and otitis media when specified. Undifferentiated post-dive symptoms included chest pain, musculoskeletal pain, or central nervous system symptoms when the available information was insufficient to support a more specific diving-related diagnosis.

We grouped DCS and AGE because NEISS narratives often lack sufficient detail to distinguish them reliably, and both may present after ascent with neurologic, cardiopulmonary, or systemic symptoms requiring urgent evaluation. Because these conditions differ in mechanism, severity, prognosis, and management, the combined category should be interpreted as a pragmatic surveillance classification rather than a definitive clinical diagnosis.

A professional diver with more than 10 years of experience reviewed the narratives. Cases with insufficient detail were classified conservatively, and uncertain cases were adjudicated by a physician investigator.

We reported descriptive statistics for our cohorts including age, sex, race, injury type, maximum reported depth when available, and disposition (hospitalisation, fatality, treated and released/discharged from the ED, or left without being seen). Fatalities were retained if recorded in NEISS disposition; however, NEISS captures only deaths associated with ED presentation and should not be considered a comprehensive fatality surveillance system for scuba diving.

We calculated weighted national estimates of ED presentations using the NEISS-provided weight for each case and compared paediatric and adult injuries during the study period. Categorical variables, including sex distribution, hospitalisation, fatality, and injury category, were compared using Fisher's exact test. All comparative statistical analyses were performed on unweighted data. Weighted data were used only to generate descriptive national estimates. Survey-weighted comparative analyses were considered; however,

Table 1
Injury-classification definitions and representative paraphrased NEISS (National Electronic Injury Surveillance System) narratives for scuba-related emergency department presentations, United States, 2005–2024

Group	Description	Representative NEISS narrative, paraphrased
Trauma	Abrasions, lacerations, fractures, contusions, and other mechanical injuries.	*A 16-year-old male struck his head twice on a scuba tank while diving.*
Ear/sinus/ocular/dental barotrauma	Tympanic membrane rupture, middle-ear or sinus barotrauma, subconjunctival haemorrhage, and dental barotrauma.	*A 13-year-old female experienced an ear pop during a scuba class and was diagnosed with a ruptured tympanic membrane.*
Pulmonary barotrauma	Pneumomediastinum, subcutaneous emphysema, pneumothorax, or alveolar haemorrhage attributed to pressure-related lung injury.	*A 20-year-old male developed chest pain while diving at 7.6 m and was diagnosed with pneumomediastinum.*
Possible decompression sickness or arterial gas embolism	An emergency department diagnosis of decompression sickness or arterial gas embolism, or rapid ascent followed by immediate compatible symptoms when a definitive diagnosis was not documented.	*A diver surfaced rapidly from 15.2 m and presented with altered mental status after the dive.*
Otitis	Otitis externa or otitis media when specified in the diagnosis or narrative.	*A 12-year-old male developed left ear pain after scuba diving at camp and was diagnosed with otitis.*
Marine animal injury	A bite, sting, envenomation, or contact injury caused by a marine animal.	*A 17-year-old female sustained a sea-urchin spine injury while scuba diving and subsequently developed a skin infection.*
Undifferentiated post-dive symptoms	Chest pain, musculoskeletal pain, or central nervous system symptoms after diving when the available narrative did not support a more specific diving-related diagnosis.	*A 13-year-old male developed discomfort in the elbows, hands, and knees during scuba lessons without sufficient information for a more specific diagnosis.*
Immersion pulmonary edema	A documented diagnosis of immersion pulmonary oedema.	*A 46-year-old female developed worsening dyspnoea during a dive to 22.6 m and was diagnosed with immersion pulmonary edema.*
Drowning/aspiration	Aspiration, submersion injury, drowning, or near drowning associated with scuba-related activity.	*A 14-year-old male became frightened while practicing regulator removal at a depth of 3.7 m during a scuba class, experienced a seizure, and was diagnosed with near drowning.*
Other medical presentations	Medical presentations temporally associated with scuba activity but not attributed to diving physiology.	*A 68-year-old male sustained facial burns when a cigarette ignited while he was wearing oxygen.*

small numbers within several paediatric injury categories limited the stability of weighted estimates. We therefore used unweighted case-level data for comparative analyses and weighted data only for descriptive national estimates. Accordingly, *P*-values reflect comparisons within the observed NEISS sample. All analyses were conducted in Python.

Results

COHORT

We identified 68 paediatric scuba-related emergency department visits in the NEISS sample from 2005–2024 (Table 2), corresponding to an estimated 2,442 national presentations during the study period, or 122 annually.

Table 2
Demographic characteristics and disposition of paediatric and adult scuba-related emergency department presentations in the NEISS (National Electronic Injury Surveillance System) sample, United States, 2005–2024; IQR – interquartile range

Variable	Paediatric (≤ 18 years)	Adult (> 18 years)	*P*-value
Observed cases, *n*	68	617	–
Weighted national estimate, 2005–2024	2,442	25,917	–
Estimated annual presentations	122	1,296	–
Male sex, *n*/N (%)	48/68 (70.6%)	454/617 (73.6%)	0.567
Median age, years (IQR)	13 (10–16)	40 (29–51)	–
Hospitalization, *n*/N (%)	7/68 (10.3%)	132/617 (21.4%)	0.038
Fatality, *n*/N (%)	0/68 (0.0%)	7/617 (1.1%)	1.000

Table 3
Injury patterns among paediatric and adult scuba-related emergency department presentations in the NEISS (National Electronic Injury Surveillance System) sample, United States, 2005–2024; *$P < 0.05$; AGE – arterial gas embolism; DCS decompression sickness

Injury category	Paediatric, *n* (%)	Adult, *n* (%)	*P*-value
Trauma	28 (41.2%)	161 (26.1%)	0.010*
Possible DCS/AGE	7 (10.3%)	154 (25.0%)	0.006*
Ear/sinus/ocular/dental barotrauma	11 (16.2%)	116 (18.8%)	0.742
Otitis	12 (17.6%)	73 (11.8%)	0.175
Drowning/aspiration	4 (5.9%)	35 (5.7%)	1.000
Undifferentiated post-dive symptoms	4 (5.9%)	37 (6.0%)	1.000
Other medical presentations	1 (1.5%)	17 (2.8%)	1.000
Marine animal injury	1 (1.5%)	12 (1.9%)	1.000
Pulmonary barotrauma	0 (0.0%)	8 (1.3%)	1.000
Immersion pulmonary oedema	0 (0.0%)	4 (0.6%)	1.000

The paediatric cohort was 70.6% male (n = 48) and had a median age of 13 years (interquartile range 10–16). Race was recorded as White in 52.9% (n = 36), Black/African American in 1.5% (n = 1), Asian in 1.5% (n = 1), and other in 2.9% (n = 2); race was unspecified in 41.2% (n = 28). Dive depth was reported for 11 cases, with a mean (SD) of 12.3 (8.4) m and a maximum of 29.9 m.

Trauma was the most common injury category (41.2%, n = 28), followed by otitis (17.6%, n = 12), ear/sinus/ocular/dental barotrauma (16.2%, n = 11), possible DCS or AGE (10.3%, n = 7), and drowning/aspiration (5.9%, n = 4). Undifferentiated post-dive symptoms occurred in four patients (5.9%); chest pain in two, musculoskeletal pain in one, and central nervous system symptoms in one. Marine animal injury and other medical presentations each occurred in one patient (1.5%). No paediatric cases involved pulmonary barotrauma or immersion pulmonary oedema.

Overall, seven paediatric patients (10.3%) were hospitalised. Hospitalisation occurred in three of seven patients with possible DCS/AGE (42.9%), one of four with drowning/aspiration (25.0%), and three of 28 with trauma (10.7%). No hospitalisations occurred among patients with otitis, ear/sinus/ocular/dental barotrauma, marine animal injury, other medical presentations, or undifferentiated post-dive symptoms.

COMPARISON TO ADULT INJURIES

Paediatric patients accounted for 68 of 685 scuba-related emergency department visits (9.9%) from 2005–2024 (Table 3). Sex distribution was similar between paediatric and adult patients (70.6% vs 73.6% male, P = 0.567), but paediatric patients were less often hospitalised (10.3% vs 21.4%, P = 0.038). They were more likely to present with trauma (41.2% vs 26.1%, P = 0.010) and less likely to present with possible DCS/AGE (10.3% vs 25.0%, P = 0.006). Other injury categories did not differ significantly between groups. No paediatric cases of pulmonary barotrauma or immersion pulmonary oedema were identified, compared with eight and four adult cases, respectively. No paediatric fatalities were recorded, compared with seven adult fatalities (Table 2).

Discussion

Our study investigated paediatric scuba-related injuries presenting to emergency departments in the United States.

We hypothesised that paediatric divers would be more likely than adults to present with severe diving injuries because of differences in experience and understanding of diving physiology. Instead, paediatric patients in our cohort were less likely to be hospitalised or to present with DCS/AGE and were more likely to present with trauma. Rates of ear/sinus/ocular/dental barotrauma, otitis, and drowning/aspiration did not differ between groups.

These findings describe patterns among injured patients presenting to emergency departments and should not be interpreted as evidence that paediatric scuba diving carries lower overall injury risk. Potential explanations include structured paediatric safeguards, such as instructor supervision, age-based training limits, depth restrictions, medical screening, and staged progression to open-water diving. Adults may also have more variable exposure, dive profiles, comorbidities, training recency, and supervision. Because NEISS does not capture these factors, they remain possible explanations rather than conclusions derived from this study.

Our findings were partially consistent with prior paediatric diving research. In a review of Divers Alert Network calls involving minors, final diagnoses were most often ear, nose, throat, or sinus conditions (32%) or musculoskeletal trauma (16%), while DCS and AGE accounted for 11% of cases.[14] The proportion of possible DCS/AGE in our paediatric cohort was similar (10.3%), but trauma was more common (41.2%). This difference may reflect the data sources: emergency department surveillance captures routine injuries such as lacerations and fractures, whereas minor otic symptoms and equalisation problems may be more likely to prompt a DAN call, outpatient evaluation, or self-care. Buzzacott et al. likewise found mechanical trauma to be the most common injury category, although their reported DCS rate was lower than the overall DCS/AGE proportion in our combined cohort (6% vs 23.5%).[7] Their stricter DCS definition may explain this difference and highlights the difficulty of distinguishing DCS, AGE, and undifferentiated post-dive symptoms from brief surveillance narratives.

Pulmonary barotrauma warrants specific mention despite the absence of paediatric cases in our cohort. Serious paediatric pulmonary barotrauma and AGE have been reported after shallow or first-time scuba exposure, including pool-based dives.[10,11] In our study, pulmonary barotrauma occurred only among adults; however, this likely reflects the small paediatric sample, the rarity of the outcome, and the ED-based sampling frame rather than an absence of risk. Immersion pulmonary oedema was also absent in paediatric patients, which may be consistent with its association with older age and comorbidity, although this comparison is limited by small numbers.[15]

Current recommendations from medical organisations emphasise a cautious, highly supervised approach to paediatric scuba diving. The South Pacific Underwater Medicine Society (SPUMS) recommends that paediatric divers younger than 10 years old should not scuba dive, and that all individuals under 15 undergo formal medical risk assessment and evaluation of physical and psychological maturity.[16] They recommend against paediatric divers diving with epilepsy, congenital heart defects, insulin-dependent diabetes, structural lung disease, behavioral disorders, bleeding disorders, tympanostomy tubes, or migraine with aura. The Dutch Society for Diving and Hyperbaric Medicine (DSDHM) also provides guidance, recommending pool-based diving for paediatric divers aged 8–10, limited warm-water open-water diving for ages 10–12, and progressively broader participation after age 12 while still avoiding deep, cold, or high-risk dives and avoiding decompression dives until age 16.[17] Although recommendations from the SPUMS and the DSDHM differ in specific age thresholds and restrictions, they provide a good framework for safe paediatric diving.

Our study has several limitations. NEISS was designed to capture consumer product-related injuries rather than diagnoses specific to diving physiology. As such, the brief, non-standardised narratives sometimes lacked enough detail to distinguish decompression sickness, arterial gas embolism, pulmonary barotrauma, immersion pulmonary oedema, and undifferentiated post-dive symptoms reliably. Although cases were classified conservatively, results depended on both emergency department recognition of diving-related diagnoses and reviewer interpretation of the available narratives. The paediatric cohort was also small, with 68 cases and seven hospitalisations over 20 years, limiting comparisons of rare outcomes; therefore, the absence of paediatric pulmonary barotrauma or immersion pulmonary oedema should not be interpreted as evidence that these injuries do not occur. NEISS also lacks exposure data, including the number of paediatric divers or dives, certification status, supervision, experience, training pathway, and dive profile, so this study cannot estimate absolute injury risk or determine whether paediatric diving is safer than adult diving. In addition, scuba exposure is not evenly distributed across the United States, and differences between coastal and landlocked regions may influence regional presentation patterns. Access to dive medicine professionals and hyperbaric consultation or treatment may also affect diagnostic recognition, transfer, and hospitalisation decisions. Additionally, the NEISS includes injuries related to scuba equipment that did not occur while in the water, such as an injury that occurred while moving dive equipment. We felt it was important to include these injuries because although they did not occur in the water, they are still a hazard of the sport. Lastly, the NEISS only captures fatalities associated with emergency department presentation and is not a comprehensive source for paediatric scuba-related mortality.

Conclusions

Contrary to our hypothesis, paediatric patients presenting to United States emergency departments with scuba-related injuries were more likely than adults to present with trauma and less likely to present with possible DCS or AGE. They were also less likely to be hospitalised. These findings describe emergency department presentation patterns rather than overall injury risk among paediatric scuba divers. Supervision and adherence to safe diving practices remain essential, and further research is needed to characterise paediatric scuba-related fatalities.

References

1 Divers Alert Network. Participation in recreational scuba diving [Internet]. 2016 Jan 14. [cited 2026 Jun 21]. Available from: https://dan.org/safety-prevention/diver-safety/divers-blog/participation-in-recreational-scuba-diving/.

2 Winkler B, Tetzlaff K, Muth CM. Accidents and incidents during SCUBA-diving events in children. German Journal of Sports Medicine. 2011;62:42–6.

3 Vandenhoven G, Collard F, Schamp E. Children and diving: medical aspects. Eight years' sports medical follow-up of the first scuba diving club for children in Belgium. SPUMS Journal. 2003;33:70–3. [cited 2026 Jun 21]. Available from: https://dhmjournal.com/images/IndividArticles/33June/Vandenhoven_dhm.33.2.70-73.pdf.

4 Kennedy-Little D, Sharman T. Pulmonary barotrauma. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 Jan. [updated 2023 Jun 26]. [cited 2026 Jun 21]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK559293/. PMID: 32644719.

5 Francis TJR, Smith DH, editors. Describing decompression illness: the forty-second Undersea and Hyperbaric Medical Society workshop. Bethesda (MD): Undersea and Hyperbaric Medical Society; 1991.

6 Moon RE, Mitchell SJ. Decompression sickness: current recommendations. Undersea Hyperb Med. 2025;52:55–64. PMID: 40249722.

7 Buzzacott P, Schiller D, Crain J, Denoble PJ. Epidemiology of morbidity and mortality in US and Canadian recreational scuba diving. Public Health. 2018;155:62–8. doi: 10.1016/j.puhe.2017.11.011. PMID: 29306625.

8 Buzzacott P, Chimiak J, Nochetto M, Denoble PJ. Paediatric scuba-related morbidity at US emergency departments. Ann Epidemiol. 2017;27:539. doi: 10.1016/j.annepidem.2017.07.082.

9 Professional Association of Diving Instructors. Bubblemaker. [Internet]. [cited 2026 Jun 21]. Available from: https://store.padi.com/en-us/ns/courses/bubblemaker/p/bubblemaker/.

10 Johnson V, Adkinson C, Bowen M, Ortega H. Should children be SCUBA diving?: Cerebral arterial gas embolism in a swimming pool. Pediatr Emerg Care. 2012;28:361–2. doi: 10.1097/PEC.0b013e31824d9d14. PMID: 22472651.

11 Le Guen H, Halbert C, Gras Le Guen C, Coulange M. Serious pulmonary barotrauma in a child after first-time scuba dive. Arch Pediatr. 2012;19:733–5. doi: 10.1016/j.arcped.2012.04.014. PMID: 22682518.

12 United States Consumer Product Safety Commission. NEISS frequently asked questions [Internet]. [cited 2026 Jun 21]. Available from: https://www.cpsc.gov/Research--Statistics/NEISS-Injury-Data/Neiss-Frequently-Asked-Questions.

13 Schroeder T, Ault K. The NEISS sample: design and implementation, 1997 to present [Internet]. Bethesda (MD): U.S. Consumer Product Safety Commission; 2001. [cited 2026 June 21]. Available from: https://www.cpsc.gov/s3fs-public/2001d010-6b6.pdf.

14 Helfrich ET, Saraiva CM, Chimiak JM, Nochetto M. A review of 149 Divers Alert Network emergency call records involving diving minors. Diving Hyperb Med. 2023;53:7–15. doi: 10.28920/dhm53.1.7-15. PMID: 36966517. PMCID: PMC10318175.

15 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.

16 Elliott E, Smart D, Lippmann J, Banham N, Nochetto M, Roehr S. South Pacific Underwater Medicine Society (SPUMS) position statement regarding paediatric and adolescent diving. Diving Hyperb Med. 2024;54:338–43. doi: 10.28920/dhm54.4.338-343. PMID: 39675742. PMCID: PMC11779525.

17 Buwalda M, Querido AL, van Hulst RA. Children and diving, a guideline. Diving Hyperb Med. 2020;50:399–404. doi: 10.28920/dhm50.4.399-404. PMID: 33325022. PMCID: PMC8026229.

Conflicts of interest and funding: nil

Submitted: 16 April 2026
Accepted after revision: 19 July 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

Prevalence of laryngopharyngeal reflux symptoms in divers

Bastien A Valencia-Sanchez[1], Natalie Weiss[1], Evelyn Echevarria Cruz[2], Raana Zakeri[3,4], Frauke Tillmans[3], Amy Rutt[1]

[1] *Department of Otolaryngology – Head and Neck Surgery, Mayo Clinic Florida, Jacksonville, FL, USA*
[2] *Nova Southestern University – Kiran C. Patel College of Osteopathic Medicine, Davie, FL, USA*
[3] *Divers Alert Network, Durham, NC, USA*
[4] *Department of Epidemiology, UNC Gillings School of Global Public Health, University of North Carolina at Chapel Hill, NC, USA*

Corresponding author: *Dr Amy Rutt, Department of Otolaryngology – Head & Neck Surgery, Mayo Clinic in Florida, 4500 San Pablo Rd, Jacksonville, FL 32224, USA*
ORCiD: *0000-0002-7704-6328*
rutt.amy@mayo.edu

Keywords
DAN – Divers Alert Network; Diving medicine; ENT; Epidemiology; Health surveys; Recreational divers; Technical diving

Abstract
(Valencia-Sanchez BA, Weiss N, Echevarria Cruz E, Zakeri R, Tillmans F, Rutt A. Prevalence of laryngopharyngeal reflux symptoms in divers. Diving and Hyperbaric Medicine. 2026 30 September;56(3):309–316. doi: 10.28920/dhm56.3.309-316. PMID: 42743571.)
Introduction: Laryngopharyngeal symptoms (LPS) refer to aerodigestive complaints that may result from the retrograde flow of gastric contents into the upper aerodigestive tract. These symptoms may be under-recognised in populations exposed to mechanical or environmental stressors. Divers may be at increased risk due to factors such as pressure changes and body positioning. This study characterised the prevalence and severity of LPS among divers and identified associated risk factors.
Methods: A cross-sectional, anonymous survey was distributed to members of the Divers Alert Network (DAN). Collected data included demographics, diving-related behaviours, medical history, and LPS burden measured using the Reflux Symptom Index (RSI).
Results: Of the 309 survey responses analysed, 47.2% were male with a median age of 50.0 years (IQR: 40.0–60.0). Among 291 divers with complete RSI data, 26.1% (95% CI, 21.4–31.5%) had abnormal RSI scores, representing a +16.0 percentage-point increase (95% CI, 11.3%–21.4%) over non-diving population estimates ($P < 0.001$). In multivariable analysis, female sex (adjusted prevalence rate [aPR] 1.59, 95% CI 1.07–2.37) and gastroesophageal reflux disease (GERD) history (aPR 1.75, 95% CI 1.16–2.65) were independent predictors. Recreational divers had a lower risk (aPR 0.59, 95% CI 0.39–0.90). Approximately 5% of respondents described their symptoms as life-threatening.
Conclusions: Divers may represent an under-recognised population at elevated risk for LPS. Female sex and GERD history were associated with greater symptom burden, while recreational diving appeared less provocative compared to technical diving. These findings may have implications for risk stratification and symptom management in the diving community.

Introduction

Laryngopharyngeal reflux disease (LPRD) is characterised by the retrograde flow of gastric contents into the upper aerodigestive tract. Unlike gastroesophageal reflux disease (GERD), LPRD often presents without classic heartburn or regurgitation symptoms and is characterised by laryngopharyngeal symptoms (LPS) such as chronic cough, throat clearing, and hoarseness, in conjunction with objective evidence linking these symptoms to gastroesophageal reflux.[1–3] LPRD remains a challenging condition to manage, as definitive diagnostic and therapeutic gold standards have yet to be established.[4] Despite increased recognition of LPRD's clinical and economic burden, certain populations may face elevated risk due to mechanical or environmental exposures.

Underwater diving has the potential to exacerbate gastroesophageal reflux through several mechanisms. First, tight wetsuits and increased ambient pressure experienced during underwater descent may increase intra-abdominal pressure through mechanical compression.[5] Prone or head-down positions during diving can also favor the upward migration of gastric contents. On ascent, expanding gas in the stomach can trigger belching or vomiting in divers with weak lower oesophageal sphincters or full stomachs.[6] Though limited, prior forensic reports and series have suggested an association between underwater diving and life-threatening reflux-related events.[6–9] Despite this, there has been no systematic assessment of LPS burden in the diving population to date.

To address this gap, we conducted an international, cross-sectional survey among adult members of the Divers Alert

Network (DAN), a non-profit organisation dedicated to improving the safety of the diving community. The primary objective was to characterise the prevalence and severity of LPS in divers using the validated Reflux Symptom Index (RSI).[10] Secondary objectives included examining associations between abnormal RSI scores and demographic, medical, and diving-related variables to identify predictors for future investigation. This work provides the first population-level estimate of LPS burden in the diving population.

Methods

STUDY DESIGN AND POPULATION

This anonymous, cross-sectional survey study was reviewed and approved by the DAN Institutional Review Board (IRB # 042-25-26), and informed consent was obtained from all participants. An anonymous online questionnaire was distributed to members of the DAN via a public post on DAN's official Facebook page.[11] Participation was voluntary, and no incentives to participate were offered. The survey period spanned from July to October 2025. While DAN has a large international membership base, the number of members who viewed the survey post is unknown, and therefore a response rate could not be determined. Participants were required to be adults (over 18 years of age) and included both technical and recreational divers. Incomplete responses, defined as those missing more than 75% of the total questions, were excluded from the final analysis.

DATA COLLECTION

Demographic data, diving experience, relevant medical history, diving-associated symptoms, and behaviors were collected. Demographic data included height, weight, body mass index (BMI), sex, race, ethnicity, educational level, and employment status. Relevant medical history included prior medical conditions, voice-related medical problems, bariatric/laryngeal surgery, and active medication use. Diving-associated symptoms included regurgitation, laryngospasm, heartburn, vomiting, difficulty inhaling, coughing, globus sensation, postnasal drip, throat clearing, dysphagia, and hoarseness. Interruption of regular diet was defined as avoidance, reduction, or modification of a participant's typical food or meal intake due to LPS. Collected diving-related behaviors included eating, consuming alcohol, soda, energy drinks, coffee, or smoking prior to diving. Diving categories were not mutually exclusive, as participants could hold multiple credentials (e.g., recreational and technical certifications).

SURVEY INSTRUMENTS

The primary outcome was LPS burden measured by the RSI, a validated, nine-item questionnaire originally developed to quantify reflux-related complaints.[10] While the RSI was historically associated with 'laryngopharyngeal reflux', it more accurately captures the presence and severity of LPS rather than diagnosing LPRD, which requires objective evidence linking chronic, troublesome LPS to gastroesophageal reflux. The symptoms covered include hoarseness, throat clearing, excess throat mucus, swallowing difficulties, coughing, choking, globus sensation, and classic GERD symptoms such as heartburn or chest pain. Each item is scored on a Likert scale from 0 (indicating no problem) to 5 (indicating severe problems), with a total possible score of 45 thus higher scores indicate more severe symptoms. The RSI has demonstrated high internal consistency, test-retest reliability, and responsiveness to treatment across several populations.[10,12,13] A score of 13 or higher has been described as abnormal, as it corresponds to the 95th percentile among healthy controls (without LPRD) in the original validation study.[10]

STATISTICAL ANALYSIS

All statistical analyses were performed using IBM SPSS Statistics for Windows, version 28.0 (IBM Corp., Armonk, NY). Continuous variables were reported as means with standard deviations (SD) or as medians with interquartile ranges (IQR), contingent on their distribution. Categorical variables were presented as frequencies and percentages. Normality was assessed using the Shapiro-Wilk test, and equality of variances using Levene's test. The one-sample binomial test was used to compare the observed prevalence of elevated RSI scores (≥ 13) among divers to a previously published non-diving population rate of 10.15%.[14] Between-group comparisons were conducted using chi-square or Fisher's exact tests for categorical variables and Mann Whitney U or Kruskal Wallis tests for non-normally distributed continuous variables. Multivariable Poisson regression with robust standard errors was used to identify independent predictors of RSI ≥ 13. Patients with missing clinicodemographic variables were excluded from the corresponding analyses. A P-value of < 0.05 was considered statistically significant.

Results

COHORT CHARACTERISTICS

A total of 436 responses were recorded. After eliminating 127 incomplete responses, 309 divers were included in the final analysis. The median age of participants was 50.0 years (IQR: 40.0, 60.0) with a roughly equal sex distribution (47.2% male, 45.3% female). In terms of ethnic/racial distribution, most participants self-identified as non-Hispanic (81.6%) white (85.8%). The majority of participants had a high level of education, with 71.2% having completed a four-year college degree or higher. Table 1 summarises the demographic characteristics of the study cohort.

Table 1
Baseline demographic characteristics of the final study cohort (n = 309). IQR – interquartile range; LL – lower limit; SD – standard deviation; UL – upper limit

Characteristics		*n* (%)	Mean (SD)	Median [IQR]	Range: LL–UL
Age (years)		286 (92.6)	49.8 (13.8)	50.0 [40.0–60.0]	18–81
Height (cm)		288 (93.2)	172.8 (9.5)	173.0 [167.5–178.5]	150–204
Weight (kg)		289 (93.5)	81.6 (17.5)	79.7 [69.6–89.8]	43.0–158.8
Body mass index (kg·m^{-2})		288 (93.2)	27.3 (5.2)	26.5 [23.4–29.7]	14.1–48.8
Sex	Male	146 (47.2)	-----	-----	-----
	Female	140 (45.3)	-----	-----	-----
	Prefer not to answer	1 (0.3)	-----	-----	-----
Race	White	265 (85.8)	-----	-----	-----
	Other/prefer not to answer	17 (5.5)	-----	-----	-----
	Asian	4 (1.3)	-----	-----	-----
	American Indian or Alaska native	2 (0.7)	-----	-----	-----
	Native Hawaiian or other Pacific Island	1 (0.3)	-----	-----	-----
	Black or African American	1 (0.3)	-----	-----	-----
Ethnicity	Not Hispanic or Latino	252 (81.6)	-----	-----	-----
	Hispanic or Latino	25 (8.1)	-----	-----	-----
	Unsure/Prefer not to answer	7 (2.3)	-----	-----	-----
Education	More than 4-year college degree	155 (50.2)	-----	-----	-----
	4-year college graduate	65 (21.0)	-----	-----	-----
	Some college or 2-year degree	54 (17.5)	-----	-----	-----
	High school or GED	13 (4.2)	-----	-----	-----
	Some high school, did not graduate	1 (0.3)	-----	-----	-----
Diving	Recreational	217 (70.2)	-----	-----	-----
	Technical	43 (13.9)	-----	-----	-----
	Recreational instructor	90 (29.1)	-----	-----	-----
	Technical instructor	15 (4.9)	-----	-----	-----
	Open Water	87 (28.2)	-----	-----	-----
	Advanced	151 (48.9)	-----	-----	-----
	Freediver	17 (5.5)	-----	-----	-----
	Other	31 (10.0)	-----	-----	-----

A range of scuba-diving backgrounds were represented in our cohort. Most (70.2%) of the participants consisted of recreational divers, followed by advanced divers (48.9%), recreational instructors (29.1%), technical divers (13.9%), and freedivers (5.5%). The majority of participants (82.5%) reported eating prior to diving, with a mean interval of 1.5 (SD 0.7) hours between eating and dive initiation. Alcohol consumption the night before diving was also reported by 50.5% of divers, with an average intake of 1.8 (1.0) standard drinks. A smaller subset (7.4%) reported consuming alcohol immediately before or between dives. Additional pre-/inter-dive behaviors included cigarette use (11.7%), electronic cigarette use (5.5%), marijuana use (1.6%), and consumption of coffee (56.0%), soda (43.7%), and energy drinks (15.9%).

Common medical comorbidities included GERD (26.2%), allergies (24.3%), hypertension (14.9%), and asthma (7.4%). A small subset of respondents reported prior laryngeal or upper airway conditions including chronic cough (6.8%), vocal cord paresis/paralysis (1.6%), uncorrected upper airway obstruction (0.6%), or swallowing disorders (0.3%). Few had undergone prior laryngeal surgery (2.3%). In terms of medication use, 13.6% were actively taking proton pump inhibitors (PPIs), 7.4% were on glucagon-like peptide-1 (GLP-1) agonists, and 4.9% reported histamine-2 (H2) antagonist use. Few participants had undergone formal reflux testing, including pH impedance (4.5%) or wireless capsule testing (1.3%). Table 2 summarises the baseline clinical characteristics of the final study cohort.

Table 2
Baseline clinical characteristics of the final study cohort ($n = 309$). GLP-1 – glucagon-like peptide-1

Characteristics ($n = 309$)	n (%)
Prior medical conditions	
Hypertension	46 (14.9)
Diabetes	16 (5.2)
Allergies	75 (24.3)
Asthma	23 (7.4)
Patent foramen ovale	8 (2.6)
Other lung/heart condition	12 (3.9)
Gastroesophageal reflux disease	81 (26.2)
Hiatal hernia	27 (8.7)
Gastric ulcer	7 (2.3)
Fundoplication	5 (1.6)
Bariatric surgery	9 (2.9)
Other	38 (12.3)
Prior voice-related medical problems	
Inflammatory conditions	5 (1.6)
Vocal cord paresis/paralysis	5 (1.6)
Uncorrected upper airway obstruction	2 (0.6)
Laryngocele	1 (0.3)
Inducible laryngeal obstruction/vocal cord dysfunction	2 (0.6)
Chronic cough	21 (6.8)
Swallowing disorder	1 (0.3)
Other	13 (4.2)
Prior laryngeal/upper airway surgery?	7 (2.3)
Are you actively taking any of the following medications?	
Anticholinergics	1 (0.3)
Calcium channel blockers	7 (2.3)
Progesterone	17 (5.5)
Tricyclic antidepressants	5 (1.6)
GLP-1 agonist	23 (7.4)
Proton pump inhibitor	42 (13.6)
Histamine-2 receptor antagonist	15 (4.9)
Other	34 (11.0)

SYMPTOM BURDEN DURING DIVES

LPS were commonly reported during dives. The most frequently endorsed symptoms included coughing (60.5%), regurgitation (49.5%), and heartburn (49.2%). Less commonly reported symptoms included throat clearing (38.5%), postnasal drip (36.6%), globus sensation (24.6%), hoarseness after surfacing (23.6%), and vomiting (17.8%). Laryngospasm during dives was reported by 23 divers (7.4%). Table 3 provides a summary of the LPS of the final study cohort.

Among divers who reported the number of episodes experienced in the past year, throat clearing occurred most frequently (mean 18.6 episodes per year), followed by globus sensation (mean 16.1 episodes per year), hoarseness (mean 15.2 episodes per year), postnasal drip (mean 15.0 episodes per year), and heartburn (mean 14.5 episodes per year). Laryngospasm (5.8 episodes per year), regurgitation (5.0 episodes per year), and vomiting (5.0 episodes per year) occurred less frequently. Notably, symptom distributions were wide, with several participants reporting up to 200 episodes per year for some items (Table 3).

Symptoms were described as life-threatening by 13 divers (4.2%) and severe enough to interrupt diet in 63 (20.4%). Of 216 respondents, the average depth at which symptoms occurred was 17.8 (13.2) m. Symptoms occurred most commonly underwater (37.2%) or both underwater and at the surface (37.2%).

REFLUX SYMPTOM INDEX (RSI)

Ninety-four percent of participants ($n = 291$) completed the RSI section of the survey. The mean total RSI score was 8.9 (8.3) (range 0–41). Among 291 divers with complete RSI data, 26.1% (95% CI, 21.4–31.5%) met the RSI ≥ 13 threshold. Compared to a non-diving population prevalence of 10.15%,[14] this was significantly higher ($P < 0.001$), representing a +16.0 percentage-point increase (95% CI, +11.3%–21.4%).

Among individual RSI items, the most frequently reported symptoms (score ≥ 2) were related to upper throat irritation: excess throat mucus/postnasal drip (42.9%), heartburn or indigestion (49.5%), and frequent throat clearing (36.1%). Globus sensation (27.5%), coughing (24.8%), and hoarseness (19.9%) were also common. In contrast, more severe symptoms such as difficulty swallowing (15.1%) or breathing/choking episodes (6.8%) were less frequently reported. Mean item scores were highest for heartburn-related complaints (mean 1.8 [SD 1.6]) and excess mucus/postnasal drip (mean 1.6 [1.6]), reflecting the most burdensome symptoms among the cohort. Table 4 provides a summary of the RSI responses for the final study cohort.

BETWEEN-GROUP COMPARISONS AND PREDICTORS OF ABNORMAL RSI

Abnormal RSI scores (≥ 13) were more frequently observed in females (30.9%) compared to males (21.2%; $P = 0.043$), although total RSI scores did not significantly differ by sex (median 7 [IQR 11] vs 6.5 [IQR 8]; $P = 0.225$). Divers with a history of GERD had higher rates of RSI ≥ 13 (38.3%) compared to those without GERD (21.4%; $P = 0.003$), and their total RSI scores were also significantly elevated (median 10 [IQR 9] vs 5 [IQR 9]; $P < 0.001$).

Table 3

Baseline upper aerodigestive symptoms of the final study cohort (*n* = 309); LL – lower limit; SD – standard deviation; UL – upper limit

Characteristics:	*n* (%)	Mean (SD)	Range: LL–UL
Experienced the following symptoms while diving:			
Regurgitation	153 (49.5)	-----	-----
Laryngospasm	23 (7.4)	-----	-----
Heartburn	152 (49.2)	-----	-----
Vomiting	55 (17.8)	-----	-----
Difficulty taking a breath "in"	42 (13.6)	-----	-----
Coughing	187 (60.5)	-----	-----
"*Lump*" in the throat	76 (24.6)	-----	-----
Excess mucus in the throat/postnasal drip	113 (36.6)	-----	-----
Frequent urge to throat-clear	119 (38.5)	-----	-----
Trouble swallowing	45 (14.6)	-----	-----
Hoarseness (after surfacing)	73 (23.6)	-----	-----
How many times have you experienced the following symptoms during the last year?			
Regurgitation	104 (33.7)	5.0 (7.9)	0–50
Laryngospasm	19 (6.1)	5.8 (8.6)	0–30
Heartburn	103 (33.3)	14.5 (26.2)	0–200
Vomiting	42 (13.6)	5.0 (14.8)	0–80
Difficulty taking a breath "*in*"	28 (9.1)	6.6 (11.7)	0–50
Coughing	113 (36.6)	12.0 (22.8)	0–200
"*Lump*" in the throat	67 (21.7)	16.1 (28.7)	0–200
Excess mucus in the throat	43 (13.9)	15.0 (33.0)	0–200
Frequent urge to throat-clear	66 (21.4)	18.6 (29.3)	0–200
Trouble swallowing	25 (8.1)	20.0 (41.3)	0–200
Hoarseness (after surfacing)	42 (13.6)	15.2 (21.2)	0–100
Regular diet was interrupted by symptoms	63 (20.4)	-----	-----
Considered symptoms to be life-threatening	13 (4.2)	-----	-----
Depth when last experienced symptoms (m)?	216 (69.9)	17.8 (13.2)	0–140
Location where symptoms occurred:			
Underwater	115 (37.2)	-----	-----
Surface	40 (12.9)	-----	-----
Both	115 (37.2)	-----	-----
Neither	29 (9.4)	-----	-----
Do you tend to eat prior to diving?			
Yes	255 (82.5)	-----	-----
No	53 (17.2)	-----	-----
How long before diving do you typically eat? (h)	237 (76.7)	1.5 (0.7)	0–4
Do you ever drink alcohol the night before diving?			
Yes	156 (50.5)	-----	-----
No	151 (48.9)	-----	-----
How much alcohol do you typically drink (servings)?	142 (46.0)	1.8 (1.0)	1–5
Have you done any of the following before or between dives?			
Consumed alcohol	23 (7.4)	-----	-----
Smoked cigarettes	36 (11.7)	-----	-----
Smoked marijuana	5 (1.6)	-----	-----
Used an e-cigarette or vaporiser	17 (5.5)	-----	-----
Drink soda	135 (43.7)	-----	-----
Drink energy drinks	49 (15.9)	-----	-----
Drink coffee	173 (56.0)	-----	-----
Have you ever had one of the following reflux tests?			
pH impedance test	14 (4.5)	-----	-----
Wireless capsule test	4 (1.3)	-----	-----

Table 4
Reflux Symptom Index responses for the final study cohort (n = 291); SD – standard deviation

Reflux symptom index item	*n* (%)						Mean (SD)
	0	1	2	3	4	5	
Hoarseness or a problem with your voice	212 (72.9)	18 (6.2)	19 (6.5)	27 (9.3)	8 (2.7)	4 (1.4)	0.7 (1.2)
Clearing your throat	127 (43.6)	58 (19.9)	38 (13.1)	27 (9.3)	28 (9.6)	12 (4.1)	1.3 (1.5)
Excess throat mucus or postnasal drip	114 (39.2)	50 (17.2)	33 (11.3)	42 (14.4)	34 (11.7)	16 (5.5)	1.6 (1.6)
Difficulty swallowing foods, liquids, or pills	214 (73.5)	31 (10.7)	17 (5.8)	16 (5.5)	4 (1.4)	7 (2.4)	0.6 (1.2)
Coughing after you ate or after lying down	169 (58.1)	49 (16.8)	29 (10.0)	20 (6.9)	14 (4.8)	9 (3.1)	0.9 (1.4)
Breathing difficulties or choking episodes	242 (83.2)	26 (8.9)	14 (4.8)	5 (1.7)	1 (0.3)	-----	0.3 (0.7)
Troublesome or annoying cough	178 (61.2)	54 (18.6)	17 (5.8)	17 (5.8)	13 (4.5)	11 (3.8)	0.9 (1.4)
Sensation of something sticking in your throat or a lump in your throat	158 (54.3)	51 (17.5)	32 (11.0)	23 (7.9)	13 (4.5)	12 (4.1)	1.0 (1.4)
Heartburn, chest pain, indigestion, or stomach acid coming up	97 (33.3)	50 (17.2)	47 (16.2)	43 (14.8)	35 (12.0)	19 (6.5)	1.8 (1.6)
Reflux symptom index total score							8.9 (8.3)

Diving-related symptoms showed strong associations with elevated RSI scores. Abnormal RSI scores were significantly more common among divers who experienced laryngospasm (52.4% vs 23.0%; $P = 0.008$), vomiting (41.2% vs 22.3%; $P = 0.005$), coughing (33.3% vs 13.3%; $P < 0.001$), and hoarseness after surfacing (49.3% vs 17.9%; $P < 0.001$), though the latter two associations may be partially influenced by the fact that coughing and hoarseness are included as RSI scoring items. Active PPI use (vs. non-use) was also associated with elevated RSI scores (median 9 [IQR 10] vs 6 [IQR 10]; $P < 0.04$).

In a multivariable Poisson regression model, GERD history (adjusted prevalence ratio [aPR] 1.75, 95% CI 1.16–2.65; $P = 0.008$) and female sex (aPR 1.59, 95% CI 1.07–2.37; $P = 0.022$) emerged as independent predictors of RSI ≥ 13. Recreational divers had a significantly lower likelihood of elevated RSI (aPR 0.59, 95% CI 0.39–0.90; $P = 0.014$), while technical divers showed a non-significant trend toward higher prevalence (aPR 1.54, 95% CI 0.95–2.49; $P = 0.08$). Other diver types, including scuba instructors and freedivers, did not show measurable associations. No significant associations were observed for age, PPI use, asthma, allergy, alcohol use, or eating before diving.

Discussion

In this cross-sectional survey study, over a quarter (26.1%) of divers had abnormal RSI scores. This prevalence is significantly higher than the 10.15% reported in the non-diving population using the same diagnostic tool and cutoff threshold.[14] Additional factors associated with an increased rate of abnormal RSI scores included a history of GERD (aPR 1.75, 95 % CI 1.16–2.65, $P = 0.008$) and female sex (aPR 1.59, 95 % CI 1.07–2.37, $P = 0.022$). On the other hand, recreational diving was associated with a significantly lower prevalence of abnormal RSI scores (aPR 0.59, 95 % CI 0.39–0.90, $P = 0.014$), suggesting that exposure intensity and diving technique may influence the risk of experiencing LPS.

In the absence of a diagnostic gold standard, general population estimates of LPRD prevalence vary depending on the diagnostic tool, cutoff threshold, and population studied. Additionally, this heterogeneity can also be attributed to the nonspecific nature of LPR symptoms, which can be mimicked by other inflammatory and functional conditions.[15] The RSI is commonly used to quantify LPS in the general population because it is validated, reliable, and easy to administer.[10] Existing studies on LPS prevalence have exclusively focused on non-diving populations, ranging from otolaryngology outpatient clinics to general practices. Xiao and colleagues conducted a large-scale multicenter survey study across otolaryngology clinics in China were approximately 10% of the study population had an RSI score > 13.[14] In contrast, Kamani and colleagues employed pseudo-random sampling of 2,000 adults from four general practices in England and found that 34.4% of respondents had LPS. Notably, their study used a lower threshold (RSI > 10) which may overestimate the prevalence of LPS in this particular population.[16]

We also found that LPS were common during dives, with approximately 60% reporting coughing, 50% regurgitation, and 40% frequent throat clearing. Laryngospasm was reported by approximately 8% of participants and almost 5% described their symptoms as life-threatening. While laryngospasm was included as a self-reported symptom in the questionnaire, we acknowledge that the term may have been interpreted variably by respondents and may not necessarily reflect clinically confirmed laryngospasm. These findings align with prior reports of reflux-associated incidents in divers, which, though not specific to LPRD, highlight potential safety concerns of reflux-like symptoms underwater. Petri et al. described a self-contained underwater breathing apparatus (scuba) diving fatality in a previously healthy, properly trained, and fairly experienced diver.[9] Forensic analysis and postmortem findings suggested that an episode of vomiting followed by panic and removal of the breathing regulator resulted in death by drowning. Similarly, Novomesky and colleagues reported two diving-related incidents with vomiting and aspiration of gastric contents, with one of the cases resulting in fatality.[6] Finally, a series of scuba-related fatalities in military personnel revealed that almost 12% (4/34) of divers had evidence of gastric content aspiration, suggesting that acute reflux-related events may be an underdiagnosed cause of mortality in the diving population.[7]

Our findings are concordant with known LPRD risk factors, including female sex and GERD history, likely reflecting hormonal influences and compromised esophageal sphincter function.[12,17] The association between abnormal RSI scores and a history of GERD is consistent with existing literature, which estimates that approximately 50% of patients with LPR have concomitant GERD.[15,18] However, it is important to note that LPR is not merely an extension of GERD; it involves distinct pathophysiological mechanisms, primarily linked to upper esophageal sphincter dysfunction and a significantly higher mucosal sensitivity in the laryngopharynx compared to the esophagus.[1,18] Mechanical factors intrinsic to diving (e.g., compressive wetsuits, and head-down positioning) may facilitate retrograde flow of gastric contents.[5] Additionally, pressure fluctuations during descent and ascent may promote aerophagia, belching, or regurgitation, particularly in divers with pre-existing anatomical or comorbid risk factors.[6] This mechanical facilitation is especially concerning given the unique fragility of the laryngopharyngeal mucosa; while the oesophageal mucosa can resist up to 50 potential episodes of reflux per day without tissue damage, the laryngopharyngeal tissues can be damaged by as few as four episodes.[1,18] This vulnerability suggests that the physiological stressors of diving could exacerbate even minor, sub-clinical reflux into LPS. Finally, the high prevalence of symptoms in the diving population may be explained by the reflex theory of LPR, which posits that the stimulation of oesophageal nerve receptors by gastric contents can trigger vagally mediated laryngeal reflexes, such as chronic coughing and throat clearing.[15,18] In the high-pressure and physically demanding environment of diving, these reflexes may be more easily triggered, leading to the chronic persistent symptoms observed in this cohort. As a result, divers should remain especially cognisant of the modifiable risk factors associated with a potentially increased risk of reflux-related events. Avoiding large, slowly digesting meals at least two hours before a dive, as well as avoiding substances that may reduce esophageal sphincter pressures including ethanol, tobacco, coffee, carbonated beverages and certain medications (e.g., anticholinergics, calcium channel blockers, progesterone, among others) should be encouraged for those concerned among the diving community.[6,9,19]

While this work provides the first quantitative, population-level data on LPS burden in the diving population, it is not free of limitations. The self-reported nature of this study may be subject to recall or reporting bias, especially for symptoms that have non-reflux related causes such as cough, throat clearing, and hoarseness. To standardise symptom assessment, we utilised the RSI, a widely validated patient-reported outcome measure with strong psychometric properties. Because objective reflux testing was not feasible in this cross-sectional survey, the RSI was used to quantify LPS burden; however, it should be acknowledged that it does not establish a clinical diagnosis of GERD or LPRD. Selection bias is another potential limitation of this study, given the low response rate and older age distribution of respondents, which may not fully reflect the broader diving population. However, this demographic profile is consistent with the membership of the DAN and includes a broad range of certification levels, contributing to generalisability. We also acknowledge that the 10.15% prevalence reported by Xiao et al., was drawn from an otolaryngology clinic-based sample and may reflect a population with a higher baseline risk for LPS. Nevertheless, our finding of a 26.1% prevalence (95% CI, 21.4–31.5%) using the same RSI threshold highlights the need for increased awareness in the diving population. Finally, causality cannot be inferred. It remains unclear whether diving exacerbates LPS or whether those with more severe LPS are more aware of their symptoms during dives. Nevertheless, this limitation is inherent to the cross-sectional study design and given the potential for mortality, the association remains noteworthy.

Our findings suggest that divers may have an increased prevalence and severity of LPS, with potential implications for both safety and quality of life. While current guidelines have adequately covered cardiovascular, pulmonary, neurologic, otologic and rhinologic conditions, there is scant literature on the risk of upper aerodigestive conditions during diving.[20–22] Interventional studies or prospective tracking of LPS onset relative to dive exposure may help inform future diving fitness recommendations or practice guidelines. Finally, multidisciplinary collaboration between gastroenterology, otolaryngology, speech language pathology, and dive medicine may be an essential component to optimise health and safety in the constantly growing diving population.

Conclusions

In this international survey of divers, we identified a substantially higher prevalence of abnormal (≥ 13) RSI scores compared to the non-diving population. Female sex and GERD history were associated with an increased rate of abnormal RSI scores, while recreational diving appeared less provocative compared to other scuba diving categories. Increased awareness and preventive strategies may improve both safety and quality of life in the diving population.

References

1 Koufman JA, Aviv JE, Casiano RR, Shaw GY. Laryngopharyngeal reflux: position statement of the committee on speech, voice, and swallowing disorders of the American Academy of Otolaryngology-Head and Neck Surgery. Otolaryngol Head Neck Surg. 2002;127:32–5. doi: 10.1067/mhn.2002.125760. PMID: 12161727.

2 Lechien JR, Vaezi MF, Chan WW, Allen JE, Karkos PD, Saussez S, et al. The Dubai definition and diagnostic criteria of laryngopharyngeal reflux: The IFOS consensus. Laryngoscope. 2024;134:1614–24. doi: 10.1002/lary.31134. PMID: 37929860.

3 Yadlapati R, Weissbrod P, Walsh E, Carroll TL, Chan WW, Gartner-Schmidt J, et al. The San Diego consensus for laryngopharyngeal symptoms and laryngopharyngeal reflux disease. Am J Gastroenterol. 2026;121:322–36. doi: 10.14309/ajg.0000000000003482. PMID: 40197644. PMCID: PMC12353988.

4 Krause AJ, Walsh EH, Weissbrod PA, Taft TH, Yadlapati R. An update on current treatment strategies for laryngopharyngeal reflux symptoms. Ann N Y Acad Sci. 2022;1510(1):5–17. doi: 10.1111/nyas.14728. PMID: 34921412. PMCID: PMC9012673.

5 Patrician A, Dujić Z, Spajić B, Drviš I, Ainslie PN. Breath-hold diving - the physiology of diving deep and returning. Front Physiol. 2021;12:639377. doi: 10.3389/fphys.2021.639377. PMID: 34093221. PMCID: PMC8176094.

6 Novomeský F, Janík M, Hájek M, Krajčovič J, Straka L. Vomiting and aspiration of gastric contents: a possible life-threatening combination in underwater diving. Diving Hyperb Med. 2018;48:36–9. doi: 10.28920/dhm48.1.36-39. PMID: 29557100. PMCID: PMC6467823.

7 Goldhahn RT Jr. Scuba diving deaths: a review and approach for the pathologist. Leg Med Annu. 1977;1976:109–32. PMID: 865216.

8 Novomesky F, Krajcovic J, Stuller F. Vomitus as a cause of fatal diving accident of a commercial diver. Soud Lek. 2010;55:5–7. PMID: 21275228.

9 Petri NM, Stipancevic H, Sutlovic D, Gojanovic MD. Death of a scuba diver caused by vomiting and panic: a case report. Am J Forensic Med Pathol. 2011;32:186–9. doi: 10.1097/PAF.0b013e3181d3de7a. PMID: 20190635.

10 Belafsky PC, Postma GN, Koufman JA. Validity and reliability of the reflux symptom index (RSI). J Voice. 2002;16:274–7. doi: 10.1016/s0892-1997(02)00097-8. PMID: 12150380.

11 Diver's Alert Network (DAN). [cited 2025 Nov 11]. Available from: https://dan.org/.

12 Lechien JR, Huet K, Finck C, Khalife M, Fourneau AF, Delvaux V, et al. Validity and reliability of a French version of reflux symptom index. J Voice. 2017;31(4):512.e1–512.e7. doi: 10.1016/j.jvoice.2016.11.020. PMID: 28069467.

13 Podzimek J. Validity and reliability of a German version of reflux symptom index. J Voice. 2025:S0892-1997(25)00251-6. doi: 10.1016/j.jvoice.2025.06.027. PMID: 40681378.

14 Xiao S, Li J, Zheng H, Yan Y, Li X, Zhang L, et al. An epidemiological survey of laryngopharyngeal reflux disease at the otorhinolaryngology-head and neck surgery clinics in China. Eur Arch Otorhinolaryngol. 2020;277:2829–38. doi: 10.1007/s00405-020-06045-0. PMID: 32449029.

15 Lechien JR, Akst LM, Hamdan AL, Schindler A, Karkos PD, Barillari MR, et al. Evaluation and management of laryngopharyngeal reflux disease: state of the art review. Otolaryngol Head Neck Surg. 2019;160(5):762–82. doi: 10.1177/0194599819827488. PMID: 30744489.

16 Kamani T, Penney S, Mitra I, Pothula V. The prevalence of laryngopharyngeal reflux in the English population. Eur Arch Otorhinolaryngol. 2012;269:2219–25. doi: 10.1007/s00405-012-2028-1. PMID: 22576243.

17 Liu Z, Zhang C, Wang X, Zhang J, Liu L, Wang J, et al. Characteristics of laryngopharyngeal reflux in patients of different genders and ages. J Voice. 2025;39:764–9. doi: 10.1016/j.jvoice.2022.11.035. PMID: 36567235.

18 Cui N, Dai T, Liu Y, Wang YY, Lin JY, Zheng QF, et al. Laryngopharyngeal reflux disease: Updated examination of mechanisms, pathophysiology, treatment, and association with gastroesophageal reflux disease. World J Gastroenterol. 2024;30:2209–19. doi: 10.3748/wjg.v30.i16.2209. PMID: 38690022. PMCID: PMC11056915.

19 Maret-Ouda J, Markar SR, Lagergren J. Gastroesophageal reflux disease: A review. JAMA. 2020;324(24):2536–47. doi: 10.1001/jama.2020.21360. PMID: 33351048.

20 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.

21 Lechner M, Sutton L, Fishman JM, Kaylie DM, Moon RE, Masterson L, et al. Otorhinolaryngology and diving-Part 2: Otorhinolaryngological fitness for compressed gas scuba diving: a review. JAMA Otolaryngol Head Neck Surg. 2018;144:259–63. doi: 10.1001/jamaoto.2017.2616. PMID: 29450499.

22 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.

Acknowledgements

The authors would like to thank the Divers Alert Network and all participants who contributed to the development of this project.

Conflicts of interest and funding: nil

Submitted: 6 March 2026
Accepted after revision: 16 May 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

Technical reports

Forensic analysis of an unwitnessed fatal diving accident using in-water investigative reconstruction

Veronika Rybárová[1], František Novomeský[1], Radek Prygl[2], Jiří Šejba[2]

[1] *Department of Forensic Medicine and Medicolegal Expertise, Jessenius Faculty of Medicine, Comenius University in Bratislava, University Hospital, Martin, Slovak Republic*
[2] *Department of Special Diving Operations and Training, Police of the Czech Republic, Czech Republic*

Corresponding author: *Dr Veronika Rybárová, PhD, Department of Forensic Medicine and Medicolegal Expertise, Jessenius Faculty of Medicine, Comenius University in Bratislava, University Hospital, Kollárova 2, 036 01 Martin, Slovak Republic*
ORCiD: *0000-0003-2619-3333*
veronika.rybarova@uniba.sk

Keywords
Accidents; Autopsy; Diving deaths; Forensic diving medicine; Re-enactment; Simulation; Underwater accident reconstruction

Abstract

(Rybárová V, Novomeský F, Prygl R, Šejba J. Forensic analysis of an unwitnessed fatal diving accident using in-water investigative reconstruction. Diving and Hyperbaric Medicine. 2026 30 September;56(3):317–322. doi: 10.28920/dhm56.3.317-322. PMID: 42743572.)
Unwitnessed diving fatalities present significant challenges for forensic investigation. We describe a fatal diving accident involving a 32-year-old experienced female diver during a deep night dive in freshwater. The diver became separated from her buddy and was recovered at depth in a head-down position, with evidence of entanglement of the central manifold valve of the twin-tank open-circuit breathing apparatus by a thin guideline. Autopsy findings were consistent with drowning. Technical examination of the diving equipment revealed no mechanical malfunction. Given the absence of eyewitnesses and inconclusive assessment of the accident circumstances, an underwater reconstruction was conducted in collaboration with a police diving unit. Using the original breathing apparatus and controlled underwater conditions, the reconstruction evaluated the technical feasibility of various accident scenarios, including entanglement, buoyancy redistribution, and loss of equipment control. The reconstruction demonstrated that unintentional formation of a simple guideline loop around the central manifold valve could occur during attempts at disentanglement. Partial inflation of the buoyancy control device and drysuit, combined with mechanical constraint of the breathing apparatus due to entanglement, resulted in inversion into a head-down position and displacement of the breathing apparatus following release of the waist strap. These configurations substantially impaired the diver's self-rescue capability and were consistent with objective findings documented at recovery. This case underscores the forensic value of controlled underwater reconstruction as a complementary investigative tool in unwitnessed technical diving fatalities and highlights its potential contribution to accident analysis and prevention.

Introduction

Most diving fatalities occur in situations without direct witnesses, leaving investigators with limited information about the diver's final minutes of life. In such cases, the body is typically recovered, examined at the scene, and subsequently subjected to autopsy; however, the immediate circumstances preceding death often remain unverifiable. This uncertainty creates considerable scope for divergent interpretations of the accident sequence and underlying mechanism.

Previous studies have shown that post-mortem findings and equipment inspection alone rarely allow reliable differentiation between incapacitation, entrapment, loss of buoyancy control, or behavioural factors during the terminal phase of a dive.[1–5] Consequently, unwitnessed diving fatalities remain among the most challenging scenarios in aquatic death investigation. Methods capable of integrating scene documentation, equipment analysis, and controlled underwater investigative reconstruction may therefore represent a valuable adjunct for improving the interpretative framework in such cases.[6,7]

Underwater reconstructions have been proposed as a valuable – although still infrequently implemented – approach to reducing interpretative uncertainty. When logistically feasible, such reconstructions allow investigators to reproduce critical mechanical factors, assess equipment behaviour under realistic conditions, and evaluate the plausibility of competing accident hypotheses.

The concept of re-enactment as an investigative tool in diving accidents was first reported in 1967 and was originally

designed for internal expert use within the Royal Australian Navy.[8] Edmonds and Caruso have described the use of controlled re-enactments in the investigation of unexplained diving fatalities, particularly in cases where conventional post-mortem examination and equipment testing failed to provide a convincing explanation for the fatal event. Such re-enactments aim to replicate selected aspects of the fatal dive under controlled conditions in order to identify potential adverse mechanisms, rather than to establish a definitive sequence of events.[9,10]

In contrast, the present report applies a deliberately restricted underwater investigative reconstruction focused on evaluation of technical configurations and physical constraints, without attempting full replication of the fatal dive. The authors encountered a case involving the fatality of an experienced female diver during a deep night dive in which the diver became separated from her buddy. The body was discovered at depth in a pronounced head-down position, entrapped by a thin line, with no direct evidence explaining how the incident unfolded.

Given the uncertainties surrounding the terminal events, forensic specialists, in collaboration with the Police divers of the Czech Republic, initiated an underwater investigative reconstruction of the presumed crisis scenario. The reconstruction aimed to reproduce the mechanical constraints under which the diver became entrapped and to assess the functionality and configuration of her breathing apparatus under comparable conditions, while also exploring feasible self-rescue options. This article presents the case circumstances, recovery findings, autopsy and technical examination results, and the outcomes of the underwater reconstruction, and discusses the forensic value of reconstruction-based analysis in the investigation of unwitnessed technical diving fatalities.

Case report

A 32-year-old female diver (XX) participated in a night dive with four other experienced divers to a depth of approximately 45 metres of freshwater (mfw). The divers began their descent at approximately the same time; one diver conducted the dive solo, while the remaining divers dived in pairs. Upon reaching the bottom at a depth of approximately 45 mfw, diver XX and her buddy initiated a joint ascent toward the surface, following the bottom profile. Due to disturbance of bottom sediment, visibility deteriorated markedly despite the use of torches, leading to separation of the dive pair. After switching off his light, the buddy did not observe any light signal from diver XX and therefore initiated a solo ascent to the surface while adhering to the planned decompression schedule. The remaining divers in the group also surfaced without complications. The police were informed of the disappearance of diver XX approximately two hours later.

According to statements from the other members of the diving group, diver XX did not report any problems prior to the last dive. She appeared relaxed, in a good mood, and was joking during dive preparation. She informed her buddy that she was not carrying her personal diving knife on this dive, as it was reportedly damaged. Diver XX did, however, carry a hook-shaped cutting tool with a sharp blade suitable for cutting thin cords and fishing lines. The tool was stored in the side pocket of the right leg of her drysuit and secured with a lanyard and a bolt-snap. The diver also wore relatively oversized dry gloves, which partially limited manual dexterity. The twin-set open-circuit scuba cylinders (2 × 10 L) were relatively short. In an attempt to lower the centre of gravity, diver XX had made a modification by attaching a standard 50-mm weight belt, screwed to the lower tank band. Two standard lead blocks (3 kg each) were threaded onto this belt, forming a freely hanging auxiliary ballast ‘tail‘ suspended from the twin-set when the diver was in an upright position.

Recovery and scene findings

The body of diver XX was located at a depth of 28 mfw and recovered by police divers. At the scene, the body was maintained in a pronounced head-down position, oriented vertically with the feet upwards, and held in place by a taut 5-mm guideline secured by a simple loop around the central manifold valve between the cylinders, without knot formation (Figure 1).

Both legs and boots of the drysuit were inflated. The breathing apparatus had slipped over the diver’s head; the waist strap was unfastened, and the shoulder straps were

Figure 1
Configuration of the breathing apparatus as recovered, showing a simple loop formed around the central manifold valve between the cylinders, without knot formation



loosened and displaced onto the diver's forearms. The auxiliary ballast 'tail' described above was suspended downward on the external side of the cylinders.

The primary and back-up second-stage regulators were hanging freely alongside the diver's body. The buoyancy control device (BCD) was partially inflated, approximately to half of its volume. The low-pressure hose for drysuit inflation was disconnected from the drysuit inflator valve. The diver's mask contained greenish water mixed with blood and whitish foam.

Autopsy and technical expertise findings

Autopsy findings were consistent with drowning, without any remarkable pathological changes. No traumatic injuries or significant natural disease processes were identified that could have independently explained death.

Technical examination of the diving equipment did not reveal any mechanical malfunction. All components of the breathing apparatus were found to be functional and appropriate for use. The breathing gas used was compressed air; however, at the time of expert examination, the cylinders were empty.

Data from the personal dive computer were extracted and analysed; the recorded dive profile documented a critical phase consistent with a crisis event.

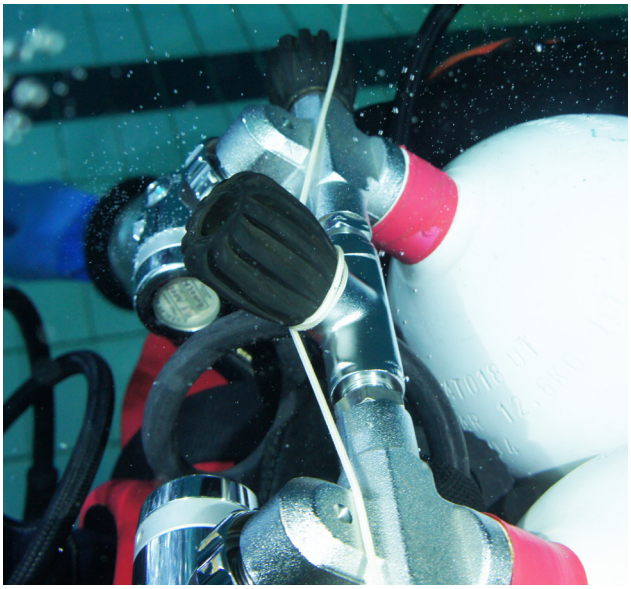
Investigative experiment (reconstruction)

An investigative reconstruction was conducted by divers of the Department of Special Diving Operations and Training of the Police of the Czech Republic in an indoor swimming pool with a maximum depth of 5.5 mfw. The reconstruction was performed in the presence of a forensic expert witness in the investigation of diving fatalities (Slovak Republic); photographic documentation was performed by the expert witness, while video recording was carried out by the police divers.

The objective of the reconstruction was to situationally reconstruct plausible scenarios involving entanglement of a guideline around the central manifold valve of the breathing apparatus and to assess the practical ability of a diver to self-extricate from such entanglement using their own means. Prior to commencement, an identical guideline was positioned by police divers approximately 1 m above the pool bottom.

The test subject was a police diver equipped with a drysuit identical to that used by diver XX. The test diver used the original breathing apparatus of diver XX, including the buoyancy control device (BCD) and the auxiliary ballast tail. Underwater, the test diver was deliberately positioned in predefined configurations in accordance with the reconstruction protocol.

Figure 2
Simple, non-knotted loop of the guideline formed around the central manifold valve of the breathing apparatus during the reconstruction



Subsequent actions, including attempts at self-release from the guideline, were continuously documented using photographic and video recording. The expert witness and police divers guided the test diver underwater into configurations considered most consistent with the probable real-world circumstances of the diving accident.

This investigative reconstruction was designed to evaluate technical feasibility and physical constraints only; no conclusions regarding intent, precise sequence of events, or causality were derived from the experiment alone.

Principal findings of the reconstruction

During the reconstruction, the test diver was able to create a simple, non-knotted loop of the guideline around the central manifold valve of the breathing apparatus using one hand while manipulating the line in an attempt to remove it. This occurred despite the use of oversized dry gloves, which limited manual dexterity (Figure 2).

In an attempt to free from the guideline, partial inflation of the BCD and the drysuit, in combination with firm fixation of the breathing apparatus by the guideline, resulted in rotation into a vertical inverted (head-down) position. Redistribution of gas into the lower parts of the drysuit further contributed to this inversion (Figure 3).

Following disconnection of the drysuit inflator hose and unfastening of the waist strap of the breathing apparatus while already in a vertical inverted position, the breathing apparatus immediately slipped over the diver's head. This resulted in immobilisation and was assessed as a critical situation requiring immediate intervention by the surrounding police divers (Figure 4).

Figure 3
Inversion into a vertical head-down position caused by partial inflation of the buoyancy control device and drysuit, with gas redistribution within the drysuit during fixation of the breathing apparatus by the guideline

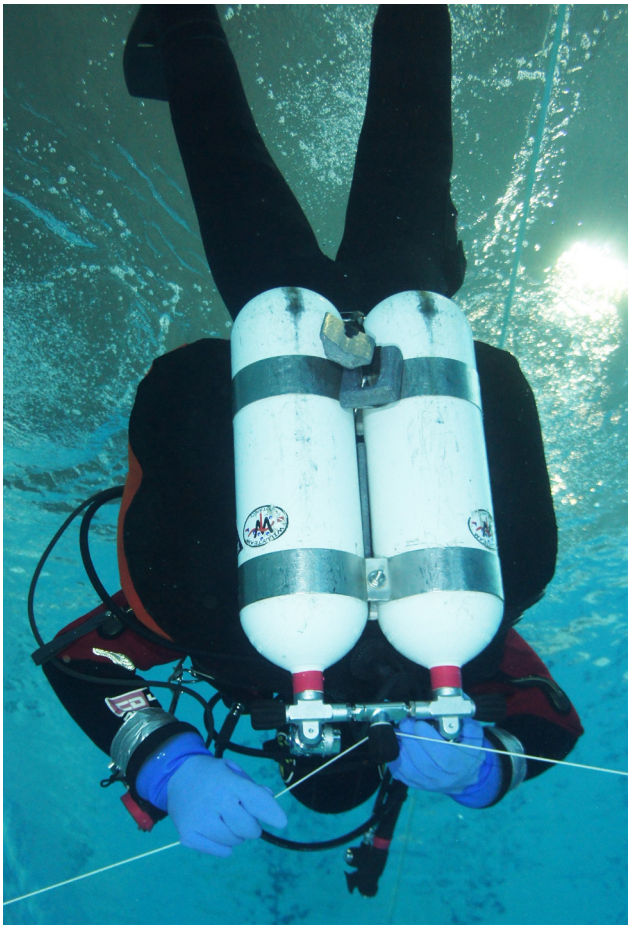


Figure 4
Breathing apparatus displacement over the diver's head in a vertical head-down position following release of the waist strap and disconnection of the drysuit inflator hose



In no tested body position was self-extrication from the simple loop of the guideline around the central manifold valve of the breathing apparatus achievable using manual efforts alone.

Interpretation of the diving accident based on reconstruction

The following interpretation is based on integration of recovery scene findings, autopsy results, technical examination of the diving equipment, and outcomes of the underwater investigative reconstruction. It does not claim to establish the exact sequence of events or the diver's intent.

During a night dive at a depth of approximately 30 mfw in green-brown water with markedly reduced visibility (approximately 0.8 m), diver XX likely experienced accidental entanglement of the central manifold valve of the twin-cylinder breathing apparatus with a thin guideline (diameter 5 mm) secured underwater between fixed structures. Such entanglement would have abruptly arrested the diver's forward movement.

In response, diver XX plausibly attempted to remove the guideline manually. Under conditions of darkness, near-zero visibility, and reliance on tactile perception alone – further compromised by oversized dry gloves – it is technically plausible that a simple, non-knotted loop of the guideline could have been unintentionally formed around the central manifold valve during manipulation of the line. This manoeuvre would have occurred in a confined and difficult-to-access area behind the diver's head, at depth, and under conditions conducive to acute stress and panic.

Following unsuccessful attempts at manual release, partial inflation of the BCD and the drysuit may have been initiated in an attempt to relieve tension or regain mobility. Redistribution of gas into the lower portions of the drysuit plausibly resulted in inversion into a vertical head-down position. In this configuration, the guideline would have been subjected to upward tension towards the surface, forming a configuration resembling an inverted V relative to the diver's body position.

At no stage was the cutting tool carried in the right thigh pocket of the drysuit used. This omission may have been related to difficulty accessing the pocket, interference from oversized dry gloves, or cognitive narrowing associated with panic.

In the final phase of the event, diver XX appears to have attempted to abandon the entire breathing apparatus and initiate an ascent without it. This likely involved disconnection of the drysuit inflator hose followed by unfastening of the waist strap of the breathing apparatus. In the inverted position, the unsecured breathing apparatus – additionally burdened by freely suspended auxiliary ballast – would have immediately slipped over the diver's head, resulting in loss of the regulator from the mouth. This sequence plausibly led to uncontrolled water aspiration and subsequent drowning.

Discussion

This case illustrates the interpretative challenges inherent in unwitnessed diving fatalities. Although sometimes used by experienced police investigators in some jurisdictions, published contemporary case-based reports integrating recovery findings, systematic equipment examination, and controlled underwater reconstruction in unwitnessed diving fatalities remain limited.[8–10] Despite comprehensive recovery documentation, autopsy findings consistent with drowning, absence of detectable mechanical malfunction of the diving equipment, and no evidence of alternative natural or traumatic causes of death, the immediate sequence of events leading to death could not be reliably established on the basis of post-mortem findings alone.

As in many diving incidents, particularly during night dives, the critical phase of the accident occurred underwater, outside direct observation, and under environmental conditions that severely limited situational awareness and self-rescue options. Reduced visibility, depth-related physiological stressors, and task loading likely contributed to rapid escalation of the incident once entanglement occurred. The underwater investigative reconstruction demonstrated that a simple, non-knotted loop of a guideline around the central manifold valve of the breathing apparatus could be formed unintentionally during manual attempts at line removal. When combined with partial inflation of the BCD and drysuit, and with the breathing apparatus remaining fixed by the guideline, this configuration resulted in inversion into a vertical head-down position. Under these conditions, disconnection of the drysuit inflator hose and unfastening of the waist strap led to immediate displacement of the breathing apparatus over the diver's head, resulting in loss of effective mobility.

The configurations observed during reconstruction were consistent with objective recovery findings, including the inverted body position, inflation of the lower portions of the drysuit, partial BCD inflation, displacement of the breathing apparatus, and disconnection of the drysuit inflator hose. Although reconstruction cannot determine intent, precise timing, or the exact sequence of actions, it provides important insight into the technical plausibility of the observed configuration and the rapid deterioration of self-rescue capability. Underwater investigative reconstructions must be conducted exclusively by appropriately trained and experienced divers under strict safety protocols. While such reconstructions cannot replicate the physiological or psychological state of a diver during a real emergency, they offer a valuable adjunct to scene documentation, equipment examination, dive profile analysis, and witness statements.

Conclusions

This case demonstrates the limitations of post-mortem findings and equipment inspection alone in the investigation of unwitnessed diving fatalities. Integration of recovery scene documentation with controlled underwater investigative reconstruction allowed evaluation of the technical plausibility of alternative accident scenarios and provided a coherent explanatory framework consistent with objective findings.

Although reconstruction cannot establish the exact sequence of events or the diver's intent, it represents a valuable complementary approach for reducing interpretative uncertainty in complex diving incidents. When applied cautiously and under strict safety conditions, underwater reconstruction may enhance forensic analysis of diving fatalities and contribute to improved understanding of accident mechanisms and preventive considerations.

References

1 Caruso JL. The Investigation of diving-related deaths. In: Payne-James J, Byard RW, editors. Encyclopedia of forensic and legal medicine. 3rd ed. Vol. 4. UK: Elsevier; 2025. p. 623–34. doi: 10.1016/B978-0-443-21441-7.00273-9.

2 Casadesús JM, Aguirre F, Carrera A, Boadas-Vaello P, Serrando MT, Reina F. Diving-related fatalities: multidisciplinary, experience-based investigation. Forensic Sci Med Pathol. 2019;15:224–32. doi: 10.1007/s12024-019-00109-2. PMID: 30915609.

3 Edmonds C. Investigation of diving fatalities. In: Edmonds C, Bennett M, Lippmann J, Mitchell SJ. Diving and subaquatic medicine. 5th ed. Boca Raton (FL): Taylor & Francis; 2016. p. 583–600.

4 Lippmann J, Caruso JL. The investigation of diving accidents and fatalities. Diving Hyperb Med. 2024;54:217–24. doi: 10.28920/dhm54.3.217-224. PMID: 39288927. PMCID: PMC11659060.

5 Tarozzi I, Franceschetti L, Simonini G, Raddi S, Machado D, Bugelli V. Black box of diving accidents: Contribution of forensic underwater experts to three fatal cases. Forensic Sci Int. 2023;346:111642. doi: 10.1016/j.forsciint.2023.111642. PMID: 36963130.

6 Becker RF. Underwater forensic investigation. 2nd ed. Boca Raton: CRC Press; 2013.

7 Teather RG. Encyclopaedia of underwater investigations. 2nd ed. Flagstaff AZ: Best Publishing; 2013.
8 Edmonds C. Reappraisals of a diving disaster. Royal Australian Navy School of Underwater Medicine Report 4/68. Sydney: RAN Submarine and Underwater Medicine Unit; 1968.
9 Edmonds C, Caruso JL. Recent modifications to the investigation of diving related deaths. Forensic Sci Med Pathol. 2014;10:83–90. doi: 10.1007/s12024-013-9491-x. PMID: 24166195.
10 Edmonds C, Caruso JL. Diving fatality investigations: recent changes. Diving Hyperb Med. 2014;44:91–6. PMID: 24986727. [cited 2026 May 15]. Available from: https://dhmjournal.com/images/IndividArticles/44June/Edmonds_dhm.44.2.91-96.pdf.

Acknowledgements

The authors would like to thank the divers of the Department of Special Diving Operations and Training of the Police of the Czech Republic for their professional cooperation, technical expertise, and assistance with the underwater investigative reconstruction.

Conflicts of interest and funding: nil

Submitted: 29 January 2026
Accepted: 16 May 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

Evaluation of external ventricular drain performance under hyperbaric conditions: a bench study

Rahulkumar Ramchandani[1], Zhiliang C Lin[1,2], Bridget Devaney[1,2], Rosalind L Jeffree[3,4], Theo Tsouras[1]

[1] *Department of Intensive Care and Hyperbaric Medicine, The Alfred Care Group, Bayside Health, Melbourne, Australia*
[2] *School of Public Health and Preventative Medicine, Monash University, Melbourne, Australia*
[3] *Neurosurgery Department, The Alfred Care Group, Bayside Health, Melbourne, Australia*
[4] *Department of Surgery, Monash University, Clayton, Victoria, Australia*

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

Keywords
EVD; Hyperbaric chambers; Intensive care; Intracranial pressure; Neurosurgery

Abstract
(Ramchandani R, Lin ZC, Devaney B, Jeffree RL, Tsouras T. Evaluation of external ventricular drain performance under hyperbaric conditions: a bench study. Diving and Hyperbaric Medicine. 2026 30 September;56(3):323–328. doi: 10.28920/dhm56.3.323-328. PMID: 42743573.)
Introduction: External ventricular drains (EVDs) are widely used for intracranial pressure (ICP) monitoring and cerebrospinal fluid (CSF) diversion in neurocritical care. Hyperbaric oxygen treatment (HBOT) exposes medical devices to increased ambient pressure; however, limited data exists regarding EVD performance and reliability under hyperbaric conditions.
Methods: A bench study was conducted to evaluate EVD performance under hyperbaric conditions. A rigid, non-compressible saline-filled column was used to assess pressure measurement stability independent of compliance effects. Additional compliance-based syringe models were explored to assess the feasibility of evaluating unclamped drainage behaviour. A commercially available EVD system (CODMAN EDS 3; Codman & Shurtleff, a Johnson & Johnson company, Raynham, MA, USA) was exposed to stepwise ambient pressures from 101 to 283 kPa (1.0–2.8 atmospheres absolute) in a multiplace hyperbaric chamber. Pressure measurements, drainage behaviour, fluid column stability, and system integrity were assessed.
Results: The CODMAN EDS 3 EVD system maintained structural integrity across tested pressure levels. While pressure measurements were stable in the rigid open glass syringe model, the closed compliance-based models demonstrated pressure variability during chamber pressurisation, including when closed to fluid drainage. Unclamped testing using these models demonstrated inconsistent fluid dynamics, precluding reliable assessment of fluid drainage behaviour under hyperbaric conditions.
Conclusions: The CODMAN EDS 3 EVD system remained structurally intact during hyperbaric exposure; however, pressure measurements varied in closed models, including when closed to fluid drainage. ICP measurements obtained during HBOT should be interpreted with caution due to potential pressure-related artefacts. Reliable continuous assessment of fluid pressure drainage behaviour remains limited by current bench model constraints. Further research incorporating advanced brain compliance simulation models and carefully designed in-vivo observational studies is required to better characterise EVD behaviour under hyperbaric conditions and to ensure safe clinical use and accurate interpretation of ICP measurements in the hyperbaric environment.

Introduction

External ventricular drains (EVDs) are integral to neurocritical care, allowing monitoring of intracranial pressure (ICP) and drainage of cerebrospinal fluid (CSF) in patients with acute neurological injury. Accurate ICP measurement guides therapeutic interventions and reliable drainage to follow as both under- and over-drainage may contribute to secondary brain injury and adverse outcomes.[1–3]

Hyperbaric oxygen treatment (HBOT) is increasingly utilised in critically ill patients, including those with neurological injury who may require concurrent ICP monitoring.[4–5] Previous clinical and experimental studies have demonstrated that HBOT can influence ICP and cerebral haemodynamics, with measurements obtained via ventricular or spinal drainage catheters showing variable responses during pressurisation and oxygen exposure.[6–9] In addition to physiological effects, increased ambient pressure during HBOT may influence pressure transduction, hydrostatic reference systems, and fluid behaviour within

EVD drainage systems. Despite these considerations, limited experimental data exists examining the behaviour, reliability, and safety of EVD systems in the hyperbaric environment. Understanding device performance under these conditions is important to ensure accurate interpretation of ICP measurements and predictable drainage to inform safe clinical practice.

This study aimed to evaluate the performance and reliability of a standard EVD system utilising the CODMAN EDS3 under controlled hyperbaric conditions using a bench model, with specific attention to pressure measurement stability, drainage behaviour where feasible, and examine potential operational limitations relevant to HBOT.

Although this study evaluated a commercially available external ventricular drain (EVD) system, other cerebrospinal fluid (CSF) drainage systems, including lumbar drains, may also be encountered in patients undergoing hyperbaric oxygen therapy. Lumbar CSF drainage has an established role in spinal cord protection and has been proposed as an adjunctive therapy in selected cases of spinal cord infarction and severe neurological decompression illness.[10,11] However, lumbar drainage systems differ from EVDs in tubing configuration, pressure relationships, compliance and their dependence on patient position. Consequently, the present study was limited to evaluation of a commercially available EVD system, and separate investigation of lumbar drainage systems under hyperbaric conditions is warranted.

Methods

This was a laboratory-based experimental bench study conducted in a multiplace hyperbaric chamber. All experiments were performed using normal saline at room temperature.

EXPERIMENTAL MODELS

Rigid open model

An open glass syringe connected to a rigid, saline-filled column was used to generate reproducible hydrostatic pressure and assess transducer behaviour independent of compliance effects (Figure 1).

Compliance-based models

To explore the feasibility of assessing drainage behaviour in closed systems, additional syringe-based models were evaluated:

- Glass syringe with plunger in situ (Figure 2)
- Plastic syringe sealed with an airtight cork and bung (Figure 3)
- Plastic syringe incorporating a one-way valve (Figure 4)

These models differed in internal volume, compliance, and sealing characteristics and were intended to approximate intracranial compliance under hyperbaric conditions.

EVD SYSTEMS

A commercially available EVD system (CODMAN EDS 3; Codman & Shurtleff, a Johnson & Johnson company, Raynham, MA, USA) was used throughout testing (Figure 5). The pressure transducer was zeroed at atmospheric pressure (101 kPa) according to manufacturer recommendations.

Figure 1
Open glass syringe model with attached to the EVD system



Figure 2
Compliance based model with glass syringe with plunger attached to EVD system



Figure 3
Closed compliance-based model with plastic syringe sealed with an airtight cork and bung attached to EVD



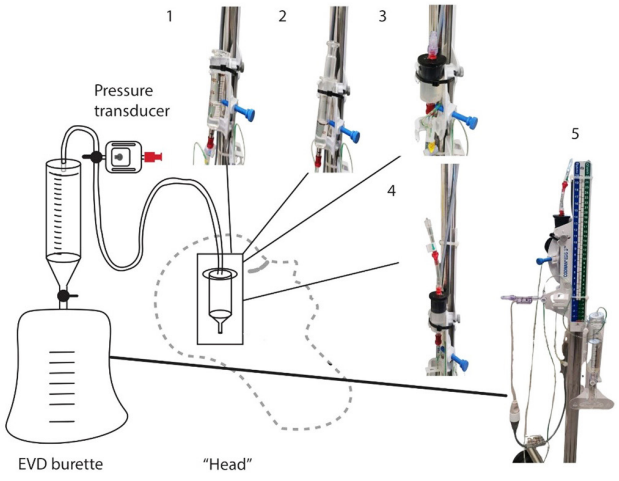
Figure 4
Closed compliance-based model with plastic syringe incorporating a one-way valve attached to the EVD system



Figure 5
EVD system mounted on an intravenous pole with the hydrostatic reference scale and saline-filled column



Figure 6
Overview of the experimental setup showing a schematic diagram alongside photographic images of the different bench models connected to the external ventricular drain system mounted on an intravenous pole for hyperbaric testing



MEASUREMENT SYSTEMS

Intracranial pressure measurements were displayed and recorded using a Philips IntelliVue patient monitoring system (Philips Healthcare, Amsterdam, The Netherlands). The EVD pressure transducer was connected to the monitor via a standard invasive pressure monitoring interface module.

The monitoring system provided continuous digital ICP values in mmHg. Measurements were recorded after stabilisation at each ambient pressure level during hyperbaric exposure.

No recalibration of the monitor or transducer was performed during pressurisation or depressurisation.

HYPERBARIC EXPOSURE PROTOCOL

The assembled systems were placed within a multiplace hyperbaric chamber, and ambient pressure was increased at the rate of 60 kPa·min^{-1} from 101.3 to 283.6 kPa (1.0–2.8 atmospheres absolute [atm abs]). Measurements were obtained after stabilisation at each pressure level. All data were collected during investigator-attended hyperbaric chamber sessions, with the investigator entering the chamber

Table 1
Pressure measurements, drainage behaviour, and mechanical integrity of external ventricular drain systems in different bench model configurations

Parameter		Open glass syringe	Glass syringe with plunger *in situ*	Plastic syringe with an airtight cork and bung	Plastic syringe with a one-way valve
Transducer-derived ICP measurements (mmHg)	101.3 kPa (1 atm abs)	10 mmHg	10 mmHg	35 mmHg	25 mmHg
	283.6 kPa (2.8 atm abs)	10 (Run1) 10 (Run 2)	-15 (Run 1) -13 (Run 2)	-20 (Run 1) -23 (Run 2)	22 (Run 1) 21 (Run 2)
Fluid column behaviour and stability at 283.6 kPa (2.8 atm abs)		Stable with no oscillation	Stable with no oscillation	Stable with no oscillation	Stable with no oscillation
Drainage volume and drainage rate where feasible (drops·min^{-1})	101.3 kPa (1 atm abs)	124	80	0	144
	283.6 kPa (2.8 atm abs)	132 (Run 1) 128 (Run 2)	98 (Run 1) 104 (Run 2)	0 (Run 1) 0 (Run 2)	192 (Run 1) 188 (Run 2)
Structural integrity at 283.6 kPa (2.8 atm abs)		No loss of structural integrity	No loss of structural integrity	No loss of structural integrity	No loss of structural integrity

to directly supervise system setup, pressurisation, and data collection. For each model configuration, measurements at 283.6 kPa (2.8 atm abs) were obtained during two separate investigator-attended hyperbaric chamber exposures to assess repeatability of pressure measurements and drainage behaviour.

DATA COLLECTION

The following parameters were recorded:
- Transducer-derived ICP measurements
- Fluid column behaviour and stability
- Drainage rate where feasible
- Mechanical integrity and pressure-related artefacts

DATA ANALYSIS

Results were analysed descriptively. Pressure measurements obtained at elevated ambient pressures were compared with normobaric baseline values. Trends and variability were assessed using graphical methods.

Results

Figure 6 provides an overview of the experimental setup and the bench model configurations used in this study. The results of testing are displayed in Table 1.

STRUCTURAL INTEGRITY AND FIRE SAFETY

The materials used in the construction of the Codman 3 EDS system were evaluated by our biomedical engineer and determined to be compliant with the applicable requirements of National Fire Protection Association (NFPA 99). Across all experimental runs, the EVD system remained structurally intact during hyperbaric exposure. No catheter failures, leakage, or structural compromise was observed at any pressure and compression rates of up to 60 kPa·min^{-1}.

PRESSURE MEASUREMENT BEHAVIOUR

In the open rigid model, transducer-derived ICP measurements remained unchanged between normobaric conditions and 283.6 kPa (2.8 atm abs) including during pressurisation and depressurisation. In contrast, closed compliance-based models demonstrated substantial pressure variability during pressurisation and depressurisation, including negative pressure readings (-15 to -20 mmHg) and discordant values despite identical starting conditions.

FLUID COLUMN BEHAVIOUR

Despite marked variability in pressure measurements, fluid columns in all models appeared visually stable at 283 kPa (2.8 atm abs), with no overt oscillation or collapse observed.

FLUID DRAINAGE BEHAVIOUR

Drainage behaviour differed between model configurations. At 283.6 kPa (2.8 atm abs), increased drainage rates were observed in the open glass syringe model, the glass syringe with plunger, and the one-way valve model. In contrast, the plastic syringe sealed with an airtight cork and bung demonstrated no drainage at either normobaric or hyperbaric conditions. Changes in drainage rate were inconsistent across models and did not demonstrate a reliable relationship with observed ICP measurements.

Discussion

This bench study demonstrated that the CODMAN EDS 3 EVD maintained structural integrity and was deemed safe to use from a fire risk perspective under hyperbaric conditions.

Previous clinical and experimental studies have shown that ICP can change during HBOT, with reports of temporary reductions in ICP and changes in cerebral blood flow when measurements are taken using ventricular or spinal catheters.[6–9] Rather than examining these physiological effects, this study focused on how EVD systems with continuous drainage capabilities behave in hyperbaric conditions. Our findings suggest that ICP readings obtained during HBOT can be influenced by how pressure is transmitted through the system and how the hydrostatic reference is affected, even when there is no true change in intracranial dynamics.

Assessing unclamped drainage behaviour proved difficult. Although the fluid columns appeared stable at higher ambient pressures, the compliance-based models produced pressure readings that were inconsistent and, in some cases, non-physiological. This highlights the challenge of trying to replicate brain compliance using simple bench models, particularly in a hyperbaric environment, and limits how confidently drainage behaviour can be interpreted using these approaches.

Whilst this study focused on an EVD system, other external CSF drainage systems, including lumbar drains, may also be encountered in patients undergoing hyperbaric oxygen therapy. Lumbar drainage systems differ from EVDs in tubing configuration, pressure relationships, compliance and their dependence on patient position. Consequently, the findings of the present study should not be directly extrapolated to lumbar drainage systems. Nevertheless, the increasing use of lumbar CSF drainage for spinal cord protection and its proposed role in selected cases of spinal cord infarction and severe neurological decompression illness highlight the importance of evaluating these systems independently under hyperbaric conditions.

CLINICAL IMPLICATIONS

From a practical perspective, these findings demonstrate that the CODMAN EDS 3 EVD system maintained structural integrity during hyperbaric exposure when clamped. Clinicians should be aware that ICP values obtained during hyperbaric exposure may be influenced by pressure-related artefacts and should therefore be interpreted cautiously, particularly when transducer zeroing has been performed at 101.3 kPa (1 atm abs).

LIMITATIONS

This study was limited by the absence of a validated compliance model capable of reliably simulating intracranial dynamics under hyperbaric conditions. Only one EVD system type was evaluated, and findings may not be generalisable to all devices or clinical scenarios.

FUTURE RESEARCH

Future work incorporating advanced compliant models or carefully designed clinical observational studies may further clarify EVD drainage behaviour during HBOT.

Further research utilising brain model simulation (advanced compliance models) and in-vivo observations is needed to establish the safety of EVD drainage and ICP interpretation in the hyperbaric environment.

Conclusions

The CODMAN EDS 3 EVD system demonstrated mechanical integrity during hyperbaric exposure; however, pressure measurement behaviour varied substantially between models and pressure-related artefacts were observed in closed compliant systems. Although fluid columns frequently appeared stable, measured pressures did not consistently reflect expected physiological behaviour. Accordingly, ICP measurements obtained during HBOT should be interpreted with caution. Based on these findings, external CSF drainage systems should be clamped during hyperbaric oxygen treatment to minimise the risk of inadvertent over-drainage or under-drainage resulting from pressure-related measurement and drainage artefacts. Following completion of treatment, drainage may be recommenced according to standard neurosurgical protocols. In circumstances where interruption of drainage is undesirable, a drainage strategy guided by physiological CSF production rates may represent a pragmatic alternative, although this approach has not been evaluated in the present study.

References

1 Miller JD, Dearden NM, Piper IR, Chan KH. Control of intracranial pressure in patients with severe head injury. J Neurotrauma. 1992;9 Suppl 1:S317–26. PMID: 1588623.

2 Smith M. Monitoring intracranial pressure in traumatic brain injury. Anesth Analg. 2008;106:240–8. doi: 10.1213/01.ane.0000297296.52006.8e. PMID: 18165584.

3 Le Roux P, Menon DK, Citerio G, Vespa P, Bader MK, Brophy GM, et al. Consensus summary statement of the International Multidisciplinary Consensus Conference on Multimodality Monitoring in Neurocritical Care: a statement for healthcare professionals from the Neurocritical Care Society and the European Society of Intensive Care Medicine. Neurocrit Care. 2014;21(Suppl 2):S1–S26. doi: 10.1007/s12028-014-0041-5. PMID: 25208678. PMCID: PMC10596301.

4 Kindwall EP, Whelan HT, editors. Hyperbaric Medicine Practice. 3rd ed. Flagstaff, AZ: Best Publishing; 2008.

5 Weaver LK. Hyperbaric oxygen in the critically ill. Crit Care Med. 2011;39(7):1784–91. doi: 10.1097/CCM.0b013e31821858d1. PMID: 21460713.

6 Miller JD, Ledingham IM, Jennett WB. Effects of hyperbaric oxygen on intracranial pressure and cerebral blood flow in

experimental cerebral oedema. J Neurol Neurosurg Psychiatry. 1970;33:745–55. doi: 10.1136/jnnp.33.6.745. PMID: 5497875. PMCID: PMC493587.

7 Miller JD, Ledingham IMcA. Reduction of increased intracranial pressure: comparison between hyperbaric oxygen and hyperventilation. Arch Neurol. 1971;24:210–6. doi: 10.1001/archneur.1971.00480330038003. PMID: 5544630.

8 Kohshi K, Yokota A, Konda N, Kinoshita Y, Kajiwara H. Intracranial pressure responses during hyperbaric oxygen therapy. Neurol Med Chir (Tokyo). 1991;31:575–81. doi: 10.2176/nmc.31.575. PMID: 1723171.

9 Rockswold SB, Rockswold GL, Vargo JM, Erickson CA, Sutton RL, Bergman TA, Biros MH. Effects of hyperbaric oxygenation therapy on cerebral metabolism and intracranial pressure in severely brain injured patients. J Neurosurg. 2001;94:403–11. doi: 10.3171/jns.2001.94.3.0403. PMID: 11235943.

10 Mathew B, Laden G. Management of severe spinal cord injury following hyperbaric exposure. Diving Hyperb Med. 2015;45:210. PMID: 26415075.

11 Ashton C, Banham N, Needham M. Acute spontaneous spinal cord infarction: utilisation of hyperbaric oxygen treatment, cerebrospinal fluid drainage and pentoxifylline. Diving Hyperb Med. 2020;50:325–31. doi: 10.28920/dhm50.4.325-331. PMID: 33325011. PMCID: PMC8026234.

Acknowledgements

The authors would like to acknowledge the Alfred Hyperbaric Service nurses – Damianos Tzavellas and Loren Beaber, as well as biomedical engineering staff for facilitating chamber access and technical assistance. We would also to like to acknowledge the testing previously performed at the Royal Hobart Hospital and presented at HTNA 2024.

Conflicts of interest and funding: nil

Submitted: 11 July 2026
Accepted after revision: 25 July 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

Review articles

Diving-related ear, nose, and throat disorders: a structured review using a phase-of-dive framework

Anna Onderkova[1]

[1] *Royal Brompton Hospital, Guy's and St Thomas' NHS Foundation Trust, London, United Kingdom*

Corresponding author: *Dr Anna Onderkova, Royal Brompton Hospital, Guy's and St Thomas' NHS Foundation Trust, Sydney St, London SW3 6NP, United Kingdom*
ORCiD: *0000-0002-4383-3040*
annaonderkova@doctors.org.uk

Keywords
Barotrauma; Decompression illness; Inner ear; Operations – diving; Profile; Valsalva manoeuvre; Vertigo

Abstract
(Onderkova A. Diving-related ear, nose, and throat disorders: a structured review using a phase-of-dive framework. Diving and Hyperbaric Medicine. 2026 30 September;56(3):329–341. doi: 10.28920/dhm56.3.329-341. PMID: 42743574.)
Introduction: The aim of this review was to synthesise evidence on ear, nose, and throat (ENT) and related head and neck complications in scuba divers, with emphasis on mechanisms of injury, dive phase-specific risk patterns, and implications for clinical management and fitness-to-dive decision-making.
Methods: A structured literature search of PubMed, Embase (Ovid), and the Cochrane Library identified systematic reviews, clinical trials, cohort studies, and case series reporting clinically relevant ENT outcomes in scuba divers. Animal studies, freediving studies, and papers focused solely on non-ENT complications were excluded. Forty analytic studies met inclusion criteria, including large cross-sectional surveys and multiple case series of inner ear decompression sickness. Study characteristics and levels of evidence were summarised, and findings were integrated into a phase-of-dive framework (descent, bottom time, ascent).
Results: Across included studies, middle-ear barotrauma was the most prevalent diving-related ENT injury, particularly during descent. Inner ear barotrauma and inner ear decompression sickness were less common but carried a substantially higher risk of persistent auditory or vestibular deficit. Additional reported conditions included sinus barotrauma, otitis externa and external auditory canal exostoses, temporomandibular joint symptoms, odontobarotrauma, and marine-exposure injuries. Evidence concerning postoperative states, following operations such as stapedectomy, tympanoplasty, mastoidectomy, and cochlear implantation, was limited, heterogeneous, and often contradictory, with few data to guide return-to-dive clearance.
Conclusion: Scuba diving-related ENT complications appear to be diverse, influenced by dive phase, and shaped by individual anatomical and physiological factors. A phase-aware framework offers clinicians a useful way to contextualise symptom onset and guide prevention and counselling, particularly for divers with prior ear or nasal surgery or implanted devices. Prospective studies are needed to refine screening practices and establish evidence-based return-to-dive recommendations.

Introduction

Scuba diving is a globally popular recreational and professional activity, with an estimated six million active divers; millions more certified but not diving regularly;[1] and (as estimated in 2017) more than 100,000 new divers qualifying annually.[2] Diving exposes participants to unique environmental stressors, particularly pressure differentials across air-containing anatomical spaces.[2,3] As a result, the head and neck region accounts for over 80% of all diving-related injuries, with up to 65% involving the ear alone.[2,4,5] Diving-related pathology includes a wide range of ear, nose and throat (ENT) and adjacent complications, ranging from otic barotrauma, alternobaric vertigo, sinus barotrauma,[3,4,6] and inner ear decompression illness.[2,7] Even simple otitis externa is highly prevalent among divers and can compromise diving fitness,[2,8] as even seemingly minor complications can significantly impair a diver's situational awareness and may indirectly increase the risk of other serious dive-related injuries.[8,9]

Despite this burden, awareness of available clinical guidance remains limited. A UK survey of 790 scuba divers found that 79% reported ear disorders since learning to dive and 40% met validated criteria suggesting Eustachian tube dysfunction, yet only 27% were aware of published ear health guidance for divers.[10] This gap between the prevalence of diving-related ENT complaints and diver awareness of evidence-based preventive strategies underscores the clinical relevance of a comprehensive, accessible overview of this topic.

A growing number of reviews have addressed ENT pathologies in isolation or in subsets. These include

studies focused on otologic injury,[2] rhinologic and oromaxillofacial complications,[6] and differentiation of inner ear decompression illness from barotrauma.[3] However, they are often narrowly scoped, reflecting anatomical or physiological silos rather than a comprehensive clinical picture. Therefore, this review seeks to deliver a clinically oriented overview of ENT disorders organised by dive phase. This structure mirrors how divers commonly report the onset of their symptoms, thereby improving clinical relevance and practical applicability.

Methods

A structured literature search was conducted in PubMed, Embase (Ovid), and the Cochrane Library, using MeSH headings and free-text keywords related to scuba diving, with terms covering otologic, rhinologic, sinus, pharyngeal, laryngeal, and orofacial conditions relevant to diving-related injury. Forty studies met all inclusion criteria and were included in this structured review. The database search was supplemented by hand-searching and the documented search string is presented in Figure 1. Additional mechanistic and physiological references are cited in the text where relevant to contextualise findings but were not included in the evidence synthesis. Study methodological quality was assessed based on study design, sample size, potential for bias, and reporting completeness; these assessments are summarised in *Supplementary Table 1. No validated risk-of-bias instrument (e.g., ROBINS-I, GRADE) was formally applied given the heterogeneity of study designs.

AI-assisted writing tools were used for language refinement only. Literature identification, selection, interpretation, and synthesis, as well as figure illustration and data representation, were performed by the author.

Findings are organised by dive phase (descent, bottom time, ascent) to highlight mechanisms of injury, contextualise associated risk factors and outcomes, and offer practical insights for prevention and management.

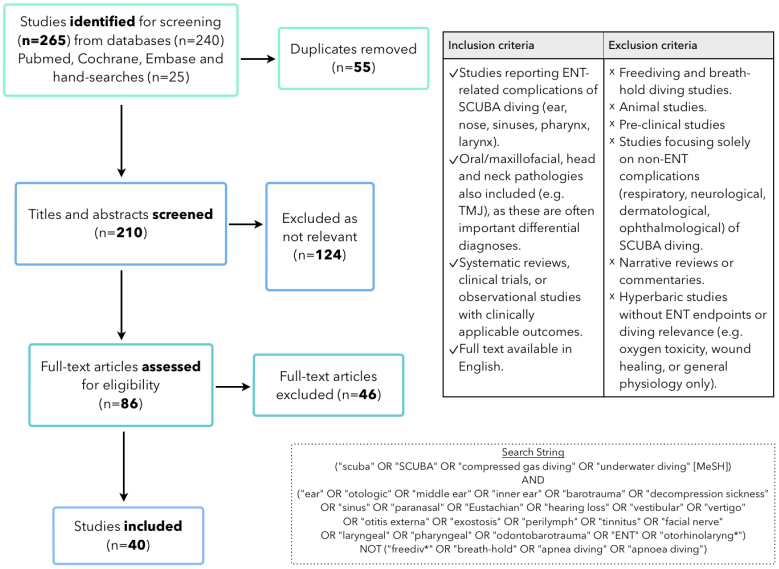
Results

EVIDENCE BASE AND STUDY CHARACTERISTICS

The available evidence on diving-related ENT disorders is unevenly distributed across condition types: barotrauma (particularly middle ear barotrauma) is extensively studied, while inner ear decompression sickness, fitness-to-dive after otological surgery, and long-term effects of repetitive diving on the head and neck are comparatively under-researched. Much of the published literature comprises small observational cohorts and case reports (see *Supplementary Table 1) and focuses on barotrauma, reflecting both the rarity of some of the other presentations and the logistical difficulty of studying divers prospectively. This heterogeneity highlights the need for larger, standardised prospective studies that can clarify the natural history and optimal management of inner ear decompression sickness; establishing evidence-based return-to-dive guidance following inner ear barotrauma, perilymphatic fistula repair,

***Note:** Supplementary Table 1 is available online on our website: https://www.dhmjournal.com/index.php/journals?id=437

Figure 1
Literature search and screening process



and otological implant surgery; and characterising the long-term audiological effects of cumulative diving exposure with appropriate control groups.

MECHANISMS OF INJURY

Several pathophysiological processes contribute to the spectrum of ENT complications encountered in divers. These are summarised in Figure 2.

Barotrauma and Boyle's law

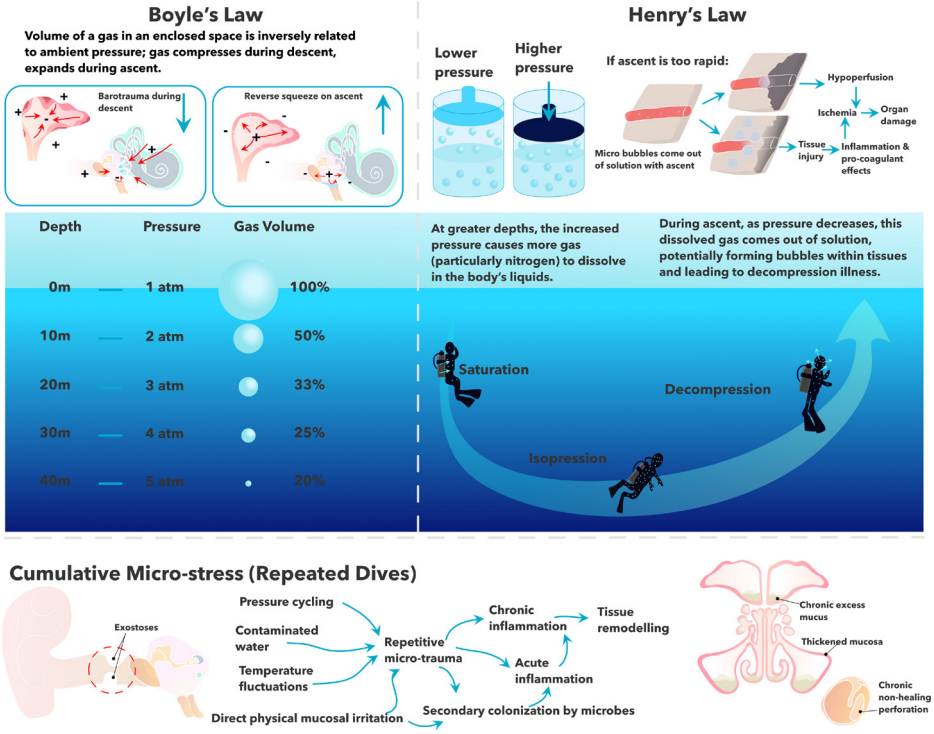
Barotrauma arises due to pressure differentials across anatomical compartments, including both gas-filled and fluid-filled spaces, during changes in ambient pressure. According to Boyle's law, the volume of gas within a closed cavity is inversely proportional to ambient pressure, leading to gas compression during descent and expansion during ascent. Failing to equalise these pressure changes adequately can result in significant mechanical stress: negative pressure on descent leads to mucosal oedema, haemorrhage, and tissue deformation, while positive pressure on ascent may cause outward distention and rupture of affected tissues.[6] The severity and nature of barotrauma depend primarily on the compliance and ventilation of affected anatomical compartments and the degree and duration of pressure imbalance. At the tissue level, these pressure changes can lead to compromised vascular integrity, ischaemia, inflammatory responses, and direct mechanical injury.[2,7]

Decompression and Henry's law

Decompression sickness (DCS) presents with a wide spectrum of manifestations, ranging from mild skin or musculoskeletal symptoms to serious neurological involvement. It can also specifically affect the inner ear (IEDCS). During descent, increased ambient pressure results in a higher inspired partial pressure of inert gases, typically nitrogen, which dissolve into tissues proportional to depth, bottom time, individual tissue perfusion and diffusion characteristics (Henry's Law).[7] Upon ascent, the ambient pressure reduction allows these inert gases to come out of solution and be eliminated via alveolar gas exchange. If the ascent occurs too rapidly, exceeding the body's ability to adequately eliminate dissolved gas, inert gas can form bubbles within the venous circulation and tissues.[2] The vestibular portion of the ear is vulnerable due relatively slow gas washout and thus heightened susceptibility to gas supersaturation and consequent bubble formation or amplification of any small bubbles arriving in the arterial circulation.[11] Inert gas bubbles may directly interact with endothelial cells in the labyrinthine blood vessels, initiating an inflammatory cascade, coagulation activation, and subsequent hypoxic tissue damage; or alternatively, direct mechanical damage caused by microbubble formation within the endolymphatic and perilymphatic spaces may injure local microvessels and membranous structures.[5,12] Additionally, arterialised venous bubbles traveling through a right-to-left cardiac shunt, such as a patent foramen ovale may embolise the labyrinthine artery, contributing to inner ear ischaemia.[13]

Figure 2

An illustrated overview of diving-associated injury mechanisms; atm − atmospheres



Physiological modelling has further quantified this selective inner ear vulnerability: nitrogen washout half-times have been estimated at 8.8 minutes for the inner ear versus 1.2 minutes for the brain following a simulated air dive, implying that the inner ear remains supersaturated with inert gas for substantially longer after surfacing.[14] This creates a post-surfacing temporal window during which venous gas emboli traversing a right-to-left shunt may preferentially grow within inner ear tissue due to inward diffusion of supersaturated gas, whereas equivalent bubbles entering the brain microcirculation would not grow and would redistribute to the venous side. This provides a mechanistic explanation for both the selective vulnerability of the inner ear and the characteristic latency of IEDCS symptoms. The differential gas kinetics between the vestibular organ and the cochlea within the inner ear itself may further explain why vestibular presentations predominate in IEDCS: the cochlea has greater blood flow and smaller tissue volume, implying faster inert gas washout and a shorter window of susceptibility than the vestibular apparatus.[15]

Cumulative exposure and inflammatory remodeling

Repetitive diving exposure, particularly in cold water, can induce low-grade, chronic tissue stress that culminates in structural changes over time. Persistent moisture, temperature fluctuations, and pressure variations can predispose to otitis externa and, over time, to the formation of external auditory canal exostoses.[16] Similarly, the tympanic membrane may thicken, atrophy, or develop non-healing perforations in seasoned divers, which manifests as hearing loss. Long-term auditory effects due to diving *per se* are controversial: although often implied. For example, in a 12-year prospective cohort of professional divers, high-frequency hearing thresholds deteriorated significantly at 4 and 8 kHz, with greater loss in divers performing more than ~650 lifetime dives, suggesting a cumulative effect.[9] Chronic ET dysfunction, even in the absence of acute symptoms, can contribute to these sequelae over time.[17]

Similar inflammatory changes occur within the nasal and paranasal sinus mucosa. Acute sinusitis can arise following isolated episodes of sinus barotrauma or when ostial obstruction from transient mucosal edema traps pathogens, resulting in infection and inflammation. Chronic sinusitis (common among experienced divers, even in the absence of overt symptoms), the result of repeated mechanical stress, particularly when combined with environmental factors such as cold water or allergens, may gradually impair mucociliary clearance mechanisms and promote inflammatory remodeling and fibrosis of the mucosa.[6]

INJURIES BY PHASE OF DIVE

The physiological demands of scuba diving shift throughout every dive; understanding the phase-specific distribution of injuries aids in both diagnosis and targeted prevention strategies. However, it should be noted that many of the pathologies described below can appear in multiple phases of a dive, given varying dive profiles. Important elements of the following narrative are depicted in Figure 3.

Descent phase injuries

The descent is often the most pressure-intensive phase of the dive and is thus characterised by various forms of barotrauma. As ambient pressure increases rapidly, air-filled cavities must be actively equalised to prevent negative pressure effects. Middle ear barotrauma (MEBt), as described earlier, is the most prevalent complication during this phase, consistent with cohort data demonstrating that ~11% of divers present with MEBt and 24% report Eustachian tube (ET) dysfunction.[8] These figures, derived from a tertiary referral cohort, likely underestimate the true population burden. In the largest survey to date, 81% of 1,881 Finnish recreational divers reported having experienced MEBt at some point while diving, with 38% of those affected having used medications and 1% requiring ENT surgery.[18] The strongest independent risk factor was poor subjective success with the Valsalva manoeuvre, followed by poor Toynbee success, highlighting equalisation technique as a primary modifiable target. A UK survey of 790 divers similarly found that 40% met validated criteria for ET dysfunction and 35% had aborted a dive due to ear problems.[10]

ET dysfunction is caused by inadequate ET opening, resulting (during descent) in tympanic membrane retraction, haemorrhage, or rupture (see Figure 4 for grading of MEBt).[2,19] The risk is highest in novice divers or those diving with upper respiratory tract infections or allergic rhinitis, but it can and does affect divers of all experience levels.[2,20] In a condensed four-day open-water certification course, 48 of 67 novice candidates (72%) developed MEBt at some point during training, with language of instruction showing no significant association with incidence, suggesting that comprehension of equalisation instructions alone is not the primary barrier.[21] Repetitive diving compounds this risk: in a prospective study of 28 recreational divers performing 437 dives over six consecutive days, MEBt prevalence reached 36.5% by the final examination and increased with the number of dives per day, while greater experience was protective.[22] Tympanometric data from the same cohort demonstrated a significant positive shift in middle ear pressure from day one to day three and a progressive decrease in compliance across the diving week, most pronounced in less experienced divers, thus indicating that the physiological burden of repetitive diving accumulates measurably even within a single trip. A consideration particularly relevant for liveaboard divers or those undertaking three to four dives daily.[23]

To prevent MEBt, divers should descend slowly when possible and be trained in multiple gentle but effective equalisation techniques, such as the Toynbee or Frenzel maneuvers, rather than simply forcing Valsalva manoeuvres, which can increase the risk of inner ear damage as well (see Figure 5).[2,3] Furthermore, pre-dive screening for Eustachian

Figure 3

Phase-of-dive distribution of ENT and related disorders in scuba divers. Disorders are mapped to the phases of descent, bottom, and ascent, illustrating when specific injuries most commonly occur

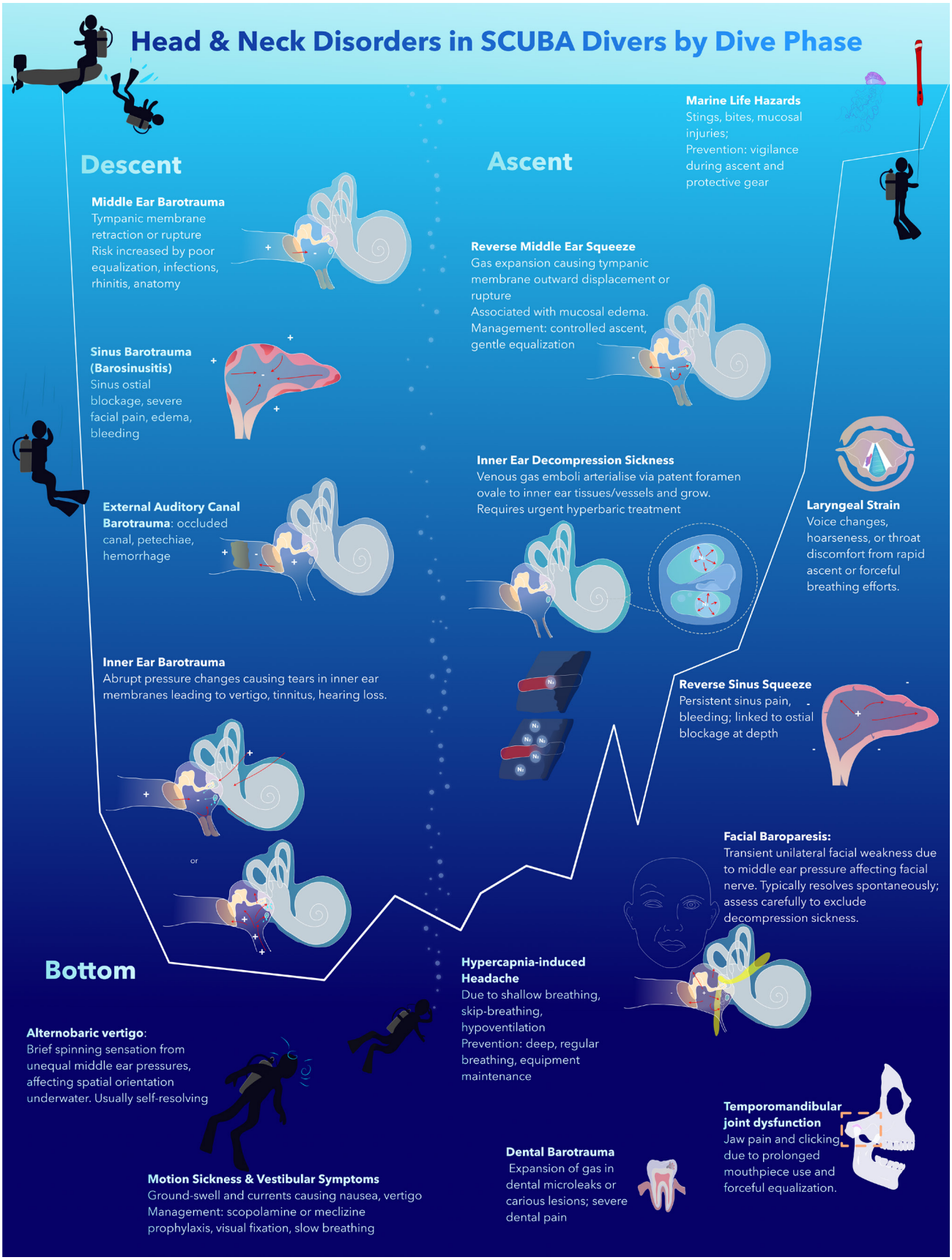
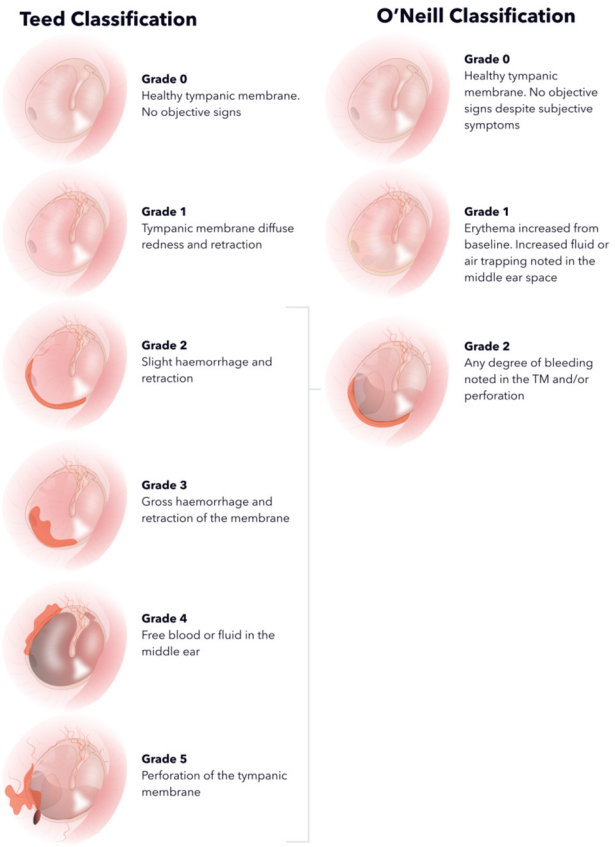


Figure 4
Clinical assessment of the tympanic membrane using Teed (grades 0–5) and O'Neill (grades 0–2) classifications. Management recommendations by grade include Teed 0–2 / O'Neill 1: nasal/oral decongestants, analgesia, and autoinflation techniques; advise delay of further diving until symptoms clear. Teed 3–4 / O'Neill 2: consider a short course of oral steroids, antibiotics if rupture or contamination present; avoid diving and obtain ENT follow-up. Teed 5 (tympanic membrane perforation): as above, plus possible tympanoplasty; heal time typically 4–12 weeks before resumption of diving



dysfunction and prophylactic nasal decongestants or antihistamines are advised for divers with known rhinitis, although evidence for benefit is limited.[2,20,24] Objective pre-dive screening may complement subjective self-assessment: a nine-step tympanometric test battery combined with mastoid pneumatisation assessment achieved a positive predictive value of 86% for identifying military divers who subsequently developed symptomatic MEBt across 774 dives, suggesting a potential role for structured ET function testing before return to diving after injury.[25] However, immediate management of suspected barotrauma includes aborting the dive at the onset of significant pain or vertigo and seeking ENT evaluation if symptoms persist post-dive.[2]

Inner ear barotrauma (IEBt) can arise through abrupt pressure differentials between middle and inner ear spaces being transmitted through the oval or more commonly, round window, potentially resulting in tears of intra-labyrinthine structures such as Reissner's membrane, or rarely, rupture of the round window itself and thus resulting in a perilymphatic fistula. Clinical presentation typically involves sudden vertigo, acute tinnitus, hearing loss, or imbalance, usually noted during descent; however, it can occasionally occur during ascent as well. Cochlear symptoms (tinnitus and hearing loss) are significantly more common than vestibular symptoms in IEBt, a pattern that helps distinguish it from IEDCS, where vestibular presentations tend to predominate.[3,5] Notably, divers attempting forceful Valsalva maneuvers, especially in the setting of delayed middle ear equalisation may inadvertently increase the risk of IEBt by creating an acute and intense pressure differential across labyrinthine membranes.[12] Perilymphatic fistula can also result from forceful equalisation on descent, and symptomatic audio-vestibular loss may appear hours after surfacing, thus making it difficult to identify precisely when injury occurred. Classic features are acute vertigo 'blocked-water' aural fullness, sensorineural hearing loss, and a nystagmus that beats away from the affected ear.

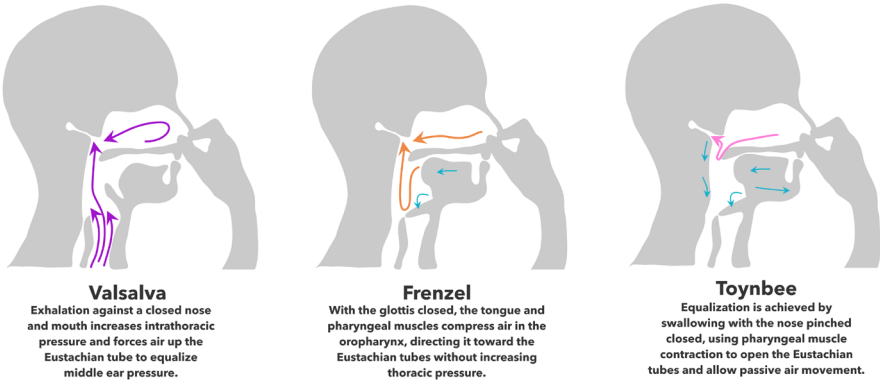
Management involves immediate cessation of diving, head elevation, stool softeners, and high-dose oral corticosteroids (prednisolone 1 $mg \cdot kg^{-1}$ for five to seven days before tapering). Persisting or progressive symptoms beyond 48 h may warrant exploratory tympanotomy with fascia patching. Outcomes have not been intensively studied, but a 26-diver series showed residual deficits in 78%, suggesting IEBt can frequently result in permanent cochlear injury.[13] Longer-term surgical outcome data are available from a series of 11 perilymphatic fistula cases following ear barotrauma, of which eight occurred after scuba diving. Of those explored surgically via an endaural approach, a cochlear fenestral fistula was visualised in seven with active perilymph leakage in six. After temporal fascia grafting, dizziness resolved promptly and early stable subtotal hearing recovery was achieved in seven patients at a median follow-up of 7.4 years, with tinnitus improving in four of six affected cases.[26] These findings support early surgical exploration when perilymph fistula is suspected and symptoms progress beyond 48 hours. Differentiating IEBt from IEDCS in the acute setting remains clinically challenging given overlapping cochleovestibular presentations. A systematic review of 302 manuscripts reported tinnitus in 73% and symptomatic vertigo in 46% of IEBt patients.[27]

Prevention hinges on coaching divers in gentle equalisation and excluding return to diving until audiovestibular function and Eustachian-tube testing are completely normal.[2,28] Long-term follow-up suggests that, with restored ET function and gentle equalisation, recurrent IEBt is uncommon in returning divers,[29] unless they have subtle anatomical vulnerabilities such as widened cochlear aqueducts or internal acoustic canals, underscoring the role of individual predisposition in injury risk.[30]

Less frequently, external ear canal barotrauma can develop when an unvented air pocket between cerumen, exostoses, or a tight hood and the tympanic membrane creates a

Figure 5

Common middle-ear equalisation techniques used in scuba diving. The Valsalva maneuver is the most widely practiced but carries a higher risk of inner-ear barotrauma if performed too forcefully. The Frenzel and Toynbee maneuvers offer gentler alternatives, using controlled muscle contractions and swallowing actions to safely ventilate the middle ear without significantly elevating intrathoracic or intracranial pressure. Other techniques also exist and divers should ideally master multiple techniques to ensure reliable and safe equalisation across different diving scenarios



suction force, producing canal petechiae, serous exudate, or frank haemorrhage. Acute treatment involves removing predisposing blockages (cerumen clearance, canalplasty for exostoses); prophylactic drops may also be recommended. Preventive emphasis is on slow, head-up descents in susceptible divers.[2,7,28]

Barosinusitis (sinus squeeze) similarly arises during descent when sinus ostia fail to ventilate adequately. The resulting vacuum effect within the sinus may cause mucosal oedema, capillary rupture, or pain localised to the frontal or maxillary regions.[6,31] Pre-existing anatomical obstructions, such as septal deviation or nasal polyposis, may further predispose divers to sinus barotrauma.[8,31] Survey data quantify the population burden: in the same Finnish cohort of 1,881 recreational divers, 49% reported having experienced sinus barotrauma while diving, with 32% of those affected having used medications. Independent risk factors included pollen allergy and regular smoking (both modifiable), as well as frequent (three or more per year) upper respiratory tract infections.[32] These factors complement the anatomical predispositions already described and offer practical targets for pre-dive counselling. To minimise the risk, divers should abstain from diving during episodes of illness or significant nasal congestion. Management strategies should emphasise preventive measures, such as gentle equalisation techniques, optimal pre-dive nasal hygiene, and judicious use of prophylactic topical nasal corticosteroids or short-acting decongestants (e.g., oxymetazoline) to manage mucosal oedema and inflammation.[6] Divers with recurrent symptoms should undergo thorough ENT evaluation to assess for underlying pathology or anatomical factors that may necessitate surgical intervention.[6,33]

Rarely, divers who attempt forceful equalisation techniques during descent may place excessive mechanical strain on the larynx or pharyngeal soft tissues. This can result in minor mucosal injuries, localised edema, or even vocal fold haemorrhage, presenting as throat discomfort, odynophagia, transient voice changes, or mild stridor during or shortly after the dive.[5,34] Divers experiencing these symptoms should use gentler, controlled equalisation techniques or seek formal ENT evaluation if symptoms persist post-dive.

Any injuries thus sustained during descent can cause pain and discomfort throughout the whole dive and the indirect effects of this, not just on coloring the experience overall, but also on situational awareness and thus safety by extension, should not be minimised.

Bottom phase challenges and injuries

Once at depth, ambient pressure stabilises, and active equalisation demands generally decrease. Nonetheless, divers may still experience sustained mechanical stress, particularly from equipment or posture.[4,6,12] Some divers report jaw discomfort during this phase, potentially linked to the application of pressure from ill-fitting mouthpieces and stress on the temporomandibular joint (TMJ) but this can also be referred pain from equalisation techniques, although this remains under-characterised.[12] Pressure-related microtrauma during descent and equalisation efforts may exacerbate pre-existing TMJ disorders. Though underreported, such complaints are not uncommon among seasoned divers and may influence diving tolerance and equipment selection (need for custom-fit mouthpieces or physical therapy to reduce strain).[20]

Divers may be prone to headaches during the bottom phase related to hypercapnia, often secondary to hypoventilation, shallow breathing, or ‘skip-breathing’ techniques.[35] Increased partial pressure of carbon dioxide results in cerebral vasodilation, causing headaches, which can significantly impair situational awareness and diver safety. To mitigate this risk, divers should be advised to practice regular and deep tidal breathing, avoid excessive exertion

underwater, and ensure their equipment is properly maintained to facilitate efficient breathing.[35]

On most dives, divers are safe from motion sickness once underwater. However, ground swell at depth can generate semicircular canal stimulation out of phase with visual cues, triggering nausea and retching into the regulator. Persistence of seasickness initiated from the surface can also last well into the dive while the vestibular system attempts to adapt. Currents can exacerbate this. Pre-exposure prophylaxis includes transdermal scopolamine 1.5 mg or meclizine 50 mg orally one hour before diving, alongside alcohol avoidance and hydration. Underwater, divers should fixate on a stable reference and adopt slow tidal breathing to mitigate hypercapnia-driven nausea. If vomiting occurs, the general advice is to assume vertical trim, maintain regulator purge, and end the dive.[28]

Extended bottom times, particularly in cold water or enriched oxygen environments, can negatively impact mucociliary clearance, promote nasal mucosal oedema, and impair ET ventilatory function. These conditions may predispose divers to middle ear dysfunction, delayed middle ear barotrauma, or subtle ET injury due to hyperoxia-induced inflammation. Although free radical scavenger vitamins (C and E) have been studied, no clear protective effect has yet been demonstrated.[12,36] As Meyer et al. point out, we have no standardised procedure for measuring successful equalisation during a dive. However, the cumulative physiological burden of repetitive diving is postulated to extend beyond the immediate descent phase and may compound susceptibility to barotrauma throughout multi-day itineraries.[23]

Similarly, at depth, sinus barotrauma may become symptomatic or worsen if initial equalisation was incomplete, or if mucosal inflammation occurs while submerged. Persistent frontal or maxillary pain, often associated with epistaxis, may develop as a result of sustained pressure differentials within the paranasal sinuses.[6,31] Divers frequently experiencing this should refer to the preventive strategies and management described previously.

Although less common at stable depth, prolonged bottom times involving continuous use of breathing apparatus against increased inspiratory resistance or poorly maintained regulator equipment may occasionally strain laryngeal musculature, resulting in mild dysphonia or throat discomfort.[5] Such symptoms typically resolve spontaneously upon surfacing but can indicate the need for equipment evaluation or breathing technique adjustment.

Ascent phase injuries

During ascent, the reduction in ambient pressure causes expansion of gas trapped within anatomical compartments, increasing the risk of barotrauma from positive-pressure phenomena.

Reverse middle-ear block occurs when expanding air cannot adequately escape through the ET, causing outward displacement or even rupture of the tympanic membrane, intense otalgia, and conductive hearing loss. Though common during rapid or poorly controlled ascents, experienced divers can still encounter this phenomenon if mucosal oedema or inflammation develop during the dive, obstructing ET patency.[12,19] Immediate management includes halting ascent, attempting gentle equalisation, or slightly descending until symptoms alleviate, followed by slow and controlled ascent. Post-dive evaluation is advised if symptoms persist.[2]

Paranasal sinuses may similarly retain trapped air that expands upon ascent, leading to acute sinus pain ('reverse sinus squeeze') as mucosal surfaces distend, sometimes accompanied by mucosal haemorrhage.[6,31] This is more likely if mucus plugs or mucosal swelling or congestion developed at depth, restricting sinus ostial ventilation.[6] Divers experiencing persistent sinus discomfort on ascent should be managed with topical or systemic decongestants and corticosteroids and evaluated for underlying anatomical issues that could be resolved surgically for the future.[6,31]

Barosinusitis of ascent is variable in its presentation, and sometimes the only manifestation is bleeding from the same side as the sinus disorder. A differential diagnosis in these cases is anterior epistaxis, experienced by an estimated 3% of divers due to positive-pressure expansion across the congested Kiesselbach's plexus, producing brief unilateral nasal bleeding during ascent.[7] Divers experiencing recurrent post-dive epistaxis should undergo ENT review. A small number of cases of sinus barotrauma can present rather dramatically, with nausea and vomiting, a sensation of impending syncope, and disorientation.[6]

Another critical concern specific to ascent is IEDCS. IEDCS arises from inert gas bubble formation within labyrinthine vessels or tissues due to supersaturation during ascent, or from right-to-left arterialisation of venous inert gas bubbles which subsequently reach the labyrinthine artery. Symptoms commonly include acute onset vertigo, nystagmus, hearing loss, and tinnitus, typically occurring within the first hour after surfacing.[3,5,15] The largest published IEDCS case series, comprising 115 recreational scuba divers over a 13-year period, found that IEDCS represented 24% of all treated decompression sickness cases.[37] Median symptom onset was 20 minutes after surfacing, with pure vestibular disorders predominating (76.5%), cochlear deficit in 6%, and combined presentations in 17.5%. A large right-to-left shunt was detected in 77% of patients, with a striking right-sided lateralisation of injury (80%, $P < 0.001$), and incomplete recovery was observed in 68% of followed patients. A 30-year retrospective analysis of 437 DCS cases treated in Malta further demonstrated that vestibulo-cochlear decompression illness has been increasing in prevalence while musculoskeletal presentations have declined,

suggesting that evolving diving practices may be shifting the pattern of injury.[38]

A key differentiator between IEBt and IEDCS is the timing: symptoms presenting primarily during descent with associated difficulty equalising strongly suggest IEBt, whereas symptoms emerging during or shortly after ascent more likely indicate IEDCS. But the distinction may be tricky even with a good clinical history, dive profile analysis, and audiovestibular assessment, as both may present with similar clinical findings, and the two may even coexist.[39] Unlike barotrauma, however, IEDCS often occurs without significant middle ear findings and may have a delayed onset after surfacing.[3] Systematic review evidence has quantified several differentiating variables: mean depth at the causative dive was 13 metres for IEBt versus 43 metres for IEDCS, with additional discriminating factors including dive gas type, symptom onset timing relative to depth change, symptom distribution (vestibular versus cochlear predominance), and presence of associated DCS features.[3] Notably, a history of difficult middle ear equalisation could not be reliably assessed as a differentiator because it was insufficiently reported in the IEDCS literature — a significant evidence gap. Structured diagnostic aids such as the HOOYAH tool may assist clinicians in navigating this differential when remote from specialist audiovestibular assessment.[27]

Prompt recognition is critical, as IEDCS typically requires hyperbaric oxygen therapy and carries a risk of permanent auditory or vestibular dysfunction,[7] whereas IEBt is primarily managed conservatively. Outcomes data from case series underscore the severity: in a retrospective series of 23 IEDCS patients treated at a single Australian hyperbaric unit between 2014 and 2020, 88% had vestibular involvement and 38% cochlear involvement, yet median maximum depth was only 24.5 metres and median dive time 40 minutes, demonstrating that IEDCS can occur on dives unremarkable by recreational standards.[40] Median time from surfacing to hyperbaric treatment was 22 hours, and complete symptom recovery was achieved in only 58%, underscoring the importance of early recognition and rapid referral. Among 126 recreational trimix divers, 7.1% reported at least one episode of IEDCS, of whom all reported vestibular symptoms, five of nine tested had a patent foramen ovale, and three reported persistent balance problems long after the event.[41] IEBt has traditionally been managed conservatively, with recompression considered relatively contraindicated.[16] However, a recent case series of five consecutive divers with sensorineural hearing loss attributed to IEBt reported substantial audiometric improvement following hyperbaric oxygen therapy administered 24 hours to 12 days after diving, with a median improvement of 28 dB across all frequencies and 38 dB for PTA4.[42] While this represents a preliminary series requiring prospective confirmation, it suggests that the role of HBOT in IEBt-associated sensorineural hearing loss may warrant further investigation rather than blanket avoidance.

The functional consequences of IEDCS frequently persist beyond the acute phase. In a case series of 13 divers with acute IEDCS who received customised vestibular rehabilitation, vestibular and balance measures improved by discharge and at three-month follow-up; however, 67% still demonstrated objective vestibular deficits, including a positive head impulse test or nystagmus in the dark on videonystagmography, despite subjective symptom resolution.[43] This finding has important implications for return-to-dive counselling: symptom resolution alone should not be used as the sole criterion for fitness-to-dive clearance following IEDCS, and objective vestibular assessment coupled with vestibular rehabilitation should be incorporated into post-treatment evaluation.[13,43]

Transient and typically benign alternobaric vertigo may also occur from asymmetrical middle-ear venting on ascent that creates a transient labyrinthine stimulation mismatch. Usually self-limiting, it presents as a brief spinning sensation without hearing impairment, lasting only seconds to minutes (< 90 s).[5,12,44] ET dysfunction appears highly prevalent in divers with alternobaric vertigo, and allergic rhinitis was present in most affected divers in one study.[44] Severe episodes risk disorientation and uncontrolled ascent and even mild spells may still compromise a diver's spatial orientation temporarily. Furthermore, divers should be taught the 'ascend-pause-Toynbee' manoeuvre (nasal pinch + swallow) rather than forceful Valsalva and must abort the dive if vertigo persists beyond one minute. To minimise risk, underlying rhinitis or ET dysfunction should be treated before diving and tight hoods or occlusive earplugs should be discouraged.[2,7,45]

A rare complication is facial baroparesis in which rapid expansion of middle ear gas compresses the facial nerve within its canal, particularly in the presence of anatomical canal dehiscence, resulting in transient unilateral facial weakness.[46] It is also possible for fifth nerve neuropraxia to produce facial numbness in maxillary sinus barotrauma, albeit in freediving in the most recently reported case.[47] These typically resolve spontaneously, but accurate differentiation from DCS is essential to ensure appropriate and timely hyperbaric therapy when indicated.[46] Balloon dilation of the ET prevented further episodes and enabled a full return to diving, suggesting a potential therapeutic role in selected cases.[48] No prospective series exists specifically in scuba divers; a series of freedivers treated with ET balloon dilation for refractory equalisation failure reported improvement in 15 of 20 patients (10 with complete resolution), suggesting potential applicability of this technique, though direct extrapolation to scuba divers requires caution.[49] Further prospective studies in scuba divers are needed.

Although ascent-phase complications are predominantly otological or sinugenic, maxillofacial symptoms may occasionally present diagnostic challenges. Maxillary pain, for example, could reflect dental or sinus barotrauma. Odontobarotrauma may be triggered during descent if pockets

of trapped air beneath dental restorations are compressed, manifesting as acute pain or crown displacement;[6] however, odontocrexis (restoration fracture) is much more common with ascent, as rapid ambient-pressure shifts can propagate micro-bubbles beneath composite or amalgam restorations, leading to crown fracture or cusp blow-out. Incidence in frequent divers approaches 5%. Thus, routine dental reviews, sealing defective restorations, and completing endodontic work pre-dive, are recommended. Sharp fragments in situ require aborting the dive, waxing the defect, and urgent dental follow-up.[6]

Rare reports of laryngeal barotrauma have been associated specifically with rapid ascent or forceful respiratory efforts. Sudden pressure changes or excessive strain against respiratory resistance may cause transient vocal fold immobility, mucosal swelling, or soft tissue edema, manifesting as immediate post-dive hoarseness, throat tightness, or mild dyspnea.[5,33] Divers experiencing these symptoms post-ascent should seek timely ENT evaluation, given potential airway implications and the need to distinguish from more serious pulmonary barotrauma (beyond the scope of ENT manifestations but relevant to clinical safety assessments). Divers with persistent cough, chest discomfort, or neurological symptoms should receive immediate medical evaluation for DCS or arterial gas embolism, as these may accompany ascent-phase ENT symptoms.[16]

During the final metres of ascent and at the surface interval, divers are particularly exposed to risks from marine life, although such incidents can occur at any point during the dive. Facial stings from jellyfish and other cnidarians may cause severe pain, peri-lesional oedema, and oropharyngeal dysesthesia. Immediate management involves terminating the dive, performing vinegar irrigation to neutralise unfired nematocysts, removing visible tentacles, and immersing in hot water (≤ 45°C) for analgesia and toxin denaturation. Severe facial or neck stings require airway monitoring and ocular irrigation. Bites from territorial marine fauna, though rare, require thorough wound care, exploration for foreign materials, and empiric antibiotic therapy. Preventive measures include neutral buoyancy, visual vigilance during ascent, protective gear, and appropriate situational awareness.[50]

Understanding these phase-specific mechanisms also informs clinical assessment of divers with pre-existing ENT conditions, where the same physiological stressors interact with altered anatomy or prior surgery.

FITNESS TO DIVE: CONSIDERATIONS IN PRE-EXISTING ENT CONDITIONS

ENT disorders frequently impact recreational divers, with approximately 80% of adult divers experiencing at least one related complaint,[33] and many of the aforementioned disorders are avoidable. But what about ENT patients seeking advice on whether or not they can dive? Clinicians often advise blanket water precautions, but evidence regarding diving safety is mixed.

Otological conditions

Otological issues represent significant considerations for divers. Surgical history, such as stapedectomy, poses a controversial risk. While regulatory bodies typically advise against diving post-stapedectomy due to potential inner ear barotrauma, clinical studies suggest that divers with adequate ET function may safely dive post-procedure.[51,52] Similarly, mastoidectomy poses variable risks depending on the procedure type.[52] Simple mastoidectomy may not preclude diving, but more extensive atticotomy usually does due to increased risks of direct pressure transfer and deeper infections.[33,52] Patients with ossicular prostheses in particular may be at increased risk of pressure transmission to the vestibule, particularly if equalisation is incomplete. For such individuals, careful dive clearance, counseling on conservative depth limits, and early symptom recognition are critical to minimising irreversible sequelae.[2] Pre-dive screening may not be limited to ENT assessment in these cases, as for example given the high prevalence of patent foramen ovale across IEDCS case series (reported in 60 to 83% of patients across multiple cohorts[13,37,40]) raises the question of whether cardiac shunt screening should be considered for divers presenting with unexplained inner ear symptoms or recurrent vestibular episodes. A recent consensus of experts from two international diving medicine societies recommends consideration of PFO screening for divers who have suffered IEDCS.[53]

Chronic ear disorders, such as cholesteatoma or active Ménière's disease, are generally contraindications for diving due to risks of vertigo exacerbation.[33] However, chronic external auditory canal or middle ear conditions with stable postoperative states, including tympanoplasty with successful ET function, may permit diving after comprehensive evaluation.[33,52,54] Specifically, type I-IIIb tympanoplasties with stable membranes are often compatible with diving if a negative provocative test (such as tympanometry without induced nystagmus or vertigo) is documented.[54]

Similarly, limited longitudinal evidence indicates that, when audiovestibular function and ET ventilation are normalised, divers with prior ear injury may return to diving without increased risk of recurrence.[29] ET dysfunction is problematic due to risks of middle ear barotrauma. Balloon tuboplasty has shown promise in restoring diving fitness by effectively managing persistent dysfunction.[33] Acute infections, especially otitis externa, represent temporary diving contraindications until fully resolved.[54]

Hearing loss itself is not an absolute contraindication for recreational diving, but unilateral deaf divers should be

cautioned about potential total deafness from contralateral injury.[33,54] Active middle ear implants and cochlear implants have specific pressure resistance limitations and require individual assessment of vestibular function and device integrity before diving.[52,54] Emerging evidence suggests that cochlear implant recipients can safely dive within certain depth limits, but repetitive hyperbaric exposure may still carry theoretical risks, including electrode dysfunction, pressure-related vertigo, or altered impedance settings.[17] Divers with cochlear implants or ossicular prostheses may encounter device-specific issues related to sustained hyperbaric exposure. Although contemporary cochlear implants are engineered to withstand pressures encountered during typical recreational dives (approximately 25–42 meters of seawater, depending on the manufacturer), repetitive dives have rarely resulted in electrode displacement, extrusion through the tympanic membrane, or device malfunction, manifesting as vertigo or tinnitus.[16,17]

Rhinological conditions

Septal deviation significantly affecting sinus drainage predisposes divers to sinus barotrauma and should be surgically corrected before diving if severe. Chronic inflammatory nasal conditions such as allergic rhinitis and chronic rhinosinusitis require symptom control with topical treatments; acute exacerbations must be resolved before diving resumes. Divers with vascular nasal diseases, like granulomatosis with polyangiitis, face absolute contraindications due to systemic involvement risks.[33]

Previous frontal or lateral skull base fractures necessitate rigorous evaluation due to pneumocephalus risk upon ascent. A negative β-2-transferrin test and computed tomography confirming sinus patency are required before diving can be considered safe.[33,54]

Laryngological and upper airway conditions

Acute laryngitis and other upper airway infections contraindicate diving until fully resolved due to risks of laryngospasm and impaired middle-ear pressure equalisation.[33,54] Anatomical abnormalities (e.g., laryngoceles, stenosis) preclude diving unless surgically corrected due to pulmonary barotrauma risks. Inflammatory conditions affecting the larynx, such as rheumatoid arthritis or relapsing polychondritis, constitute absolute contraindications due to the risk of respiratory compromise.[33] Contrastingly, unilateral vocal cord paralysis with adequate airway compensation and normal pulmonary function does not necessarily preclude diving.[33]

Diving post-treatment for head and neck cancer requires thorough evaluation due to anatomical distortions, friable vasculature, and risks from radiotherapy-induced fibrosis and oedema. Late otological effects from radiation, such as ET dysfunction, may impede middle ear pressure equalisation, thus warranting a specialist review.[33] Many patients will not have the performance status or desire to take on the risk, but those who do wish to should be given thorough counselling.

Conclusions

The phase-of-dive framework proposed here offers a clinically useful organising principle for ENT presentations in divers: descent injuries predominantly reflect failure of equalisation under negative pressure, bottom-phase complications arise from sustained exposure and equipment factors, and ascent-phase pathology encompasses a mix of reverse barotrauma and decompression sickness whose diagnostic ambiguity carries direct management consequences. IEDCS in particular is associated with incomplete recovery even after hyperbaric treatment, with persistent objective vestibular deficits documented in the majority of patients across multiple series.

The same framework may inform fitness-to-dive assessment by anticipating where altered anatomy or prior surgery creates vulnerability. What the available evidence cannot yet support is firm, quantitative guidance: the two controlled trials included here both showed null results for pharmacological prophylaxis, and the broader literature is dominated by small observational cohorts with moderate-to-high risk of bias. Prospective studies with standardised outcome measures, validated pre-dive screening protocols, and device-specific depth guidelines for implant recipients remain a genuine priority. Until then, the most useful tools available to the clinician are a mechanistically informed history anchored to dive phase, individualised risk assessment that accounts for dive profile, cumulative exposure, and anatomical predisposition, and realistic outcome counselling, particularly following IEDCS, where subjective recovery consistently outpaces objective vestibular function in the available data. For the diver, the practical takeaway is that most serious ENT complications are not random: they reflect identifiable physiological vulnerabilities that a well-informed clinician, and a well-informed diver, can anticipate and mitigate.

References

1 Darcy K. How to accurately measure the market size of the scuba diving industry. Scubanomics. 2024. [cited 2025 Jun 26]. Available from: https://medium.com/scubanomics/how-to-accurately-measure-the-market-size-of-the-scuba-diving-industry-ceb4ac0631d3.

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 Lindfors OH, Räisänen-Sokolowski AK, Hirvonen TP, Sinkkonen ST. Inner ear barotrauma and inner ear decompression sickness: a systematic review on differential diagnostics. Diving Hyperb Med. 2021;51:328–37. doi: 10.28920/dhm51.4.328-337. PMID: 34897597. PMCID: PMC8923696.

4 Alolaiwi LA, Alzahrani FA, Al Shammari SA. Diving into discomfort: orofacial pain dynamic – A systematic review. Front Public Health. 2025;13:1553541. doi: 10.3389/fpubh.2025.1553541. PMID: 40416680. PMCID: PMC12098605.

5 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.

6 Livingstone DM, Lange B. Rhinologic and oral-maxillofacial complications from scuba diving: a systematic review with recommendations. Diving Hyperb Med. 2018;48:79–83. doi: 10.28920/dhm48.2.79-83. PMID: 29888379. PMCID: PMC6156823.

7 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(3):252–8. doi: 10.1001/jamaoto.2017.2617. PMID: 29450472.

8 Klingmann C, Praetorius M, Baumann I, Plinkert PK. Otorhinolaryngologic disorders and diving accidents: an analysis of 306 divers. Eur Arch Otorhinolaryngol. 2007;264:1243–51. doi: 10.1007/s00405-007-0353-6. PMID: 17639445.

9 Skogstad M, Eriksen T, Skare Ø. A twelve-year longitudinal study of hearing thresholds among professional divers. Undersea Hyperb Med. 2009;36:25–31. PMID: 19341125.

10 St Leger Dowse M, Waterman MK, Jones R, Smerdon GR. Aural health awareness and incident prevention in UK scuba divers. Diving Hyperb Med. 2022;52:22–6. doi: 10.28920/dhm52.1.22-26. PMID: 35313369. PMCID: PMC9177430.

11 Doolette DJ, Mitchell SJ. Biophysical basis for inner ear decompression sickness. J Appl Physiol. 2003;94:2145–50. doi: 10.1152/japplphysiol.01090.2002. PMID: 12562679.

12 Mallen JR, Roberts DS. SCUBA medicine for otolaryngologists: Part I. Diving into SCUBA physiology and injury prevention. Laryngoscope. 2020;130:52–8. doi: 10.1002/lary.27867. PMID: 30776099.

13 Klingmann C, Praetorius M, Baumann I, Plinkert PK. Barotrauma and decompression illness of the inner ear: 46 cases during treatment and follow-up. Otol Neurotol. 2007;28:447–54. doi: 10.1097/MAO.0b013e318030d356. PMID: 17417111.

14 Mitchell SJ, Doolette DJ. Selective vulnerability of the inner ear to decompression sickness in divers with right-to-left shunt: the role of tissue gas supersaturation. J Appl Physiol (1985). 2009;106:298–301. doi: 10.1152/japplphysiol.90915.2008. PMID: 18801958.

15 Mitchell SJ, Doolette DJ. Pathophysiology of inner ear decompression sickness: potential role of the persistent foramen ovale. Diving Hyperb Med. 2015;45:105–10. PMID: 26165533. [cited 2026 Jan 18]. Available from: https://dhmjournal.com/images/IndividArticles/45June/Mitchell_dhm.45.2.105-110.pdf.

16 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.

17 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.

18 Lindfors OH, Räisänen-Sokolowski AK, Suvilehto J, Sinkkonen ST. Middle ear barotrauma in diving. Diving Hyperb Med. 2021;51:44–52. doi: 10.28920/dhm51.1.44-52. PMID: 33761540. PMCID: PMC8313771.

19 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.

20 Duplessis C, Fothergill D, Gertner J, Hughes L, Schwaller D. A pilot study evaluating surfactant on eustachian tube function in divers. Mil Med. 2008;173:1225–32. doi: 10.7205/milmed.173.12.1225. PMID: 19149344.

21 Blake DF, Gibbs CR, Commons KH, Brown LH. Middle ear barotrauma in a tourist-oriented, condensed open-water diver certification course: incidence and effect of language of instruction. Diving Hyperb Med. 2015;45:176–80. PMID: 26415068. [cited 2026 January 18]. Available from: https://www.dhmjournal.com/images/IndividArticles/45Sept/Blake_dhm.45.3.176-180.pdf.

22 Jansen S, Meyer MF, Boor M, Felsch M, Kluenter H-D, Pracht ED, et al. Prevalence of barotrauma in recreational scuba divers after repetitive saltwater dives. Otol Neurotol. 2016;37:1325–31. doi: 10.1097/MAO.0000000000001158. PMID: 27636390.

23 Meyer MF, Boor M, Jansen S, Pracht ED, Felsch M, Klünter M, et al. Influence of repetitive diving in saltwater on pressure equalization and Eustachian tube function in recreational scuba divers. Diving Hyperb Med. 2017;47:216–22. doi: 10.28920/dhm47.4.216-222. PMID: 29241230. PMCID: PMC6706334.

24 Moayedi S, Gizaw A, Sweet S, Sethuraman K, Witting M. Pseudoephedrine prophylaxis does not prevent middle ear barotrauma in hyperbaric oxygen therapy. Undersea Hyperb Med. 2025;52:101–7. PMID: 40819351.

25 Uzun C. Evaluation of predive parameters related to eustachian tube dysfunction for symptomatic middle ear barotrauma in divers. Otol Neurotol. 2005;26:59–64. PMID: 15699720.

26 Morvan J-B, Gempp E, Rivière D, Louge P, Vallee N, Verdalle P. Perilymphatic fistula after underwater diving: a series of 11 cases. Diving Hyperb Med. 2016;46:72–5. PMID: 27334993. [cited 2026 Jan 10]. Available from: https://www.dhmjournal.com/images/IndividArticles/46June/Morvan_dhm.46.2.72-75.pdf.

27 Rozycki SW, Brown MJ, Camacho M. Inner ear barotrauma in divers: an evidence-based tool for evaluation and treatment. Diving Hyperb Med. 2018;48:186–93. doi: 10.28920/dhm48.3.186-193. PMID: 30199891. PMCID: PMC6205852.

28 Ears & Diving. Divers Alert Network. n.d. [cited 2025 Jun 27]. Available from: https://dan.org/health-medicine/health-resource/dive-medical-reference-books/ears-diving/.

29 Parell GJ, Becker GD. Inner ear barotrauma in scuba divers: A long-term follow-up after continued diving. Arch Otolaryngol Head Neck Surg. 1993;119:455–7. doi: 10.1001/archotol.1993.01880160103016. PMID: 8457309.

30 Shupak A. Recurrent diving-related inner ear barotrauma. Otol Neurotol. 2006;27:1193–6. doi: 10.1097/01.mao.0000231499.69404.22. PMID: 16983314.

31 Chen T, Pathak S, Hong EM, Benson B, Johnson AP, Svider PF. Diagnosis and management of barosinusitis: A systematic review. Ann Otol Rhinol Laryngol. 2023;132:50–62. doi: 10.1177/00034894211072353. PMID: 35130739.

32 Lindfors OH, Räisänen-Sokolowski AK, Suvilehto J, Sinkkonen ST. Sinus barotrauma in diving. Diving Hyperb Med. 2021;51:182–9. doi: 10.28920/dhm51.2.182-189. PMID: 34157734. PMCID: PMC8426117.

33 Lechner M, Sutton L, Fishman JM, Kaylie DM, Moon RE, Masterson L, et al. Otorhinolaryngology and diving—Part 2: Otorhinolaryngological fitness for compressed gas scuba diving: A review. JAMA Otolaryngol Head Neck Surg. 2018;144:259–63. doi: 10.1001/jamaoto.2017.2616. PMID: 29450499.

34 Talmi YP, Finkelstein Y, Zohar Y. Decompression sickness induced hearing loss. A review. Scand Audiol. 1991;20:25–8. doi: 10.3109/01050399109070786. PMID: 1842265.

35 Burkett JG, Nahas SJ. Diving headache. Curr Pain Headache Rep. 2019;23(7):46. doi: 10.1007/s11916-019-0787-8. PMID: 31147799.

36 Mutzbauer TS, Neubauer B, Mueller PH, Sigg O, Tetzlaff K. Can eustachian tube ventilatory function impairment after oxygen diving be influenced by application of free radical scavenger vitamins C and E? Laryngoscope. 2001;111:861–6. doi: 10.1097/00005537-200105000-00020. PMID: 11359167.

37 Gempp E, Louge P. Inner ear decompression sickness in scuba divers: a review of 115 cases. Eur Arch Otorhinolaryngol. 2013;270:1831–7. doi: 10.1007/s00405-012-2233-y. PMID: 23100085.

38 Azzopardi CP, Caruana J, Matity L, Muscat S, Meintjes WAJ. 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.

39 Adkisson GH, Meredith AP. Inner ear decompression sickness combined with a fistula of the round window. Case report. Ann Otol Rhinol Laryngol. 1990;99(9 Pt 1):733–7. doi: 10.1177/000348949009900913. PMID: 2396811.

40 Mason JS, Buzzacott P, Gawthrope IC, Banham ND. A retrospective review of divers treated for inner ear decompression sickness at Fiona Stanley Hospital hyperbaric medicine unit 2014–2020. Diving Hyperb Med. 2023;53:243–50. doi: 10.28920/dhm53.3.243-50. PMID: 37718299. PMCID: PMC10735645.

41 Guenzani S, Mereu D, Messersmith M, Olivari D, Arena M, Spanò A. Inner-ear decompression sickness in nine trimix recreational divers. Diving Hyperb Med. 2016;46:111–6. PMID: 27334999. [cited 2026 Jan 10]. Available from: https://www.dhmjournal.com/images/IndividArticles/46June/Guenzani_dhm46.2.111-116.pdf.

42 Smart D. Five consecutive cases of sensorineural hearing loss associated with inner ear barotrauma due to diving, successfully treated with hyperbaric oxygen. Diving Hyperb Med. 2024;54:360–7. doi: 10.28920/dhm54.4.360-367. PMID: 39675746. PMCID: PMC12018693.

43 Stokes RJ, Watts D, Smerdon G, Hall SD, Bunn L, Marsden J. Vestibular rehabilitation and recovery in divers with inner ear decompression sickness: a case series. Diving Hyperb Med. 2025;55:236–45. doi: 10.28920/dhm55.3.236-245. PMID: 40986919. PMCID: PMC12790930.

44 Kitajima N, Sugita-Kitajima A, Kitajima S. Altered eustachian tube function in SCUBA divers with alternobaric vertigo. Otol Neurotol. 2014;35:850–6. doi: 10.1097/MAO.0000000000000329. PMID: 24751737.

45 Klingmann C, Knauth M, Praetorius M, Plinkert PK. Alternobaric vertigo--really a hazard? Otol Neurotol. 2006;27:1120–5. doi: 10.1097/01.mao.0000235373.78116.a8. PMID: 17130801.

46 Hyams AF, Toynton SC, Jaramillo M, Stone LR, Bryson PJ. Facial baroparesis secondary to middle-ear over-pressure: a rare complication of scuba diving. J Laryngol Otol. 2004;118:721–3. doi: 10.1258/0022215042244813. PMID: 15509373.

47 Canarslan Demir K, Yücel Z. Maxillary sinus barotrauma with infraorbital nerve paraesthesia after breath-hold diving. Diving Hyperb Med. 2024;54:230–2. doi: 10.28920/dhm54.3.230-232. PMID: 39288929. PMCID: PMC11659071.

48 Utz ER, Wise SR. Navy diver with recurrent facial nerve baroparesis treated with eustachian tube balloon dilation. Laryngoscope. 2019;129:E412–E414. doi: 10.1002/lary.28221. PMID: 31400145.

49 Giunta AA, Liberati L, Pellegrino C, Ricci G, Rizzo S. Eustachian tube balloon dilation in treatment of equalization problems of freediving spearfishermen. Diving Hyperb Med. 2019;49:9–15. doi: 10.28920/dhm49.1.9-15. PMID: 30856662. PMCID: PMC6526055.

50 Nochetto M, Denoble P. Hazardous marine life. Divers Alert Network. n.d. [cited 2025 Jul 3]. Available from: https://dan.org/health-medicine/health-resource/dive-medical-reference-books/hazardous-marine-life/.

51 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.

52 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 Hyperb. Med. 2026;56:177–84. doi: 10.28920/dhm56.2.177-184. PMID: 42290578.

53 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.

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

Conflicts of interest and funding: nil

Submitted: 18 January 2026
Accepted after revision: 22 May 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

Mental health and neurodevelopmental factors relevant to recreational fitness-to-dive assessment: a structured review and clinical considerations

Calina Ouliaris[1], Bram Querido[2], Thijs T Wingelaar[3,4], Neil D Banham[5]

[1] *Spring Hill, Brisbane, Australia*
[2] *Praktijk Querido, Hilversum, the Netherlands*
[3] *Dutch Society of Diving and Hyperbaric Medicine, Bilthoven, the Netherlands*
[4] *Royal Netherlands Navy, Diving Medical Center, Den Helder, the Netherlands*
[5] *Department of Hyperbaric Medicine, Fiona Stanley Hospital, Murdoch WA, Australia*

Corresponding author: *Dr Neil Banham Department of Hyperbaric Medicine, Fiona Stanley Hospital, 11 Robin Warren Drive, Murdoch WA 6150, Australia*
ORCiD: *0000-0002-1737-5859*
neil.banham@health.wa.gov.au

Keywords
Medicals – diving; Panic; Psychiatry; Psychotropic drugs; Risk assessment; Scuba diving; Stress

Abstract
(Ouliaris C, Querido B, Wingelaar TT, Banham ND. Mental health and neurodevelopmental factors relevant to recreational fitness-to-dive assessment: a structured review and clinical considerations. Diving and Hyperbaric Medicine. 2026 30 September;56(3):342–349. doi: 10.28920/dhm56.3.342-349. PMID: 42743575.)
Introduction: Mental health and neurodevelopmental factors, including psychiatric disorders and psychotropic medication use, are commonly identified during recreational diving medical screening. However, guidance for fitness-to-dive assessment in this context remains limited and often relies on diagnostic categories and theoretical risk, contributing to variability in clinical decision-making.
Methods: A structured review of the literature was conducted to identify evidence relevant to fitness-to-dive assessment where mental health and neurodevelopmental factors may be relevant. Searches of PubMed were supplemented by manual review of key references. Studies addressing cognitive function, behaviour, psychiatric stability, medication and substance effects and environmental influences in recreational diving were included and synthesised thematically.
Results: Twenty-nine studies were included. The evidence base was heterogeneous and comprised primarily observational studies, experimental research on cognitive performance under diving conditions and a smaller number of reviews and guidance-oriented publications. Across the literature, functional capacity, behavioural responses under stress and psychiatric stability emerged as central determinants of diving safety. Cognitive performance is reduced under diving conditions, suggesting limited cognitive reserve and increased vulnerability to impairment. Panic and maladaptive behavioural responses represent key pathways to adverse outcomes. Medication use alone is not a consistent predictor of risk, although side effects and periods of clinical instability are relevant. Environmental factors inherent to diving may further amplify underlying vulnerabilities.
Conclusions: Fitness-to-dive assessment in the context of mental health and neurodevelopmental factors cannot be determined by diagnosis alone. The available evidence supports a multidimensional, context-dependent approach incorporating functional capacity, behavioural reliability, psychiatric stability, medication effects and environmental demands. Assessment should be individualised and grounded in clinical judgement, recognising that risk is probabilistic rather than binary. These findings may support more consistent and clinically relevant decision-making in recreational diving.

Introduction

Medical fitness-to-dive assessments aim to identify health conditions that may increase the risk of injury, illness, or death in the underwater environment. Contemporary fitness-to-dive assessments are health risk assessments that aim to minimise medical risk rather than provide absolute certifications of safety.[1–3] Scuba diving imposes distinctive physiological, cognitive and behavioural demands, requiring emotional regulation, situational awareness, problem-solving and reliable behavioural responses under stress, including during emergencies.[1,4,5]

While many physical risk factors in diving, such as pulmonary function, cardiovascular fitness, or seizure risk, can be assessed against physiological thresholds or defined markers of severity, psychiatric risk is more context-dependent and less readily quantifiable. Psychiatric disorders are heterogeneous, often fluctuate over time and may interact

with stress, cognition and behaviour in ways that are difficult to predict in dynamic environments such as diving.[6,7]

Current approaches to fitness-to-dive assessment where mental health or neurodevelopmental factors may be relevant remain inconsistent. Diver medical screening processes routinely identify psychiatric diagnoses and psychotropic medication use as indications for further evaluation.[2,3] However, clinicians often lack structured guidance on how to interpret psychiatric diagnoses, medication effects, functional impairment and symptom stability in relation to diving risk.[8,9] Moreover, psychiatric diagnosis alone has limited predictive value for real-world functioning, as individuals with the same diagnosis may differ substantially in symptom severity, stability, insight and functional capacity.[6,10]

This review focuses on fitness-to-dive assessments in recreational (no-decompression) compressed air diving, as distinct from occupational, military and technical diving where operational demands, training requirements and environmental exposures differ, limiting generalisability. Within this scope, the review considers mental health and neurodevelopmental factors relevant to fitness-to-dive assessments, encompassing psychiatric disorders, neurodevelopmental conditions and associated cognitive, behavioural and functional considerations.

Neurodevelopmental conditions warrant particular consideration due to their distinct developmental and neurocognitive characteristics. These conditions typically emerge in childhood and are characterised by enduring differences in attention, executive functioning, cognitive flexibility and behavioural regulation. This contrasts with many other psychiatric disorders, which may present with more episodic changes in mood, perception or behavioural control.[11,12] Although these domains overlap with broader functional considerations relevant to diving safety, the implications for fitness-to-dive assessment may differ, with greater emphasis placed on adaptation, environmental structure, task management and individual variability in functioning.[13–15]

Existing guidance from diving medicine organisations recognises that the diagnosis of a psychiatric disorder, particularly when associated with sustained stability and preserved functional performance, is not in itself a contraindication to recreational diving.[9,16] Guidance provides general recommendations regarding fitness-to-dive, including consideration of psychiatric disorders; however, it remains largely framed in diagnostic terms and does not consistently address how functional capacity, symptom stability, behavioural responses and medication effects should be interpreted in relation to diving risk. While this approach may be appropriate for certain physical health conditions with defined physiological parameters, it is less readily applicable to mental health and neurodevelopmental conditions, where risk is often more context-dependent and shaped by interactions between cognition, behaviour and environmental demands.[6] Concerns regarding inconsistent assessment or perceived automatic exclusion may contribute to under-disclosure, with potential implications for diver safety and may be compounded by broader stigma associated with psychiatric diagnoses and treatment.[17–19] Together, these challenges create tension between promoting safe access, avoiding inappropriate exclusion and meeting medico-legal responsibilities.

Diving-related risk arises from the interaction between individual characteristics and environmental demands, including cognitive impairment associated with depth and gas effects, increased task load and the potential for rapid escalation of stress responses.[1,4,5] Evidence from diving medicine and human factors research indicates that behavioural factors such as situational awareness, decision-making under pressure and behavioural reliability (the ability to maintain consistent and self-preservative behaviour under stress), are central to diving safety even in the absence of diagnosed psychiatric disorder.[4,5,20] Panic and anxiety represent specific expressions of these broader mechanisms and are widely recognised contributors to recreational diving incidents and fatalities.[4,20–22] These findings highlight the importance of not relying on diagnostic categories alone.

Currently, clinicians are often required to rely on individual judgement when assessing fitness-to-dive in individuals where mental health and neurodevelopmental considerations may be relevant.[8] This review presents a structured examination of the literature relating to mental health and neurodevelopmental factors relevant to fitness-to-dive in recreational scuba diving. The review is intentionally structured around functional domains while recognising that diagnostic categories remain clinically relevant. This reflects the clinical reality that diving-related risk cannot be determined by diagnosis alone, but requires consideration of broader cognitive, behavioural, functional and contextual factors. Findings are synthesised to support clinical interpretation and more consistent decision-making.

Methods

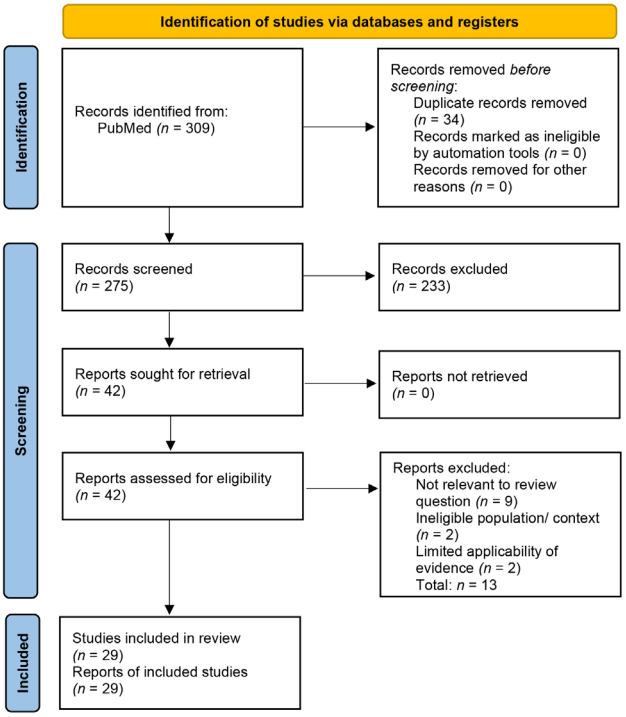
This review was conducted using a structured literature search and study selection process informed by PRISMA guidelines.[23] The objective was to identify and synthesise evidence relevant to mental health and neurodevelopmental factors in recreational fitness-to-dive assessment, with a focus on clinically relevant interpretation rather than formal quantitative synthesis.

The PubMed database was searched from inception to April 2026 using a structured, multi-stage approach combining Medical Subject Headings (MeSH) and targeted supplementary searches. A primary search using MeSH terms for "*diving*" and "*mental disorders*" was conducted to identify core literature. Additional targeted searches were conducted to capture relevant domains not consistently

Table 1
PubMed search queries

Core search (“Diving”[MeSH]) AND (“Mental Disorders”[MeSH])
Cognitive and behavioural domain (“Diving”[MeSH]) AND (“Mental Disorders”[MeSH]) AND (cognition[tiab] OR cognitive[tiab] OR behaviour[tiab] OR behavior[tiab] OR “human factors”[tiab] OR executive[tiab])
Psychotropic medication domain (“Diving”[MeSH]) AND (“Psychotropic Drugs”[MeSH] OR psychotropic[tiab] OR antidepressant*[tiab] OR anxiolytic*[tiab] OR benzodiazepine*[tiab] OR SSRI[tiab] OR SNRI[tiab] OR lithium[tiab])
Fitness-to dive domain (“Diving”[MeSH]) AND (“fitness to dive”[tiab] OR “fit to dive”[tiab] OR “medical clearance”[tiab] OR “risk assessment”[tiab] OR “diving medical”[tiab]).

Figure 1
PRISMA diagram for the search and study selection



indexed under these headings, including cognitive and behavioural factors, psychotropic medication use and fitness-to-dive and medical clearance terminology. These supplementary searches used the combinations of MeSH and free-text terms within titles and abstracts shown in Table 1. Search terms were developed from key conceptual domains and iteratively refined to balance sensitivity and specificity.

Duplicate records were removed prior to screening. Records were screened by title and abstract, followed by full-text review and assessed for eligibility according to predefined inclusion and exclusion criteria. Studies were included if they examined recreational scuba diving or closely related contexts and addressed mental health or neurodevelopmental factors, psychotropic medication, cognition, behaviour, or functional performance relevant to diving safety.

Studies were excluded based on lack of relevance to the review question, ineligible population or context, or limited applicability of the evidence (e.g., animal studies or single case reports). The study selection process is summarised in a flow diagram (Figure 1) that follows PRISMA terminology, distinguishing between “records” (search results), “reports” (full-text articles assessed) and “studies” (included articles).[23]

Study characteristics and findings relevant to fitness-to-dive assessment were reviewed and synthesised. Given heterogeneity in study design and outcomes, formal quantitative synthesis was not undertaken. Findings were synthesised thematically using domains reflecting key constructs relevant to fitness-to-dive assessment, including functional capacity, psychiatric stability, behavioural responses, medication and substance effects, neurodevelopmental conditions and environmental influences. These domains were refined iteratively in the process of reviewing the included studies.

No formal quality appraisal tool was applied due to heterogeneity of study designs.

Results

A total of 309 records were identified through database searching. After removal of duplicates, 275 records were screened by title and abstract and 42 full-text articles were assessed for eligibility. Following application of inclusion and exclusion criteria, 29 studies were included in the final analysis (Figure 1).

The included literature varied in design, methodology and outcome measures, comprising primarily observational studies and experimental research examining the cognitive effects of diving, alongside a smaller number of narrative reviews, position statements and guidance-oriented publications. Direct evidence relating to mental health and neurodevelopmental factors in diving contexts was limited, with much of the literature focusing on broader cognitive, behavioural and human factors relevant to diving safety.

The available evidence varied in scope, methodology and study population.

Across the included studies, fitness-to-dive assessment is best understood as a multidimensional construct in which risk arises from the interaction between domains, rather than from any single factor in isolation. To support clinical interpretation, findings were synthesised across six thematic domains relevant to mental health and neurodevelopmental considerations in fitness-to-dive assessment. These domains were identified through iterative review of the included studies and reflect clinically relevant constructs associated with diving safety and risk. Five domains relate primarily to individual factors relevant to diving safety, while 'Environmental demands' was considered as a contextual domain that may impact on the expression and significance of vulnerabilities identified across other domains. A recurring finding was that diagnosis alone appeared insufficient to explain diving-related risk.

FUNCTIONAL CAPACITY

Across the literature, functional capacity emerged as a key determinant of diving safety. Diving requires sustained attention, executive functioning, situational awareness and the ability to manage task load while responding to unexpected events.[9,13,24]

Experimental studies demonstrated measurable impairments in cognitive performance under diving conditions, including reductions in attention, working memory, psychomotor speed and information processing.[25–31] These effects have been observed even at recreational depths and are consistent with the combined effects of increased partial pressure of inert gases, including nitrogen narcosis and environmental task loading.[26,31] Cognitive impairment under these conditions may reduce error detection, slow reaction time and impair information processing relevant to decision-making.[32,33]

These findings suggest that, in the underwater environment, divers may operate with reduced cognitive reserve, understood here as the finite cognitive resources available to an individual to maintain effective cognitive performance despite competing task demands, environmental stressors and physiological impairment.[25,26,29] Individuals with pre-existing vulnerabilities, including those related to psychiatric disorders, neurodevelopmental differences or medication effects, may have reduced capacity to compensate for additional environmental stressors.[9,14,15,34,35] This has direct implications for fitness-to-dive assessment, as individuals who function adequately in routine environments may demonstrate reduced cognitive performance under diving conditions.[25,29]

Clinical and review-based literature emphasised that fitness-to-dive assessment should prioritise the ability to understand procedures, plan and execute tasks, maintain situational awareness and adapt to changing conditions.[9,13,36,37] Impairments in these domains are not always apparent in routine clinical settings but may become functionally significant in dynamic and time-sensitive underwater environments.[21,26] Functional capacity reflects an individual's ability to process information and manage task demands. However, diving-related risk is not determined by capacity alone, but also by how that capacity is expressed under conditions of stress, where behavioural responses become a key determinant of safety.

BEHAVIOURAL RESPONSES AND RELIABILITY

Behavioural responses under stress are identified as critical determinants of diving safety.[13,24] Panic and acute anxiety represent well-recognised pathways to adverse outcomes, including rapid ascent, loss of buoyancy control and deviation from established dive procedures.[19,24]

Evidence from behavioural models and observational studies suggests that adverse events in recreational diving often arise from a chain of behavioural and cognitive errors rather than single-point failures.[24,26] Under conditions of stress, cognitive impairment may occur, contributing to reduced situational awareness and problem-solving capacity and thereby increasing the likelihood of maladaptive responses.[25,26] Panic may further amplify these processes, leading to loss of behavioural control and unsafe actions.[24]

These behavioural responses are not confined to individuals with diagnosed psychiatric disorders or neurodevelopmental conditions;[19] however, some conditions may increase susceptibility to heightened stress reactivity, panic, or impaired emotional regulation.[13–15,34,38] Prior panic episodes, difficulty tolerating physiological sensations such as dyspnoea, or reduced capacity to maintain behavioural control under pressure are therefore clinically relevant considerations in fitness-to-dive assessment.[24]

Survey and qualitative studies demonstrated that divers may underestimate risk or rationalise unsafe behaviours, particularly in the context of substance use or overconfidence.[19,39,40] These findings underscore the importance of assessing stress tolerance, behavioural reliability and prior responses to challenging situations as part of fitness-to-dive assessment.

PSYCHIATRIC STABILITY

Evidence indicates that psychiatric diagnosis alone appears to have limited predictive value for diving safety. Clinical relevance lies in psychiatric stability, including sustained symptom control, treatment adherence, preserved insight and consistent day-to-day functioning.[9,34,38,41]

Instability, fluctuating symptoms, or impaired insight may increase vulnerability to impaired judgement or behavioural dysregulation underwater, particularly in environments where stress may escalate rapidly. Conditions associated

with impaired reality testing and/or dissociation may carry higher potential risk due to the possibility of compromised situational awareness or behavioural control.[24,34,38,41]

The literature emphasises that fitness-to-dive assessment should focus on current functioning and stability over time, rather than diagnostic category alone.[9] This includes consideration of relapse patterns, early warning signs and the individual's ability to recognise and respond to deterioration. In episodic psychiatric conditions, functional recovery and restoration of reliable judgement may lag behind symptomatic improvement, underscoring the importance of longitudinal assessment rather than reliance on symptom status alone.[34,38]

MEDICATION AND SUBSTANCE EFFECTS

Medication use is increasingly encountered among recreational divers and represents an important consideration in fitness-to-dive assessment.[9,42] Observational data indicate that many divers with medical comorbidities take regular medications, often across multiple classes and may continue to dive safely, suggesting that medication use alone is not a sufficient determinant of risk.[42] Rather than acting as absolute contraindications, medications are best understood in terms of their potential impact on cognitive function, physiological stability and behavioural responses under stress.[9]

Psychotropic medications, along with other commonly prescribed drug classes, are associated with side effects such as sedation, cognitive impairment, reduced concentration and autonomic or cardiovascular effects. These effects may impair attention, situational awareness and response capacity in the underwater environment. Side effects are often most pronounced during periods of treatment initiation, dose adjustment, or when multiple medications are used concurrently and may introduce variability in cognitive and behavioural function.[9,35]

Despite these concerns, empirical evidence regarding the interaction between medications and diving-specific risks remains limited. Current literature does not demonstrate a clear or consistent increase in severe narcosis attributable to medication use alone and the mechanisms by which medications interact with the diving environment, particularly conditions of increased ambient pressure, are not well defined.[43] Observational studies suggest that it is often difficult to distinguish the effects of medications from those of the underlying medical or psychiatric disorders for which they are prescribed.[42] This is further complicated through factors such as variability in adherence, under-recognition of side effects and challenges in risk appraisal.[9] As such, medication effects are best interpreted within a broader clinical context rather than as isolated risk factors.

In addition to prescribed medications, substance use represents a relevant but less well-characterised domain. Alcohol use has been associated with impaired cognitive performance, reduced reaction time and poorer decision-making, all of which are directly relevant to diving safety.[40,44] Cannabis use has similarly been associated with altered perception, impaired attention and changes in risk appraisal, which may affect both functional capacity and behavioural responses underwater.[39]

Evidence regarding other illicit substances in diving is limited. However, given their known effects on cognition, perception and physiological regulation, these substances may be relevant to risk in the diving environment. Patterns of substance use may be associated with risk-taking behaviour, reduced adherence to safety recommendations and variability in decision-making.[19,39] Beyond its direct pharmacological effects, substance use may reflect underlying psychiatric disorders, psychiatric instability or maladaptive coping strategies and may also meet diagnostic criteria for a substance use disorder.[11,12] Substance use may further contribute to mental health deterioration, precipitate relapse and interfere with treatment adherence.

These findings support a contextual and individualised approach in which medication and substance use are evaluated alongside the underlying condition, functional capacity and behavioural responses under stress.

NEURODEVELOPMENTAL FACTORS

Although many functional domains relevant to neurodevelopmental conditions overlap with broader fitness-to-dive considerations, these conditions warrant separate discussion due to their enduring developmental and neurocognitive characteristics.

Emerging evidence relating to neurodevelopmental conditions, including attention deficit hyperactivity disorder (ADHD) and autism spectrum disorder (ASD), highlights substantial individual variability in functional capacity in the context of diving.[13–15,45] These conditions are not absolute contraindications to diving and suitability is best considered on an individual basis.[14,15]

Relevant domains include attention, executive function, cognitive flexibility and the ability to adapt to changing or unexpected situations, all of which have been identified as important for safe diving performance. Difficulties with distractibility, task switching, or cognitive rigidity may affect performance in dynamic underwater environments. However, some individuals demonstrate effective functioning, particularly within structured and predictable contexts.[14,15,46]

These findings support a functional approach to fitness-to-dive assessment, with emphasis on task management, behavioural regulation and adaptability rather than diagnostic category alone.

ENVIRONMENTAL DEMANDS

Environmental factors inherent to diving, including increased ambient pressure, thermal stress and task load, may impair cognitive and psychomotor performance.[25,26,29] These effects may not be fully recognised by the diver, creating a mismatch between perceived and actual functioning.[31]

Within a fitness-to-dive assessment, environmental demands are best understood as amplifiers of underlying vulnerability rather than independent determinants of risk.

The interaction between environmental stressors and individual vulnerability is central to understanding diving-related risk. Individuals with reduced cognitive reserve, impaired insight, or increased stress reactivity may be disproportionately affected when environmental demands exceed available compensatory resources.[24] This reinforces the importance of assessing not only baseline functioning, but also the likely interaction between individual vulnerability and the anticipated diving environment.[9,36,37]

Discussion

The results of this review support a multidimensional, function-based model of fitness-to-dive assessment in which diving-related risk is not attributed to diagnostic category alone but emerges from the interaction between individual capacity and the physiological, cognitive and environmental demands of diving. This reflects a broader principle in psychiatric risk assessment, in which functional performance and behavioural reliability provide important information beyond diagnostic labels alone, particularly in dynamic and safety-critical environments.[6,9]

This does not diminish the clinical relevance of psychiatric or neurodevelopmental diagnoses. Certain disorders may be associated with differing patterns of vulnerability, including impairments in attention, executive functioning, behavioural regulation, insight, reality testing or stress tolerance. However, diagnosis alone provides limited information regarding how these vulnerabilities manifest in practice or how they interact with the demands of the diving environment. The findings of this review support a structured multidimensional approach, elements of which are reflected, to varying degrees, in contemporary diving medicine guidance.[2,3,47,48] This includes guidance for consideration of multiple factors for case-by-case assessment,[2,3,47] and guidelines in which principles of a multidimensional approach are integrated.[48] The present review seeks to make the underlying principles more explicit by integrating diagnostic, functional, behavioural and environmental considerations within a broader multidimensional approach to fitness-to-dive assessment that acknowledges interactions between domains.

From a clinical perspective, the reduction in cognitive reserve under diving conditions has important implications for clinical assessment.[31,32] Individuals who function adequately in low-demand environments may not demonstrate equivalent performance underwater, where task load, environmental stressors and physiological factors place additional demands on attention, working memory and decision-making processes.[25–27]

Assessment should therefore extend beyond baseline functioning to include the ability to maintain performance under increased demand. Relevant considerations include panic vulnerability, behavioural dysregulation, impaired reality testing, executive dysfunction and reduced self-preservative behaviour. Among these domains, self-preservative behaviour represents a clinically important construct that is insufficiently examined in the diving literature. Maladaptive elements may include active suicidality, passive self-endangerment, diminished hazard awareness, or indifference to risk, all of which may be highly consequential in the underwater environment. While such factors may be routinely considered within psychiatric risk assessment, they are rarely incorporated into standard fitness-to-dive screening tools, despite their potential relevance to diving safety.

The findings also highlight the importance of contextual assessment. Environmental stressors may amplify vulnerabilities that are otherwise well compensated for in everyday settings. Fitness-to-dive assessment should therefore consider not only factors relevant to the individual but also the anticipated demands of the diving environment and the extent to which these factors may interact.

These findings have important clinical implications. Fitness-to-dive assessment in the context of mental health and neurodevelopmental considerations is inherently qualitative and probabilistic, requiring clinical judgement applied to a combination of diagnostic, functional, behavioural and contextual factors. This creates an ongoing tension between promoting safe access to diving, avoiding unnecessary exclusion and managing medico-legal responsibilities. Overly rigid or diagnosis-based approaches may contribute to inappropriate restriction or to under-disclosure, while insufficiently structured assessments may fail to identify relevant risks.[17,18]

The evidence base remains limited and heterogeneous, comprising predominantly observational studies, small experimental studies and expert opinion. Few studies directly examine psychiatric and neurodevelopmental factors in diving contexts and the interaction between these factors and diving-specific stressors remains incompletely understood. Future research should prioritise prospective and interdisciplinary approaches to better characterise these relationships, including the role of stress responses, cognitive performance under load and the effects of medication in hyperbaric environments.

Finally, the findings suggest that development of a structured but flexible framework for fitness-to-dive assessment may be beneficial. Such a framework could provide a shared language for clinicians to integrate diagnostic, functional, behavioural and environmental considerations, articulate the basis of their recommendations and apply proportionate risk mitigation strategies. Where uncertainty exists, a conservative approach remains appropriate but should be balanced against recognition that fitness-to-dive is not a binary state and may evolve over time.

Conclusions

Fitness-to-dive assessment in the context of mental health and neurodevelopmental factors cannot be determined by diagnostic category alone. The available evidence supports a multidimensional approach that integrates diagnostic, functional, behavioural and environmental considerations in fitness-to-dive assessment. While the evidence base remains limited, the findings support an individualised and context-dependent approach to fitness-to-dive assessment.

Development of a structured but flexible framework informed by these domains may support greater consistency, transparency and safety in fitness-to-dive assessment while facilitating appropriate participation and minimising unnecessary exclusion from recreational diving.

References

1 Edmonds C, Bennett M, Lippmann J, Mitchell SJ. Medical standards for recreational divers. In: Diving and subaquatic medicine. 5th ed. Boca Raton (FL): CRC Press; 2015.

2 South Pacific Underwater Medicine Society (SPUMS). The South Pacific Underwater Medicine Society guidelines on medical risk assessment for recreational diving (6th edition); 2025. [cited 2026 Jun 15]. Available from: https://spums.au/index.php/diving-medicals/spums-full-medical.

3 Undersea and Hyperbaric Medical Society (UHMS). Diving medical guidance to the physician. Durham (NC); 2021. [cited 2026 Jun 15]. Available from: https://www.uhms.org/images/Recreational-Diving-Medical-Screening-System/forms/Diving_Medical_Guidance_EN_English_2023-05-31.pdf.

4 Kovacs C. Scuba diving and the stress response: considerations and recommendations for professional and recreational divers. Int Marit Health. 2023;74:186–91. doi: 10.5603/imh.91707. PMID: 37781945.

5 Lippmann J, Stevenson C, Taylor D, Williams J, Mohebbi M. Chain of events analysis for a scuba diving fatality. Diving Hyperb Med. 2017;47:144–54. doi: 10.28920/dhm47.3.144-154. PMID: 28868594. PMCID: PMC6159623.

6 Kotov R, Krueger RF, Watson D, Achenbach TM, Althoff RR, Bagby RM, et al. The hierarchical taxonomy of psychopathology (HiTOP): A dimensional alternative to traditional nosologies. J Abnorm Psychol. 2017;126:454–77. doi: 10.1037/abn0000258. PMID: 28333488.

7 Plana-Ripoll O, Pedersen CB, Agerbo E, Holtz Y, Erlangsen A, Canudas-Romo V, et al. A comprehensive analysis of mortality-related health metrics associated with mental disorders: a nationwide, register-based cohort study. Lancet. 2019;394(10211):1827–35. doi: 10.1016/S0140-6736(19)32316-5. PMID: 31668728.

8 Cheung T-K, Meintjes WA. The usefulness of the RSTC medical questionnaire in pre-participation health risk assessment of recreational scuba divers in Hong Kong. Diving Hyperb Med. 2021;51:173–81. doi: 10.28920/dhm51.2.173-181. PMID: 34157733. PMCID: PMC8426120.

9 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.

10 National Institute for Health and Care Excellence. Service user experience in adult mental health: improving the experience of care for people using adult NHS mental health services. Clinical Guideline CG 136. London: NICE; 2011. [cited 2026 Jun 15]. Available from: https://www.nice.org.uk/guidance/cg136.

11 American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Arlington (VA): American Psychiatric Association; 2013.

12 World Health Organization. International classification of diseases 11th Revision (ICD-11). Geneva; 2019. [cited 2026 Jun 15]. Available from: https://icd.who.int.

13 Elliott E, Smart D, Lippmann J, Banham N, Nochetto M, Roehr S. South Pacific Underwater Medicine Society (SPUMS) position statement regarding paediatric and adolescent diving. Diving Hyperb Med. 2024;54:338–43. doi: 10.28920/dhm54.4.338-343. PMID: 39675742. PMCID: PMC11779525.

14 Querido AL, van Hulst R. Diving and attention deficit hyperactivity disorder. Diving Hyperb Med. 2019;49:41–7. doi: 10.28920/dhm49.1.41-47. PMID: 30856666. PMCID: PMC6526049.

15 Querido AL, Wingelaar TT. Assessing dive fitness in individuals with autism spectrum disorder. Diving Hyperb Med. 2025;55:254–61. doi: 10.28920/dhm55.3.254-261. PMID: 40986921. PMCID: PMC12790926.

16 Roth A. Psychiatric fitness to dive and remission: Divers Alert Network (DAN); 2011. [cited 2026 Jun 11]. Available from: https://www.dan.org/alert-diver/article/psychiatric-fitness-to-dive/.

17 Clement S, Schauman O, Graham T, Maggioni F, Evans-Lacko S, Bezborodovs N, et al. What is the impact of mental health-related stigma on help-seeking? A systematic review of quantitative and qualitative studies. Psychol Med. 2015;45:11–27. doi: 10.1017/S0033291714000129. PMID: 24569086.

18 Corrigan PW, Druss BG, Perlick DA. The impact of mental illness stigma on seeking and participating in mental health care. Psychol Sci Public Interest. 2014;15:37–70. doi: 10.1177/1529100614531398. PMID: 26171956.

19 St Leger Dowse M, Whalley B, Waterman MK, Conway RM, Smerdon GR. Diving and mental health: the potential benefits and risks from a survey of recreational scuba divers. Diving Hyperb Med. 2019;49:291–7. doi: 10.28920/dhm49.4.291-297. PMID: 31828748. PMCID: PMC7039781.

20 Van Wijk CH. Personality and behavioural outcomes in diving: current status and recommendations for future research. Diving Hyperb Med. 2017;47:248–52. doi: 10.28920/dhm47.4.248-252. PMID: 29241235. PMCID: PMC6706337.

21 Buzzacott P, Pikora T, Rosenberg M, Heyworth J. Rapid ascent and buoyancy problems among Western Australian certified recreational divers. Diving Hyperb Med. 2012;42:30–5. PMID: 22437973. [cited 2026 Jun 15]. Available from:

https://dhmjournal.com/images/IndividArticles/42March/Buzzacott_dhm.42.1.30-35.pdf.
22 Flin R, O'Connor P, Crichton M. Safety at the sharp end: a guide to non-technical skills. 1st ed. London: CRC Press; 2008.
23 Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ. 2021;372:n71. doi: 10.1136/bmj.n71. PMID: 33782057. PMCID: PMC8005924.
24 Walton L. The panic triangle: onset of panic in scuba divers. Undersea Hyperb Med. 2018;45:505–9. PMID: 30428239.
25 Freiberger JJ, Derrick BJ, Natoli MJ, Akushevich I, Schinazi EA, Parker C, et al. Assessment of the interaction of hyperbaric N_2, CO_2, and O_2 on psychomotor performance in divers. J Appl Physiol (1985). 2016;121:953–64. doi: 10.1152/japplphysiol.00534.2016. PMID: 27633739.
26 Hobbs M, Higham PA, Kneller W. Memory and metacognition in dangerous situations: Investigating cognitive impairment from gas narcosis in undersea divers. Hum Factors. 2014;56:696–709. doi: 10.1177/0018720813510737. PMID: 25029895.
27 Lafère P, Hemelryck W, Germonpré P, Matity L, Guerrero F, Balestra C. Early detection of diving-related cognitive impairment of different nitrogen-oxygen gas mixtures using critical flicker fusion frequency. Diving Hyperb Med. 2019;49:119–26. doi: 10.28920/dhm49.2.119-126. PMID: 31177518. PMCID: PMC6704008.
28 Philp R, Fields G, Roberts W. Memory deficit caused by compressed air equivalent to 36 meters of seawater. J Appl Psychol. 1989;74:443–6. doi: 10.1037/0021-9010.74.3.443. PMID: 2737993.
29 Slosman DO, De Ribaupierre S, Chicherio C, Ludwig C, Montandon M-L, Allaoua M, et al. Negative neurofunctional effects of frequency, depth and environment in recreational scuba diving: the Geneva "memory dive" study. Br J Sports Med. 2004;38:108–14. doi: 10.1136/bjsm.2002.003434. PMID: 15039241. PMCID: PMC1724771.
30 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.
31 Hamilton K, Laliberté MF, Fowler B. Dissociation of the behavioral and subjective components of nitrogen narcosis and diver adaptation. Undersea Hyperb Med. 1995;22:41–9. PMID: 7742709.
32 Hobbs M. Subjective and behavioural responses to nitrogen narcosis and alcohol. Undersea Hyperb Med. 2008;35:175–84. PMID: 18619113.
33 Monteiro MG, Hernandez W, Figlie NB, Takahashi E, Korukian M. Comparison between subjective feelings to alcohol and nitrogen narcosis: a pilot study. Alcohol. 1996;13:75–8. doi: 10.1016/0741-8329(95)02016-0. PMID: 8837939.
34 Querido AL, Wingelaar TT. Psychosis and diving. Diving Hyperb Med. 2025;55:173–9. doi: 10.28920/dhm55.2.173-9. PMID: 40544145. PMCID: PMC12520999.
35 Querido AL. Diving and antidepressants. Diving Hyperb Med. 2017;47:253–6. doi: 10.28920/dhm47.4.253-6. PMID: 29241236. PMCID: PMC6708605.
36 Krzyżak J, Korzeniewski K. Medical assessment of fitness to dive. Part I. Int Marit Health. 2021;72:36–45. doi: 10.5603/MH.2021.0005. PMID: 33829471.
37 Krzyżak J, Korzeniewski K. Medical assessment of fitness to dive. Part II. Int Marit Health. 2021;72:115–20. doi: 10.5603/IMH.2021.0020. PMID: 34212351.
38 Querido AL, Wingelaar TT. Bipolar spectrum disorders in divers: risks, recognition, and recommendations. Diving Hyperb Med. 2026;56:170–6. doi: 10.28920/dhm56.2.170-176. PMID: 42290577.
39 Martin JH, Van Wijk CH, Bowden WJ. Diving, cannabis use, and techniques of neutralisation: exploring how divers rationalise cannabis use. Int Marit Health. 2019;70:88–94. doi: 10.5603/IMH.2019.0014. PMID: 31237667.
40 Perrine MW, Mundt JC, Weiner RI. When alcohol and water don't mix: diving under the influence. J Stud Alcohol. 1994;55:517–24. doi: 10.15288/jsa.1994.55.517. PMID: 7990461.
41 Smart D, Lippmann J. Epilepsy, scuba diving and risk assessment. Near misses and the need for ongoing vigilance. Diving Hyperb Med. 2013;43:37–41. PMID: 23508661. [cited 2026 Jun 15]. Available from: https://dhmjournal.com/images/IndividArticles/43March/Smart_dhm.43.1.37-41.pdf.
42 Taylor SE, Taylor DM, Pisasale D, Booth K, Lippmann J. Regular medication use by active scuba divers with a declared comorbid medical condition and victims of scuba and snorkelling-related fatalities. Diving Hyperb Med. 2021;51:264–70. doi: 10.28920/dhm51.3.264-270. PMID: 34547777. PMCID: PMC8608444.
43 Krummel T, Thiery A, Villain M, Schittly B, Brouant B. Psychotropic drug use in recreational scuba divers and its effect on severe narcosis. Int J Sports Med. 2017;38:322–8. doi: 10.1055/s-0042-122336. PMID: 28249344.
44 St Leger Dowse M, Cridge C, Shaw S, Smerdon G. Alcohol and UK recreational divers: consumption and attitudes. Diving Hyperb Med. 2012;42:201–7. PMID: 23258456. [cited 2026 Jun 15]. Available from: https://dhmjournal.com/images/IndividArticles/42Dec/StLegerDowse_dhm.42.4.201-207.pdf.
45 Naumann K, Kernot J, Parfitt G, Gower B, Winsor A, Davison K. What are the effects of scuba diving-based interventions for clients with neurological disability, autism or intellectual disability? A systematic review. Diving Hyperb Med. 2021;51:355–60. doi: 10.28920/dhm51.4.355-360. PMID: 34897600. PMCID: PMC8920902.
46 Buwalda M, Querido AL, van Hulst RA. Children and diving, a guideline. Diving Hyperb Med. 2020;50:399–404. doi: 10.28920/dhm50.4.399-404. PMID: 33325022. PMCID: PMC8026229.
47 UK Diving Medical Committee. Medical conditions. [cited 2026 Jun 11]. Available from: https://www.ukdmc.org/medical-conditions/medical-conditions/.
48 Nederlandse Vereniging voor Duikgeneeskunde (NVD; Dutch Society of Diving and Hyperbaric Medicine). Position statement on diving with ADHD/ADD; 2024. [cited 2026 Jun 11]. Available from: https://duikgeneeskunde.nl/wp-content/uploads/2026/06/NVD_position_statement_diving_with_ADHD_EN-2024-1.pdf.

Conflicts of interest and funding: nil

Submitted: 22 April 2025
Accepted after revision: 25 July 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

Ultrasound protocols used to detect vascular gas emboli in divers: a systematic review

Benjamin L Turner[1], Thijs T Wingelaar[1,2], Feiko JM de Jong[1,3], Pieter Jan AM van Ooij[1,3], Rigo Hoencamp[4,5,6]

[1] *Royal Netherlands Navy, Diving and Submarine Medical Centre, Den Helder, the Netherlands*
[2] *Royal Netherlands Air and Space Force, Center for Man in Aviation, Soesterberg, the Netherlands*
[3] *Amsterdam UMC, Location AMC, Department of Respiratory Medicine, Amsterdam, the Netherlands*
[4] *Alrijne Hospital, Department of Surgery, Leiderdorp, the Netherlands*
[5] *Erasmus MC, University Medical Center Rotterdam, Trauma Research Unit, Rotterdam, the Netherlands*
[6] *Ministry of Defense, Defense Healthcare Organization, Utrecht, the Netherlands*

Corresponding author: *Benjamin L Turner, Royal Netherlands Navy, Diving and Submarine Medical Centre, Den Helder, the Netherlands*
blturner@alrijne.nl

Keywords
Air embolism; Doppler; Echocardiography; Radiological imaging; Systematic review; Ultrasound; Venous gas embolism

Abstract
(Turner BL, Wingelaar TT, de Jong FJM, van Ooij PJAM, Hoencamp R. Ultrasound protocols used to detect vascular gas emboli in divers: a systematic review. Diving and Hyperbaric Medicine. 2026 30 September;56(3):350–360. doi: 10.28920/dhm56.3.350-360. PMID: 42743576.)
Introduction: Venous gas emboli (VGE) detected via ultrasound can be used as a surrogate marker for decompression stress. While Doppler ultrasound is the historical gold standard, two-dimensional (2D) ultrasonography offers advantages for on-site monitoring, including a wider field of view and reduced dependence on noise-free environments. This systematic review evaluates 2D ultrasonography protocols used in decompression research since the 2015 International Meeting on Ultrasound for Diving Research, identifying methodological similarities, differences, and adherence to consensus recommendations.
Methods: A search of PubMed and Scopus identified studies using 2D ultrasound to detect VGE in divers. Inclusion criteria were: (1) use of 2D ultrasound, (2) detection of VGE or monitoring of decompression stress, (3) inclusion of a diver cohort, and (4) publication after 2015. Data extraction focused on VGE scoring systems, ultrasound hardware, measurement protocols, and operator experience. Risk of bias was assessed using ROBINS-I-V2, and compliance with the 2015 consensus recommendations was evaluated.
Results: Twenty studies were included. The Eftedal-Brubakk scale was most commonly used (n = 15), with cardiac ultrasound as the primary imaging modality; one study assessed a peripheral vessel. Common shortcomings included post-dive measurements lasting less than two hours, underreporting of operator experience and hardware specifications, limited individual-level data, and inappropriate use of parametric statistics for ordinal bubble grade data. No study fully complied with all consensus recommendations.
Conclusions: This review demonstrates that, although two-dimensional ultrasound is widely used for post-dive VGE assessment, methodological heterogeneity with multiple shortcomings remain. Furthermore, nearly all studies restricted imaging to the heart, thus leaving peripheral vessel assessment largely unexplored.

Introduction

Humans entering aquatic environments undergo physiological changes. Aside from the specific cardiovascular and pulmonary adaptations, profound changes occur when a self-contained underwater breathing apparatus (scuba) is used. Descending under the water surface during scuba diving exposes the body to progressively higher ambient pressure as depth increases, causing inert gases to dissolve and accumulate within tissues. The speed of this accumulation depends not only on tissue perfusion and gas solubility but also on the surrounding pressure. When ascending, the ambient pressure is reduced. If the pressure of dissolved gas in tissue exceeds ambient pressure the gas may nucleate into bubbles.[1] Risk factors for bubble formation include longer and deeper dives (with more gas accumulation) and rapid ascents.[1]

Decompression sickness (DCS) may occur when these bubbles form in tissues or blood, causing ischaemic, inflammatory, or mechanical injury. DCS is highly variable in nature and severity and can affect multiple organs including the brain, spinal cord, inner ear, musculoskeletal system, cardiopulmonary system, and skin.[1] The quantity

of venous gas emboli in the bloodstream can be used as a surrogate marker for the amount of decompression stress experienced by the diver.[2]

Doppler ultrasound is historically considered the preferred method for quantifying inert gas bubbles in the venous blood, referred to as venous gas emboli (VGE). Doppler has been used to validate novel decompression tables.[3] Two-dimensional (2D) ultrasonography is a viable alternative tool for decompression stress monitoring. One advantage is that it allows for a more extensive field of view of the structure of interest.[3] Other advantages of two-dimensional ultrasound imaging include its potential for training a wide range of personnel and reduced dependence on a noise-free environment compared to Doppler analysis, enhancing its suitability for on-site monitoring.[3]

A 2011 review highlighted the lack of standardised ultrasound protocols for assessing decompression stress, limiting cross-study comparability.[4] In 2015, The International Meeting on Ultrasound for Diving Research was held. During this meeting, experts discussed the strengths and limitations of ultrasound monitoring and presented numerous recommendations for a standardised protocol including recommendations for measurement timing, provocative movements and data analysis.[5]

This systematic review aims to offer an overview of the 2D ultrasonography protocols used in decompression research published in peer reviewed journals and performed after 2015 consensus meeting. Methodological similarities and differences were assessed, and recommendations are provided for future studies.

Methods

This systematic review was conducted according to the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guideline.[6] On 16 September 2025, we searched PubMed and Scopus for studies using ultrasonography to detect VGE in divers. The search strategy combined three items: (1) 'diving', (2) 'decompression sickness', or 'decompression stress' and related terms and (3) 'ultrasound' and related terms using Boolean operators such as "*or*", and "*and*". The full search strings are depicted in Table 1. Only English-language articles published after 2015 were included.

INCLUSION AND EXCLUSION CRITERIA

Articles were initially screened by title and abstract, followed by a review of the full text. Inclusion criteria were: (1) use of a two-dimensional (2D) ultrasound diagnostic technique, (2) detection of venous gas emboli or monitoring of decompression stress, (3) inclusion of a cohort of divers and (4) publication after the publication of the recommendations of 2015 consensus meeting. Exclusion criteria were: (1) no description of measurement protocol provided, (2) not an original research article (e.g., a case report or a meta-analysis) and (3) no full text available. Case reports were excluded because this study focuses on evaluating the methodologies used in original research using ultrasound to assess VGE.

DATA COLLECTION

Data extraction was performed separately by two authors (BT and TW) using a standardised spreadsheet. Discrepancies

Table 1
Search query

PubMed:

("diving"[MeSH Terms] OR "diving"[All Fields] OR "Scuba"[All Fields] OR "Submersion"[All Fields])
AND
("decompression sickness"[MeSH Terms] OR "decompression"[All Fields] OR "gas embol*"[All Fields] OR "bubble*"[All Fields] OR "Venous Gas"[All Fields])
AND
("ultrasound"[Title/Abstract] OR "echo"[Title/Abstract] OR "echocardiography"[All Fields])

Scopus:

("diving" OR "scuba" OR "submersion")
AND
("decompression sickness" OR "decompression" OR "gas embol*" OR "bubble*" OR "venous gas")
AND
("ultrasound" OR "echo" OR "echocardiography")

Table 2
Summary of consensus recommendations following The International Meeting on Ultrasound for Diving Research in 2015[5]

#1	Clearly describe ultrasound operator training/experience, or ensure blinded independent review.
#2	Use the original or an expanded Eftedal-Brubakk scale for 2D imaging.
#3	Employ repeated-measures designs with participants as their own controls.
#4	Use the apical window as the standard for two-dimensional imaging.
#5	Standardise and report diver position during monitoring.
#6	Always include resting measurements with a standardized rest period; if provocation is used, describe and report both rest and provocation results.
#7	Record for at least 120 minutes post-decompression, or longer if bubbles persist.
#8	Make the first measurement ≤ 15 minutes post-dive; intervals ≤ 20 minutes during the first 120 minutes.
#9	Provide unpooled data when possible to enable reanalysis.
#10	Report individual-level outcomes regarding bubble grades (onset, maximum grade, time to max, medians, ranges, modes, zero grades) and raw data if feasible.
#11	Use non-parametric methods for bubble grade data.
#12	Acknowledge the limitations of bubble data.
#13	Record and preserve data for future review.
#14	Carefully consider comparability of different devices and methods; describe both in detail.

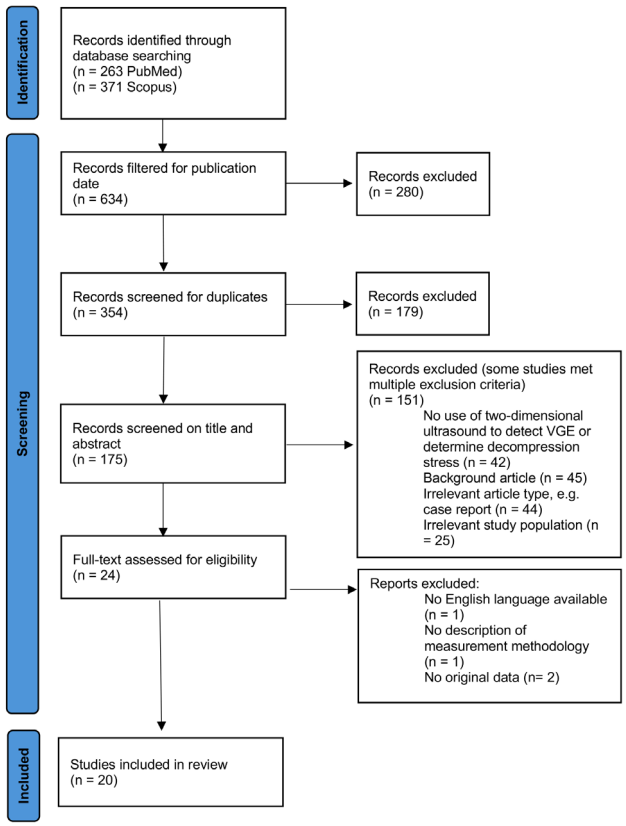
were discussed until consensus was reached. Extracted variables included: the VGE scoring system, ultrasound hardware description, number of dives and divers in the study, and study objectives, measurement locations and techniques, use of provocative movements and measurements at rest, time intervals between measurements, diver positioning, and description of the experience of the ultrasound performer.

QUALITY ASSESSMENT

Risk of bias in individual studies was assessed independently by BT and TW using the standardised ROBINS-I-V2 tool,[7] an updated version of ROBINS-I.[8] Discrepancies were resolved by discussion until consensus was reached.

Further quality assessment was performed by evaluating the studies against the recommendations of the 2015 consensus,[5] summarised in Table 2. For each recommendation a study was scored either 'Yes' if it complied with the recommendation, 'No' if it did not, and 'Partial' if it did not fully comply. For example, 'Partial' was assigned if the ultrasound technique was described as "cardiac" without specifying the apical view. Criterion 9 was scored 'Yes' if bubble score categories were reported separately; criterion 10 if individual bubble scores were presented; and criterion 11 'Partial' if both parametric and non-parametric analyses were used. Criterion 13 was not assessable. Criterion 14 was scored as 'Yes' if the study contained a clear description of utilised ultrasound hardware.

Figure 1
PRISMA flowchart



Results

The search yielded 263 PubMed and 371 Scopus records. After screening for publications before the International Meeting on Ultrasound for Diving Research 354 articles remained. Of these, 179 were duplicates and were excluded. Of the remaining unique 175 articles, 151 articles were excluded based on title and abstract. Of the remaining 24 articles, one did not provide a description of ultrasound methodology used and used an existing database[9] and one was excluded due to unavailability of the English full text.[10] Two were excluded due to the absence of original data.[11,12] The 20 remaining articles were included (Figure 1).

STUDY CHARACTERISTICS AND RISK OF BIAS

A summary of the extracted data is provided in Table 3. Study populations ranged from small, controlled cohorts of 10–20 dives[13–16] to larger multi-dive studies.[11,12,17,18] Study goals were equally diverse (Table 4), including the comparison of inter-diver bubble formation and agreement between handheld Doppler and cardiac ultrasound.

Among the studies, using the criteria of the ROBINS-I-V2 tool results in one study with a serious risk of bias and 19 with a moderate risk of bias. A complete overview of the risk of bias is depicted in Table 3. *Appendix A provides details on the scoring method.

SYNTHESIS OF METHODOLOGIES

Most studies used the Eftedal-Brubakk (EB) scale or its modifications for quantifying VGE;[15,17–25] others used VGE counting.[26–29] Two studies combined multiple scoring systems, using both VGE counting and the (modified) EB scale.[26] Two studies exclusively used VGE counting.[30,31]

Ultrasound hardware ranged from high-end scanners such as the General Electric (GE) Vivid-Q (GE Healthcare, Chicago, IL, USA)[16,20,22] and GE Logic E (GE Healthcare, Chicago, IL, USA),[17] to portable systems, including the Vivid-I (GE Healthcare, Chicago, IL, USA)[27] and Butterfly iQ.[25] Three studies did not specify the hardware used.[11,14,19,32] Probe frequencies and types were inconsistently reported.

Studies used different descriptions for the cardiac views they obtained. However, the descriptions of the majority of the studies corresponded to what is usually called the apical four-chamber view,[11–13,15,16,18,20,21,23–26,28,30–32] Two studies described extending the view to apical or multi-chamber views including the heart base, inferior vena cava, and subcostal regions.[16,23] The common theme in these positions was that all studies except one exclusively imaged the heart and did not assess (peripheral) vessels.

Half the studies measured VGE only at resting conditions.[12,15,17–19,23–27,29] Others also after limb flexion or extension,[11,16,20,21,28,30–32] after a Valsalva manoeuvre[14] or after two coughs.[22] One study had divers perform an exercise on an ergometer specifically tailored to their study goal and took ultrasound measurements prior to, during and after this exercise.[13]

Post-dive measurement timing varied. Five studies measured only once.[14,17,22–24] The fastest measurement performed was repeated as 'immediately' post-dive[15] and the latest was at 126 minutes post-dive.[29] Most studies followed specific time-intervals, while two performed scans if bubbles were present.[15,25]

Divers were most commonly positioned left lateral decubitus (or recumbent or supine lateral) position.[11,12,14,15,18–21,25,31,32] Six used the supine position.[13,16,17,22–24] Five studies did not specify the diver's positioning.[26–30]

Operator experience was rarely reported.[11,15–17,20,23,25,27–32] Some studies describe their operators as trained or certified sonographers,[13,14,24] experienced operator,[19] experienced cardiologists,[22] physicians[18,21] or experienced researcher.[26]

COMPARISON TO RECOMMENDATIONS

Compliance with the 2015 consensus recommendations is shown in Table 5. No study met all of the 14 outlined criteria; Hjell et al. complied with the largest number (n = 11).[21] It should be noted that the items included in the consensus guideline do not contribute equally to data quality. In particular, recommendations related to data collection are crucial for data quality. All studies acknowledged the limitations of using bubble data for analysis. All but one study included resting measurements and, if applicable, described the performed provocation method. Use of the EB scale or modified EB scale was common (n = 15), and repeated measures designs with participants serving as their own controls were implemented in 12 studies. The apical window was used as the standard for two-dimensional imaging in 18 studies, and diver positions were standardised and reported in 17 studies. Non-pooled bubble grading data were reported in 15 studies, and non-parametric statistical methods were appropriately applied in 12 publications. Notably, studies using VGE counting do not necessarily need to use non-parametric analyses.

As stated before, no study clearly reported the training or experience of the ultrasound operator. Other recommendations that few studies met include prolonged post-dive recording of at least 120 minutes (n = 6), reporting of individual-level bubble outcomes (n = 2), and performing the first measurement within 15 minutes post-dive and utilising intervals of 20 minutes or shorter during the first 120 minutes (n = 2).

Discussion

To our knowledge, this study represents the first systematic review of research using 2D ultrasound to assess VGE in divers since the 2015 consensus. Twenty studies met the inclusion criteria, encompassing a broad range of study sizes and objectives.

KEY FINDINGS

The key findings of this study are: (1) No study complied with all 14 recommendations of the 2015 consensus. (2) All but one study exclusively obtained cardiac imaging.

***Footnote:** Appendix A is available online on our website: https://www.dhmjournal.com/index.php/journals?id=438

Table 3

Characteristics of included studies ordered by publication date; DCIEM – Defence and Civil Institute of Environmental Medicine; DCS – decompression sickness; EB – Eftedal-Brubakk; GE – General Electric; Ref – reference number; TTE – transthoracic echocardiography, VGE – venous gas emboli

Year	Ref	Scoring system	Ultrasound hardware	Included study population	Locations	Movement	Time intervals	Position of diver	Ultrasound operator	Risk of bias
2015	15	EB	Zonare Z1	24 dives, 24 divers	Right ventricular foreshortened apical view	No	Immediately post-dive, every 15 minutes until 75 minutes or two clear scans	Supine left lateral	Not specified	Moderate
	13	Modified EB	Vivid Q, and contrast TTE (unspecified)	20 dives, 20 divers	Apical four-chamber, temporal window median cerebral artery and unspecified for TTE	Yes, prior to and during supine ergometer exercise until exhaustion	15, 30 and 45 minutes post-dive. Then every 2 minutes during exercise and after 10 minutes of recovery	Supine	Trained ultrasonographer	Moderate
2016	14	Modified EB and VGE counting	Not specified	11 dives, 11 divers	Apical four-chamber	Valsalva maneuver	60 minutes post-dive	Recumbent position	Certified sonographer	Moderate
	16	Modified EB scale	GE Vivid-Q	16 dives, 16 divers	Apical four-chamber view	Yes, at rest and after arm and leg contractions	30, 60 and 90 minutes post-dive	Supine	Not specified	Moderate
	31	VGE counting	Vivid 7	36 dives, 6 divers	Modified four-chamber view	Yes, at rest and after two knee bends with flexion	Before dive and 30 and 90 minutes post-dive	Left lateral supine position	Not specified	Moderate
2017	19	EB	Not specified	38 dives, 10 divers	Precordial	Yes, at rest and some after flexing both legs while supine or knee bends when standing upright	5, 10, 15, 20, 25, 30, 45, 60, 75, 90, 105, 120 minutes post-dive	Left lateral decubitus position	One experienced operator	Moderate
	27	VGE count	Vivid-i	Between 36 and 54 dives, 9 divers	Transthoracic echocardiography	No	30, 90 minutes post-dive	Not specified	Not specified	Moderate
	23	EB	Not specified	74 dives, 51 divers	Apical four-chamber, heart base short-axis, inferior vena cava and right atrium subcostal	No	30 minutes post-dive	Supine	Not specified	Moderate

Table 3 continued.

2018	29	VGE counting	Mindray M9	17 dives, 14 divers	Modified four-chamber view B-mode echocardiography recording	No	21, 56, 91, 126 minutes post-dive	Not specified	Not specified	Moderate
2019	22	Modified EB	Vivid Q	30 dives, 10 divers	Cardiac	At rest and after two coughs	Before dive and 30 minutes post-dive	Supine	Two experienced cardiologists	Moderate
2020	32	Modified EB	Not specified	96 dives	Apical long-axis four-chamber view	Yes, at rest and after flexion of elbow and knee	20, 40, 60, 80, 100, 120 minutes post-dive	Left decubital position	Not specified	Moderate
2021	24	EB	GE Logic e	342 dives, 41 divers	Subcostal and apical	No	40 minutes post-dive.	Supine position	Not specified	Moderate
	20	EB	GE Vivid-Q	30 dives, 10 divers	Apical four-chamber view	Yes, at rest and after limb flection.	Pre-dive once. 20, 40, 60, 80, 100, 120 minutes post-dive	Left lateral decubitus	One operator	Moderate
2022	18	EB	UltraSound EDGE II	152 dives, 48 divers	Apical four-chamber	No	Within 5–15 minutes, then every 15 minutes post-dive for 4–9 measurements	Left lateral decubitus	Two physicians	Moderate
	28	VGE count	Sonosite M-Turbo	16 dives, 8 divers	Apical four-chamber view	Yes, at rest and after two knee bends	30, 60 minutes post-dive	Not specified	Not specified	Moderate
	26	VGE counting and EB	Sonosite M-Turbo	30 dives, 10 divers	Four-chamber view	No	30, 60 minutes post-dive	Not specified	One experienced researcher	Moderate
	25	EB	Butterfly iQ and Vivid q	75 divers	Apical four-chamber view	No	Before and 20, 40, 60 minutes post-dive. Additional at 20-minute intervals if bubbles persisted	Lying on left side	Not specified	Moderate
2023	21	EB	EDGE II	154 dives, 47 divers	Apical four-chamber view	Yes, at rest and after knee extension while laying	Within 5–15 minutes, 30, 45, 60, 75, 90, 105, 120 minutes post-dive	Left lateral decubitus position	Two physicians	Moderate
2024	30	VGE counting	M9 Mindray and Sonosite M-Turbo	10 divers, 6-8 dives each	Apical four chamber view	Yes, at rest and after two knee bends	15, 30, 45, 60, 120 min post-dive.	Not specified	Two of the authors, experience not specified	Serious
2025	24	EB	GE Logic e	342 dives, 41 divers	Echocardiography using subcostal and apical approach	No	30 minutes post-dive	Lying supine position	Two experienced ultrasonographers with an international ultrasound diploma	Moderate

Table 4
The study goals of the included studies ordered by publication date; AGE – arterial gas embolism; DCS – decompression sickness; Ref – reference number; VGE – venous gas emboli

Year	Ref	Study goal
2015	15	Comparing bubble formation between divers with- and without history of DCS.
	13	Assessing post-dive AGE formation.
2016	14	Assessing the effect of in-water recompression on VGE.
	16	Assessing the impact of predive exercise on microparticles, endothelial function and VGE throughout multiple dives.
	31	Comparing the effect on post-dive VGE of different preconditioning methods: oxygenation, whole-body vibration and vibration associated with oxygenation. Assessment of how multiple days of deep diving influence bubble production, oxidative and inflammatory markers.
2017	19	Observing DCS and VGE following standardised diving protocol.
	27	Assessing the effect of different preconditioning interventions on VGE formation.
	23	Comparing the effect on inflammatory responses of two decompression procedures.
2018	29	Assessing variability in VGE counting in a standardised diving profile.
2019	22	Assessment of oxygen enriched gases on bubble formation and endothelial function.
2020	32	Testing air decompression schedules.
2021	24	Finding a relationship between dive profile and individual parameters and the severity of post-dive bubbles.
	20	Assessing decompression stress following a single dive at altitude.
2022	18	Assessing the agreement between handheld self-positioning Doppler and cardiac ultrasonography.
	28	Assessing whether pre-dive exercise reduces VGE.
	26	Monitoring post-dive physiological status and VGE dynamics.
	25	Comparison of handheld ultrasound devices to standardised ultrasound device.
2023	21	Safety evaluation of new diving table.
2024	30	Assessment of how multiple days of deep diving influence bubble production, oxidative and inflammatory markers.
2025	24	Quantifying inter- and intra-diver gas bubble variability.

(3) Ultrasound operator experience, detailed hardware descriptions and individual diver data were consistently underreported. (4) There was considerable methodological variability in measurement timing and number of total measurements per individual. (5) Multiple studies utilised parametric analyses for ordinal bubble grade data.

COMPARISON OF STUDY METHODOLOGIES

The EB scale and its modifications were the most commonly used grading systems, and cardiac ultrasound was the primary imaging modality; only one study included additional measurements of the subclavian vein. These findings are consistent with the 2015 consensus recommendations.[5] Even though the descriptions used by the authors differed, it is safe to assume that almost all used the apical four-chamber view, which is considered the preferred method for assessing VGE in the right heart.[5] No study extensively assessed (peripheral) vessels.

Measurement timing varied widely. It is commonly agreed that VGE appear in different patterns and differ over time due to a multitude of factors, including varying dive profiles and possible variability in (patho-) physiological factors within the diver.[12] Therefore, multiple measurements should be made to gain an accurate indication of the peak bubble load, as a surrogate endpoint for decompression stress.[3,33] The different timings used by the studies diminish their comparability. A reason for the limited number of measurements performed in multiple studies may be logistical or practical constraints. The recommendation is to perform measurements within 15 minutes and up to 120 minutes post-dive, with ≤ 20 minutes intervals. However, these timings can be difficult to achieve in real life, as divers may have other obligations to fulfil, including safety measures. Furthermore, the time to peak VGE varies across diving profiles. Accordingly, study protocols should be designed to ensure that peak VGE is not missed and that temporal resolution is sufficient across the entire post-dive window relevant to the exposure.

The majority of studies conducted VGE measurements at rest, with a subset incorporating assessments following provocative manoeuvres. However, these manoeuvres were not standardised across studies, ranging from lower-limb exercises (e.g., air squats) to upper-limb flexion. It is plausible that such heterogeneity in movement protocols may influence both the anatomical sites of bubble dislodgment and the overall quantity of detectable VGE.

Not all studies clearly or consistently reported differences in bubble load before and after provocation. This limitation does not necessarily detract from the validity of their findings, as bubbles dislodged by provocative manoeuvres are still considered indicative of decompression stress, and the manoeuvres themselves do not increase physiological stress. Currently, there is no scientific consensus regarding the exact interpretation of post-movement VGE grading using the EB

Table 5

Compliance of included studies with the guidelines provided by the 2015 consensus;[5] F – full compliance with the corresponding guideline item; P – partial compliance; n/a – indicates that compliance could not be assessed; empty cells indicate non-compliance

Study / Guideline Recommendation (see Table 2)	Gawthrope et al.[15]	Barak et al.[13]	Dituri et al.[14]	Madden et al.[16]	Balestra et al.[31]	Blogg et al.[19]	Germonpré et al.[27]	Spisni et al.[23]	Papadopoulou et al.[29]	Šegrt Ribičić et al.[22]	Andrew et al.[32]	Fichtner et al.[24]	Hess et al.[20]	Plogmark et al.[18]	Lambrechts et al.[28]	Balestra et al.[26]	Karimpour et al.[25]	Hjelte et al.[21]	Balestra et al.[30]	Fichtner et al.[24]	Studies in agreement with guideline
#1		P	P			P				P								P		P	0
#2	F	F	F	F		F		F		F	F	F	F	F		F	F	F		F	15
#3						F	F		P	F		F	F	F	F	F	P	F	F	F	11
#4	F	F	P	F	F	P	P	F	F	P	F	F	F	F	F	F	F	F	F	F	16
#5	F	F	F	F	F	F		F		F	F	F	F	F			F	F		F	15
#6	F	F		F	F	F	F	F	F	F	F	F	F	F	F	F	F	F	F	F	19
#7						F			F		F		F	P				F	F		6
#8	P	P		P		F								P				F			2
#9	F	F		F		F		F		F	F	F	F	F			F	F		F	13
#10		F											F					P			2
#11	F	F		F		F			F	F	F			F		P	F	F		F	11
#12	F	F	F	F	F	F	F		F	F	F	F	F	F	F	F	F	F	F	F	20
#13	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
#14	F	F		F	F		F		F	F		F	F	F	F	F	F	F	F	F	16
Items in agreement	8	9	3	8	5	9	4	6	6	8	8	8	10	9	5	6	8	11	6	9	

scale.[11] Establishing standardised criteria for this assessment is critical, as bubble loads can differ substantially between pre- and post-movement measurements.

Several additional methodological inconsistencies were noted in the analytic approaches. First, bubble data were not consistently presented in a non-pooled format. Moreover, only two studies reported individual-level bubble grading outcomes. Finally, the use of parametric statistical tests in the analysis of ordinal bubble grade scales was inappropriate, as non-parametric methods would have been more suitable.

The absence of detailed reporting on ultrasound operator experience further limits the interpretability of the included studies. Proficiency in cardiac ultrasound within the context of diving medicine is particularly important, as the real-time detection and quantification of intravascular bubbles requires specific practical experience and insufficient expertise could result in underestimation of the actual number of VGE.

Ultrasound hardware was not consistently reported across studies, and where described, a wide range of devices were employed. To date, no consensus or large-scale evaluation has been established regarding the comparability of different ultrasound systems for VGE assessment, and existing evidence suggests that levels of correlation may vary across ultrasound hardware.[25] It is reasonable to assume that high-end ultrasound platforms provide superior image quality; however, the adequacy of smaller, portable devices for accurately assessing decompression stress remains uncertain. This question is of particular operational relevance for the future application of two-dimensional ultrasound in austere environments.

STRENGTHS AND LIMITATIONS

To our knowledge, this study is the first to systematically evaluate compliance with the 2015 ultrasound consensus recommendations in published studies. This is valuable knowledge for both reviewing and updating the recommendations and for helping authors of future studies design and publish relevant data in this area of research.

Several limitations of this review should be acknowledged. First, heterogeneity in reporting of the ultrasound protocols and diving populations used limited our ability to compare the studies and draw overarching conclusions. For example, due to the underreporting of the experience of ultrasound performers, it is difficult to draw conclusions regarding the quality and applicability of the images obtained in all studies using different protocols. This does not necessarily mean that the work is flawed, but that it can be difficult for readers to assess the presented data. A potential approach for the future is for editorial boards and reviewers to ensure that submitted manuscripts adhere to the consensus statements, encouraging authors to provide the required information or to justify deviations.

Second, the included studies are predominantly observational and non-randomised in nature, conducted in controlled experimental settings. This reduces the generalisability of our findings to operational, resource-constrained diving contexts. While we acknowledge that many studies in our field have 'convenience inclusions' or 'opportunistic data collection', it would benefit if future studies clearly evaluate the 'noise' that in vivo experiments introduce and discuss this in their manuscripts when applicable. For example, imaging conducted in waterside or field environments may differ in quality from imaging performed under controlled laboratory conditions. Accordingly, if ultrasound imaging is intended for operational use, validation studies should be designed to closely replicate the environments in which the technology is ultimately expected to be applied.

Third, there has possibly been a component of publication bias, as studies showing low or no VGE may be underreported, which potentially limits our understanding of ultrasound methodologies employed for decompression stress assessment. Furthermore, most of the included studies were assessed as having a moderate risk of bias using the ROBINS-I-V2 instrument, which means that the strength of conclusions should therefore be carefully considered. While this instrument is designed for assessing interventional studies, we found that it worked quite well in this collection of studies which also included observational studies. Authors of future publications could consider reporting items that help readers to assess the risk of bias by addressing the items in this instrument.

Finally, this systematic review solely includes peer-reviewed scientific literature. It therefore does not include military or other government technical reports that are not published in scientific journals. In the field of diving medicine and research, substantial experimental research has been conducted by military and government agencies outside traditional academic journals, its inclusion would provide a more complete overview of currently used ultrasound protocols.

FUTURE RESEARCH ENDEAVOURS

Based on our findings, we recommend that future research (1) as per the 2015 consensus, adopt standardised post-dive measurement timing schedules relevant for the corresponding diving profile, (2) implement standardised and clearly reported provocation manoeuvres, (3) improve data reporting by including individual-level bubble grades and applying appropriate statistical methods, and (4), provide detailed documentation of both operator expertise and ultrasound hardware specifications.

Conclusions

This systematic review demonstrates that, although two-dimensional ultrasound is widely used for post-dive VGE assessment, methodological heterogeneity remains. The

most common shortcomings included measuring for less than two hours post-dive, underreporting operator expertise and ultrasound hardware specifications, limited availability of individual-level data and inappropriate statistical treatment of ordinal outcomes. Furthermore, nearly all studies restricted imaging to the heart, thus leaving peripheral vessel assessment largely unexplored.

References

1 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.

2 Doolette DJ. Venous gas emboli detected by two-dimensional echocardiography are an imperfect surrogate endpoint for decompression sickness. Diving Hyperb Med. 2016;46:4–10. PMID: 27044455. [cited 2025 Oct 20]. Available from: https://dhmjournal.com/images/IndividArticles/46March/Doolette_dhm.46.1.4-10.pdf.

3 Le DQ, Dayton PA, Tillmans F, Freiberger JJ, Moon RE, Denoble P, et al. Ultrasound in decompression research: fundamentals, considerations, and future technologies. Undersea Hyperb Med. 2021;48(1):59–72. doi: 10.22462/01.03.2021.8. PMID: 33648035.

4 Blogg SL, Gennser M. The need for optimisation of post-dive ultrasound monitoring to properly evaluate the evolution of venous gas emboli. Diving Hyperb Med. 2011;41:139–46. PMID: 21948499. [cited 2025 Oct 20]. Available from: https://dhmjournal.com/images/IndividArticles/41Sept/Blogg_dhm.41.3.139-146.pdf.

5 Møllerløkken A, Blogg SL, Doolette DJ, Nishi RY, Pollock NW. Consensus guidelines for the use of ultrasound for diving research. Diving Hyperb Med. 2016;46:26–32. PMID: 27044459. [cited 2025 Oct 20]. Available from: https://dhmjournal.com/images/IndividArticles/46March/Mollerlokken_dhm.46.1.26-32.pdf.

6 Page MJ, Moher D, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. PRISMA 2020 explanation and elaboration: updated guidance and exemplars for reporting systematic reviews. BMJ. 2021;372:n160. doi: 10.1136/bmj.n160. PMID: 33781993. PMCID: PMC8005925.

7 ROBINS-I V2 tool. The Cochrane Bias Methods Group and the Cochrane Non-Randomised Studies Methods Group; 2024. [updated 22 November 2024 25-09-2025]; Available from: https://sites.google.com/site/riskofbiastool/welcome/robins-i-v2.

8 Sterne JA, Hernán MA, Reeves BC, Savović J, Berkman ND, Viswanathan M, et al. ROBINS-I: a tool for assessing risk of bias in non-randomised studies of interventions. BMJ. 2016;355:i4919. doi: 10.1136/bmj.i4919. PMID: 27733354. PMCID: PMC5062054.

9 Liou K, Wolfers D, Turner R, Bennett M, Allan R, Jepson N, et al. Patent foramen ovale influences the presentation of decompression illness in SCUBA divers. Heart Lung Circ. 2015;24:26–31. doi: 10.1016/j.hlc.2014.07.057. PMID: 25130890.

10 Klenkov IR, Palenyi SR, Bychkov SA. Decompression venous gas formation intensity scale. Medico-Biological and Socio-Psychological Issues of Safety in Emergency Situations. 2024(4):95–101. doi: 10.25016/2541-7487-2024-0-4-95-101.

11 Currens JB, Doolette DJ, Murphy FG. Venous gas emboli (VGE) in 2-D echocardiographic images following movement: grading and association with cumulative incidence of decompression sickness. Diving Hyperb Med. 2025;55:44–50. doi: 10.28920/dhm55.1.44-50. PMID: 40090025. PMCID: PMC12263276.

12 Doolette DJ, Murphy FG. Within-diver variability in venous gas emboli (VGE) following repeated dives. Diving Hyperb Med. 2023;53:333–9. doi: 10.28920/dhm53.4.333-339. PMID: 38091593. PMCID: PMC10944664.

13 Barak OF, Madden D, Lovering AT, Lambrechts K, Ljubkovic M, Dujic Z. Very few exercise-induced arterialized gas bubbles reach the cerebral vasculature. Med Sci Sports Exerc. 2015;47:1798–805. doi: 10.1249/mss.0000000000000625. PMID: 25628180.

14 Dituri J, Sadler R, Siddiqi F, Sadler C, Javeed N, Annis H, et al. Echocardiographic evaluation of intracardiac venous gas emboli following in-water recompression. Undersea Hyperb Med. 2016;43:103–12. PMID: 27265987.

15 Gawthrope IC, Summers M, Macey DJ, Playford DA. An observation of venous gas emboli in divers and susceptibility to decompression sickness. Diving Hyperb Med. 2015;45:25–9. PMID: 25964035. [cited 2025 Oct 20]. Available from: https://dhmjournal.com/images/IndividArticles/45March/Gawthrope_dhm.45.25-29.pdf.

16 Madden D, Barak O, Thom SR, Yang M, Bhopale VM, Ljubkovic M, et al. The impact of predive exercise on repetitive SCUBA diving. Clin Physiol Funct Imaging. 2016;36:197–205. doi: 10.1111/cpf.12213. PMID: 25371042.

17 Fichtner A, Brunner B, Pohl T, Grab T, Fieback T, Koch T. Estimating inert gas bubbling from simple SCUBA diving parameters. Int J Sports Med. 2021;42:840–6. doi: 10.1055/a-1342-8030. PMID: 33506443. PMCID: PMC8328538.

18 Plogmark O, Hjelte C, Ekström M, Frånberg O. Agreement between ultrasonic bubble grades using a handheld self-positioning Doppler product and 2D cardiac ultrasound. Diving Hyperb Med. 2022;52:281–5. doi: 10.28920/dhm52.4.281-285. PMID: 36525686. PMCID: PMC10017197.

19 Blogg SL, Møllerløkken A, Gennser M. Observed decompression sickness and venous bubbles following 18-msw dive profiles using RN Table 11. Undersea Hyperb Med. 2017;44:211–9. doi: 10.22462/5.6.2017.2. PMID: 28779578.

20 Hess HW, Wheelock CE, St James E, Stooks JL, Clemency BM, Hostler D. Variability in venous gas emboli following the same dive at 3,658 meters. Undersea Hyperb Med. 2021;48:119–26. doi: 10.22462/03.04.2021.2. PMID: 33975402.

21 Hjelte C, Plogmark O, Silvanius M, Ekström M, Frånberg O. Risk assessment of SWEN21 a suggested new dive table for the Swedish armed forces: bubble grades by ultrasonography. Diving Hyperb Med. 2023;53:299–305. doi: 10.28920/dhm53.4.299-305. PMID: 38091588. PMCID: PMC10944666.

22 Šegrt Ribičić I, Valić M, Božić J, Obad A, Glavaš D, Glavičić I, et al. Influence of oxygen enriched gases during decompression on bubble formation and endothelial function in self-contained underwater breathing apparatus diving: a randomized controlled study. Croat Med J. 2019;60:265–72. doi: 10.3325/cmj.2019.60.265. PMID: 31187955. PMCID: PMC6563167.

23 Spisni E, Marabotti C, De Fazio L, Valerii MC, Cavazza E, Brambilla S, et al. A comparative evaluation of two decompression procedures for technical diving using inflammatory responses: compartmental versus ratio deco. Diving Hyperb Med. 2017;47:9–16. doi: 10.28920/dhm47.1.9-16. PMID: 28357819. PMCID: PMC6147226.

24 Fichtner A, Hannesen B, Stein F, Schrofner-Brunner B, Pohl T, Grab T, et al. Predicting post-dive inert gas bubble grades in non-decompression scuba diving with air: simplified model for enhanced diver safety. Sports Med Open. 2025;11(1):29. doi: 10.1186/s40798-025-00832-x. PMID: 40153151. PMCID: PMC11953488.
25 Karimpour K, Brenner RJ, Dong GZ, Cleve J, Martina S, Harris C, et al. Comparison of newer hand-held ultrasound devices for post-dive venous gas emboli quantification to standard echocardiography. Front Physiol. 2022;13:907651. doi: 10.3389/fphys.2022.907651. PMID: 35755430. PMCID: PMC9222333.
26 Balestra C, Guerrero F, Theunissen S, Germonpré P, Lafère P. Physiology of repeated mixed gas 100-m wreck dives using a closed-circuit rebreather: a field bubble study. Eur J Appl Physiol. 2022;122:515–22. doi: 10.1007/s00421-021-04856-5. PMID: 34839432. PMCID: PMC8627581.
27 Germonpré P, Balestra C. Preconditioning to reduce decompression stress in scuba divers. Aerosp Med Hum Perform. 2017;88:114–20. doi: 10.3357/amhp.4642.2017. PMID: 28095955.
28 Lambrechts K, Germonpré P, Vandenheede J, Delorme M, Lafère P, Balestra C. Mini trampoline, a new and promising way of SCUBA diving preconditioning to reduce vascular gas emboli? Int J Environ Res Public Health. 2022;19(9):5410. doi: 10.3390/ijerph19095410. PMID: 35564805. PMCID: PMC9105492.
29 Papadopoulou V, Germonpré P, Cosgrove D, Eckersley RJ, Dayton PA, Obeid G, et al. Variability in circulating gas emboli after a same scuba diving exposure. Eur J Appl Physiol. 2018;118:1255–64. doi: 10.1007/s00421-018-3854-7. PMID: 29616324.
30 Balestra C, Lévêque C, Mrakic-Sposta S, Vezzoli A, Wauthy P, Germonpré P, et al. Physiology of deep closed circuit rebreather mixed gas diving: vascular gas emboli and biological changes during a week-long liveaboard safari. Front Physiol. 2024;15:1395846. doi: 10.3389/fphys.2024.1395846. PMID: 38660539. PMCID: PMC11040087.
31 Balestra C, Theunissen S, Papadopoulou V, Le Mener C, Germonpré P, Guerrero F, et al. Pre-dive whole-body vibration better reduces decompression-induced vascular gas emboli than oxygenation or a combination of both. Front Physiol. 2016;7:586. doi: 10.3389/fphys.2016.00586. PMID: 27965591. PMCID: PMC5127795.
32 Andrew BT, Doolette DJ. Manned validation of a US Navy Diving Manual, Revision 7, VVal-79 schedule for short bottom time, deep air decompression diving. Diving Hyperb Med. 2020;50:43–8. doi: 10.28920/dhm50.1.43-48. PMID: 32187617. PMCID: PMC7276270.
33 McCune EP, Le DQ, Lindholm P, Nightingale KR, Dayton PA, Papadopoulou V. Perspective on ultrasound bioeffects and possible implications for continuous post-dive monitoring safety. Diving Hyperb Med. 2022;52:136–48. doi: 10.28920/dhm52.2.136-148. PMID: 35732286. PMCID: PMC9522608.

Disclaimer

The opinions or assertions contained herein are the private views of the authors and are not to be construed as official or reflecting the views of the Department of Defense or Dutch government. The authors are employees of the Dutch government

Conflicts of interest and funding: nil

Submitted: 30 October 2025
Accepted after revision: 20 June 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

Short communication

Evaluating the FitMáx questionnaire for fitness-to-dive assessments in a military population

Pieter D Dijk[1], Paul Clarijs[1], Thijs T Wingelaar[1]

[1] *Royal Netherlands Navy Diving and Submarine Medical Center, Rijkszee en Marinehaven, Den Helder, the Netherlands*

Corresponding author: *Dr Thijs Wingelaar, Royal Netherlands Navy Diving and Submarine Medical Center, Rijkszee en Marinehaven, 1780 CA Den Helder, the Netherlands*
tt.wingelaar@mindef.nl

Keywords
Cardiorespiratory fitness; Divers; Self-reported fitness; Recompression personnel; $\dot{V}O_2$max; $\dot{V}O_2$peak

Abstract

(Dijk PD, Clarijs P, Wingelaar TT. Evaluating the FitMáx questionnaire for fitness-to-dive assessments in a military population. Diving and Hyperbaric Medicine. 2026 30 September;56(3):361–364. doi: 10.28920/dhm56.3.361-364. PMID: 42743577.)
This study evaluated the validity of the FitMáx questionnaire for estimating $\dot{V}O_2$peak in 157 Dutch Navy divers and recompression personnel, a population characterised by high physical fitness and occupational demands. While self-reported questionnaires offer a logistically feasible alternative to resource-intensive direct testing, their accuracy in fitness-to-dive assessments remains unclear. Participants completed the FitMáx questionnaire prior to cycle ergometry with direct $\dot{V}O_2$peak measurement. Results showed a moderate correlation (r = 0.631, $P < 0.001$) between mean estimated $\dot{V}O_2$max (43.7, SD 5.34 mL·kg^{-1}·min^{-1}) and mean measured $\dot{V}O_2$peak (45.4, SD 6.52 mL·kg^{-1}·min^{-1}), with the questionnaire systematically underestimating fitness by 1.8 mL·kg^{-1}·min^{-1}, with a 95% confidence interval of agreement limit of ± 10.9 mL·kg^{-1}·min^{-1}, and a moderate intraclass correlation coefficient of 0.748 ($P < 0.001$). False-positive and false-negative rates were 8.9% and 10.8%, respectively, raising concerns about reliability for pass/fail decisions. Participants reported difficulties answering questions unrelated to their routine activities, potentially contributing to the observed variability. Given these limitations, including the homogeneous, highly fit sample and the inability to differentiate between subgroups, the Royal Netherlands Navy Diving Medical Center has not adopted the FitMáx for fitness-to-dive evaluations. While questionnaires may streamline large-scale assessments, direct exercise testing remains valuable for precise, individualised risk stratification in occupational diving.

Introduction

Diving imposes significant demands on the cardiopulmonary system, necessitating physiological adaptations to ensure safety and performance.[1] To our knowledge, there is no conclusive evidence establishing the minimum exercise tolerance required for safe diving. However, a general consensus suggests that higher exercise tolerance is associated with safer diving practices. Though no universal fitness standard exists, occupational fitness-to-dive assessments typically evaluate exercise tolerance, often measuring maximal aerobic fitness ($\dot{V}O_2$peak).[2]

$\dot{V}O_2$peak, commonly expressed in mL·kg^{-1}·min^{-1}, can be assessed through various methods.[3] These methods are broadly categorised into direct and indirect approaches. Direct measurements involve a more complex setup, where participants wear a mask to measure inhaled and exhaled air, enabling precise calculation of $\dot{V}O_2$peak. Indirect methods estimate $\dot{V}O_2$peak based on physical performance, such as the distance covered in a 12-minute run or the maximum resistance achieved on a cycle ergometer. While indirect methods are slightly less accurate than direct measurements, both approaches require specialised equipment and resources. Furthermore, both testing procedures impose 'domestic overhead' on participants, such as the need to change clothing or travel to a designated facility. These requirements may introduce operational challenges, particularly when implementing such assessments at scale.

An alternative approach to assessing $\dot{V}O_2$peak is the use of self-reported questionnaires, which significantly reduce the logistical and resource demands of traditional testing. However, many existing questionnaires demonstrate only moderate to poor correlations with directly or indirectly measured $\dot{V}O_2$peak.[4] The recently developed FitMáx questionnaire presents a promising solution, exhibiting a very high correlation (r = 0.94) between measured and estimated $\dot{V}O_2$peak, along with excellent test-retest reliability (intraclass correlation coefficient = 0.97) in an active adult population.[5,6]

If the FitMáx questionnaire proves valid and reliable in a diving population, it could substantially streamline occupational fitness-to-dive assessments by reducing the need for resource-intensive testing. We hypothesise that the

FitMáx questionnaire will demonstrate a strong correlation between estimated and measured $\dot{V}O_2$peak in this context.

Methods

This prospective study included all Navy divers and recompression personnel that underwent a fitness-to-dive assessment at the Royal Netherlands Navy Diving and Submarine Medical Center between 1 January 2025 and 30 June 2025. This study was approved by the Surgeon General of the Netherlands Armed Forces (ref: DOSCO2025013218). Even though in the Netherlands medical ethical review is not required for studies only including short questionnaires, the Declaration of Helsinki and European General Data Protection Regulation were adhered to.

Participation was entirely voluntary, with informed consent in writing, and had no effect on the medical assessment of the diver or career of the participant. Prior to cycle ergometry, which is a standard part of fitness-to-dive assessments in the Royal Netherlands Navy, subjects were asked to fill in a paper version of the FitMáx questionnaire, which asks about the sex, age, height and weight of the subject, as well as three questions regarding the estimated distance and speed the subject can walk/run, cycle and climb stairs. The questionnaire was collected by the researchers, and neither the subject's participation in the study nor the questionnaire results were disclosed to the physician conducting the medical assessment. Afterwards, the subject performed a cycle ergometry with a ramp protocol and direct measurement of the inhaled and exhaled breath using the Vyntus™ CPX metabolic cart and SentrySuite™ software package (Jaeger Medical, London ON, Canada). Data from the ergometry, including max heartrate, max resistance and peak oxygen consumption were recorded. From the answers of the FitMáx questionnaire, a $\dot{V}O_2$peak can be calculated through an algorithm, which was done after the fitness-to-dive assessment by an independent researcher. From here on, we refer to the score of the FitMáx questionnaire as $\dot{V}O_2$max, and use $\dot{V}O_2$peak for directly measured ergometry.

All data from the questionnaire, as well as parameters associated with the direct ergometry performed, were entered into a database. The difference between the estimated $\dot{V}O_2$max and measured $\dot{V}O_2$peak were tested using a paired samples *t*-test, and correlation was calculated using a Pearson's correlation coefficient. Additionally, Bland-Altman limits of agreement and intraclass correlation coefficients (ICC) were calculated. All statistical analyses were performed using SPSS version 31.0. Statistical significance was assumed at alpha < 0.05.

Results

In total 157 Navy divers and hyperbaric personnel were included, with the following baseline parameters: median age 34.0 years (interquartile range [IQR] 28.0–41.0), mean height 182.6 cm (standard deviation [SD] 8.3), mean weight 86.3 kg (SD 11.8), and 93% were male.

Cycle ergometry parameters included a median maximal heartrate of 180 beats per minute [IQR 173.3–186.0], median maximum resistance of 320 W [IQR 283–352], and median respiratory exchange ratio (RER) of 1.18 [IQR 1.14–1.25], all indicating that end of test criteria were met. Mean measured $\dot{V}O_2$peak was 45.4 $mL \cdot kg^{-1} \cdot min^{-1}$ (SD 6.52), which was statistically significantly higher than the FitMáx estimated $\dot{V}O_2$max of 43.7 $mL \cdot kg^{-1} \cdot min^{-1}$ (SD 5.34) ($t = 4.310$, $P < 0.001$). The correlation between the two variables was 0.631 ($P < 0.001$), i.e., $R^2 = 0.398$, as illustrated in Figure 1. The 5% trimmed mean difference between the two parameters was −1.8 (SD 5.2), with a range of −14.4 to 18.0. The Bland-Altman analysis (Figure 2) shows a 95% confidence interval of agreement limits of ± 10.9 $mL \cdot kg^{-1} \cdot min^{-1}$. A moderate to good degree of reliability was found between 157 measurements, with an average ICC of 0.748 with a 95% confidence interval from 0.636 to 0.823 ($F = 2.474$, $P < 0.001$)

In 34 subjects (21.7%) the $\dot{V}O_2$peak was lower than 40 $mL \cdot kg^{-1} \cdot min^{-1}$. The FitMáx questionnaire correctly estimated the VO_2peak to be lower than 40 $mL \cdot kg^{-1} \cdot min^{-1}$ in 58.8% of the cases, meaning that 14 cases (8.9% of the total population) the FitMáx would have incorrectly estimated sufficient exercise tolerance (false-negative, upper left quadrant of Figure 1). Conversely, in 17 subjects (10.8% of the total population) the estimated $\dot{V}O_2$max was lower than 40 $mL \cdot kg^{-1} \cdot min^{-1}$, while the measured $\dot{V}O_2$peak was higher than 40 (false-positive, lower right quadrant of Figure 2).

Discussion

The measured $\dot{V}O_2$max correlates moderately ($R^2 = 0.398$) to the FitMáx estimated $\dot{V}O_2$peak. The latter underestimated the fitness of the population by approximately 1.8 $mL \cdot kg^{-1} \cdot min^{-1}$, with a 95% confidence interval of agreement limits of ± 10.9 $mL \cdot kg^{-1} \cdot min^{-1}$, and a moderate to good reliability (ICC = 0.748).

Based on our sample, the correlation between estimated and measured $\dot{V}O_2$max is lower than earlier reported. Even though this difference is approximately 4% on a group level, the individual variation is considerable. Wide individual variation has been found in previous studies assessing cardiopulmonary exercise tolerance in military populations.[7] Some subjects reported that they found in hard to answer some questions of the FitMáx, for instance how many stairs they would be able to climb or at what speed they would be able to cycle continuously for one hour, if these activities were not part of their daily life. While this may also be the case for the population on which the questionnaire has been validated, this could be a possible explanation of the lower correlation and wider spread of our results.

Figure 1
Correlation between VO_2max estimated by FitMáx and measured VO_2peak

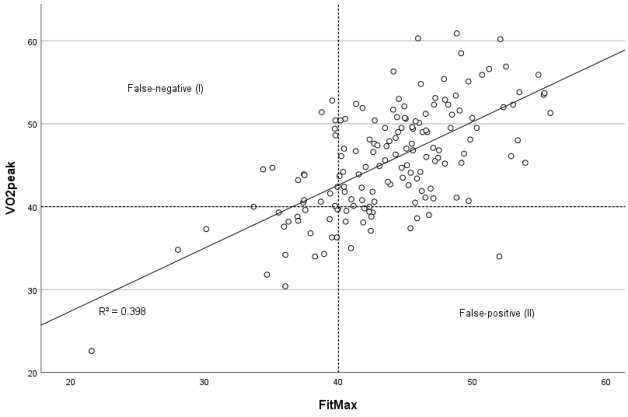
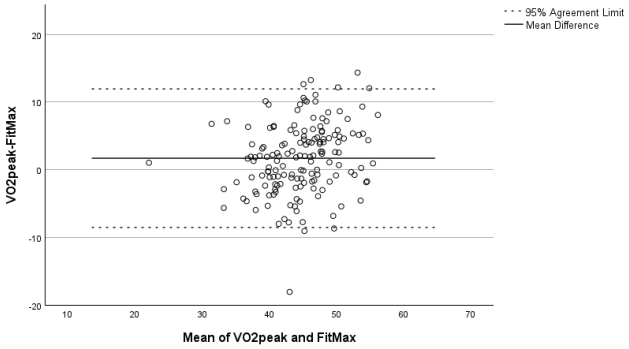


Figure 2
Bland-Altman plot of differences; VO_2max estimated by FitMáx vs measured VO_2peak



Questionnaire-based assessments of $\dot{V}O_2$peak are widely used in occupational medicine and have demonstrated moderate to strong correlations with measured exercise tolerance.[8] However, few studies have evaluated their validity in military populations or considered their utility when rendering a 'verdict', such as pass/fail fitness-to-dive assessments. A false-negative and false-positive percentage of 8.9% and 10.8%, respectively, would introduce considerable noise in exercise tolerance tests as part of fitness-to-dive assessments. This has significant consequences for diving safety, at least in a military context. How this would shift if the FitMáx questionnaire would have been used in isolation, i.e., without being followed with direct ergometry as is the current practice, is hard to predict. However, we feel that subjects could be inclined to overestimate their physical performance, as being fit to dive also has financial consequences for occupational divers as it affects their fitness-to-dive status. Conversely, if the subject would want to deliberately fail this assessment ,for instance in rapid mobilisation where not every subject may be motivated to partake in operational diving activities, if would have been easier to incorrectly answer a questionnaire than to feign reaching one's exercise tolerance in a direct or indirect test.

Many of the limitations outlined above also apply to the use of questionnaires for assessing cardiopulmonary exercise tolerance. Moreover, direct exercise testing offers additional insights, such as detailed analysis of breathing patterns, cardiac rhythm, and personalised recommendations for physical training.[9] However, such testing requires specialised equipment, trained personnel, and significant time, which may be impractical in scenarios involving large-scale assessments, such as the rapid evaluation of candidates during military mobilisation. Indirect field assessments, such as multi stage shuttle tests or other submaximal protocols , or more practical approaches like a swimming test, may be more feasible in these scenarios, allowing assessment of large numbers of recruits while minimising the logistic burden on the system. Furthermore, the financial burden of direct testing may render it unsuitable for recreational divers. However, in recreational diving many of these limitations are nonexistent. In these contexts, validated questionnaires, or a 'staged approach', e.g., where the FitMax questionnaire could be used to determine whether a subject requires exercise testing for the fitness-to-dive assessment, may serve as a pragmatic alternative, balancing feasibility with clinical utility.

Given these considerations, the Royal Netherlands Navy Diving Medical Center has not incorporated the FitMáx questionnaire into its fitness-to-dive evaluation protocol. Future iterations of the FitMáx, or other questionnaires designed to estimate VO_2peak, will be assessed for potential usage.

LIMITATIONS

First, this study was conducted among military personnel, a population characterised by a predominance of young, physically fit males. As such, the findings may not generalise to other diving populations, such as recreational divers. Caution is therefore warranted when extrapolating these results to broader or distinct groups.

Second, although the study included a substantial sample size, the resolution was insufficient to differentiate between subgroups, such as divers versus recompression personnel or varying age cohorts, among whom the questionnaire's validity may differ. Future research could explore subgroup-specific performance to refine the algorithm and improve its applicability.

Lastly, while the moderate correlation observed may have been influenced by outliers, excluding these individuals would enhance statistical validity at the group level but fail to improve clinical utility for individual assessments. The primary objective of fitness-to-dive evaluations is to identify at-risk individuals, and we believe that complex statistical adjustments are not appropriate with this practical aim in mind.

Conclusions

FitMáx demonstrated moderate correlation ($R^2 = 0.398$) with measured $\dot{V}O_2$peak in military divers but underestimated fitness (~1.8 $mL \cdot kg^{-1} \cdot min^{-1}$) with a 95% confidence interval of agreement limit of ± 10.9 $mL \cdot kg^{-1} \cdot min^{-1}$, a moderate to good reliability (ICC = 0.748). A misclassification rate of 9.8% false-negative and 10.8% false-positive, combined with the aforementioned test characteristics and potential self-reporting biases, render it insufficiently precise for fitness-to-dive evaluations in military divers. Future research could refine questionnaire-based tools to improve accuracy or expand to other domains, such as recreational diving.

References

1 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.

2 Wendling J, vanden Eede R, Elliott DH, Meintjes J, Méliet J-L, Nome T, editors. Medical assessment for work under pressure – EDTC Fitness to dive standards. 2nd ed. London, UK: International Marine Contractors Association; 2024.

3 Lee J, Zhang XL. Physiological determinants of VO_{2max} and the methods to evaluate it: A critical review. Sci Sports. 2021;36:259–71. doi: 10.1016/j.scispo.2020.11.006.

4 Schembre SM, Riebe DA. Non-exercise estimation of VO(2)max using the International Physical Activity Questionnaire. Meas Phys Educ Exerc Sci. 2011;15:168–81. doi: 10.1080/1091367X.2011.568369. PMID: 21927551. PMCID: PMC3173948.

5 Meijer R, van Hooff M, Papen-Botterhuis NE, Molenaar CJL, Regis M, Timmers T, et al. Estimating VO2peak in 18-90 year-old adults: Development and validation of the FitMáx©-questionnaire. Int J Gen Med. 2022;15:3727–37. doi: 10.2147/IJGM.S355589. Erratum in: Int J Gen Med. 2025;18:6091–4. doi: 10.2147/IJGM.S565126. PMID: 35411174. PMCID: PMC8994663.

6 Meijer R, Schep G, Regis M, Papen-Botterhuis NE, Savelberg HHCM, van Hooff M. Test-retest reliability of the FitMáx©-questionnaire in a clinical and healthy population. J Patient Rep Outcomes. 2024;8(1):3. doi: 10.1186/s41687-023-00682-9. PMID: 38175456. PMCID: PMC10767039.

7 Santtila M, Häkkinen K, Pihlainen K, Kyröläinen H. Comparison between direct and predicted maximal oxygen uptake measurement during cycling. Mil Med. 2013;178:234–8. doi: 10.7205/milmed-d-12-00276. PMID: 23495472.

8 Matsuo T, So R, Takahashi M. Workers' physical activity data contribute to estimating maximal oxygen consumption: a questionnaire study to concurrently assess workers' sedentary behavior and cardiorespiratory fitness. BMC Public Health. 2020;20(1):22. doi: 10.1186/s12889-019-8067-4. PMID: 31914965. PMCID: PMC6950791.

9 Koutlianos N, Dimitros E, Metaxas T, Cansiz M, Deligiannis A, Kouidi E. Indirect estimation of VO_2max in athletes by ACSM's equation: valid or not? Hippokratia. 2013;17(2):136–40. PMID: 24376318. PMCID: PMC3743617.

Conflicts of interest and funding: nil

Submitted: 16 April 2026
Accepted after revision: 25 July 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

World as it is

Adaptations to breath-hold diving: from traditional divers to elite athletes

Jochen D Schipke[1], Ulrich Limper[2,3], Kay Tetzlaff[4]

[1] *Research Group Experimental Surgery, University Hospital Düsseldorf, Düsseldorf, Germany*
[2] *German Aerospace Center, Institute of Aerospace Medicine, Cologne, Germany*
[3] *Department of Anaesthesiology and Critical Care Medicine, Cologne-Merheim Medical Center, Hospital of Witten/ Herdecke University, Cologne, Germany*
[4] *Department of Sports Medicine, Medical Clinic, University Hospital Tuebingen, Germany*

Corresponding author: *Professor Jochen Schipke, Research Group Experimental Surgery, University Hospital Düsseldorf, Moorenstrasse 5, D-40545 Düsseldorf, Germany*
ORCiD: *0000-0002-1747-5657*
j.schipke@gmx.org

Keywords
Adaptation; Breath-hold diving; Decompression sickness; History; Hypoxic blackout; Nitrogen; Repetitive diving; Taravana

Abstract

(Schipke JD, Limper U, Tetzlaff K. Adaptations to breath-hold diving: from traditional divers to elite athletes. Diving and Hyperbaric Medicine. 2026 30 September;56(3):365–370. doi: 10.28920/dhm56.3.365-370. PMID: 42743578.)
Breath-hold diving exposes humans to repeated episodes of profound hypoxia and hypercapnia, eliciting physiological adaptations that enable prolonged underwater performance. This article summarises current knowledge on chronic adaptations in elite breath-hold athletes and traditional diving populations, including the Bajau sea nomads of Southeast Asia and the Korean Haenyeo divers. Evidence indicates that repeated apnoea induces adaptations across multiple physiological systems. Haematological changes include increased spleen size and enhanced splenic contraction, augmenting circulating haemoglobin and oxygen stores during apnoea. In elite divers, structured training can increase resting spleen volume, whereas the Bajau exhibit genetically associated splenic enlargement linked to variants near the PDE10A gene. Cardiopulmonary adaptations include modified pulmonary vascular responses to hypoxia, improved oxygen conservation, and metabolic shifts favoring efficient mitochondrial energy production. Molecular adaptations involve enhanced antioxidant defenses and activation of hypoxia-responsive pathways that may mitigate oxidative stress associated with repeated hypoxia-reoxygenation cycles. Emerging evidence also suggests neural plasticity and possible structural brain adaptations, although the long-term neurological consequences of chronic intermittent hypoxia exposure remain uncertain. Studies of traditional diving populations indicate that both phenotypic plasticity and genetic selection contribute to diving capacity, highlighting interactions between training and evolution. Despite these benefits, breath-hold diving also carries risks, including hypoxic blackout, decompression sickness, and potential neurological injury. Understanding the mechanisms underlying human tolerance to extreme hypoxia may have implications beyond diving physiology, including applications in cardiovascular medicine, hypoxic diseases, and rehabilitation. Further longitudinal, genomic, and mechanistic studies are needed to clarify the limits, benefits, and clinical relevance of these adaptations.

Introduction

Breath-hold diving has been practiced by humans for thousands of years, driven both by subsistence needs and, more recently, recreational and competitive sport. This practice subjects the body to repeated episodes of severe oxygen deprivation, creating selective pressure for physiological adaptation. Unlike gradual altitude hypoxia, apneic diving imposes acute arterial oxygen desaturation – often below 50% – while carbon dioxide accumulates.[1,2] Elite competitive divers now achieve static apnoeas exceeding 8 min and descents beyond 100 m, performances that would rapidly incapacitate untrained individuals.[3]

Competitive diving represents the modern manifestation of a much older human relationship with underwater foraging. Traditional diving populations – including the Bajau people of the Coral Triangle, Moken sea nomads of the Andaman Sea, Japanese Amas and Korean Haenyeo divers – have practiced breath-hold diving for subsistence over multiple generations.[4-6] These groups spend hours each day diving to depths of 20–70 m to harvest fish, shellfish, and sea cucumbers. Unlike athletes who train for performance peaks, traditional divers maintain diving practices throughout their lifespans, from childhood through old age, thereby creating sustained selective pressures that potentially favor genetic adaptations alongside phenotypic plasticity.[7]

The acute diving response – bradycardia, peripheral vasoconstriction, and splenic contraction – was already characterised in marine mammals by Scholander in 1940[8] and later confirmed in humans.[9,10] These reflexes serve to conserve limited oxygen stores for vital organs during submersion. However, chronic adaptations develop over months to years of regular diving, distinguishing experienced divers from novices.[11,12] This article examines these chronic adaptations across multiple physiological systems, integrating findings from traditional populations and competitive athletes to illuminate both human plasticity and possible evolutionary adaptations to hypoxic stress.

Haematological adaptations: the splenic reservoir

SPLENIC CONTRACTION AND ENLARGEMENT IN ATHLETES

Splenic enlargement represents a well-characterised adaptation in breath-hold divers. The spleen serves as an erythrocyte reservoir; its contraction during apnoea elevates circulating haemoglobin, increasing oxygen-carrying capacity when oxygen is most limited. While splenic contraction occurs universally during apnoea, elite athletes demonstrate both larger resting splenic volumes and greater contraction magnitudes than untrained controls.[13]

Contraction can reduce splenic volume by 40 to 50% in elite divers, releasing up to 200 ml of blood and elevating haemoglobin concentration by 3 to 10% – potentially extending tolerable apnoea duration by 30 to 60 s.[14] Longitudinal training studies demonstrate that resting splenic volume increases approximately 20% after eight weeks of structured apnoea training.[15] This hypertrophy appears structural rather than transient, persisting weeks after training cessation. The mechanisms likely involve chronic sympathetic stimulation and smooth muscle proliferation within the splenic capsule, occurring independently of erythropoietin-mediated red cell production.[16] Although splenic contraction consistently increases circulating haemoglobin during apnoea, its quantitative contribution to overall diving performance remains incompletely resolved, as improvements in apnoea duration likely reflect the integrated effects of multiple cardiovascular, metabolic, and neural adaptations rather than a single mechanism. This process may involve structural remodeling of the splenic capsule, although direct evidence in humans remains limited, and current knowledge is primarily based on functional and volumetric observations following apnoea training.[14,15,17]

THE BAJAU: GENETIC ADAPTATION FOR LARGER SPLEENS

The Bajau people, sea nomads inhabiting the waters of Indonesia, Malaysia, and the Philippines, represent a case of possible genetic adaptation to diving. Bajau divers routinely descend to depths of 70 m while hunting fish with spear guns, often spending up to five hours per day underwater throughout their lifetimes.[17] Notably, a participant in one study reported a single dive duration of up to 13 min.[18] Comparative studies reveal that Bajau individuals possess spleens approximately 50% larger than neighboring non-diving populations, even among Bajau who do not actively dive, suggesting genetic rather than purely training-induced enlargement.[19] Genomic analyses identified positive selection signatures near the PDE10A gene, which regulates thyroid hormone levels and, consequently, spleen size. PDE10A variants associated with larger spleens occur at higher frequencies in Bajau than in agrarian Saluan neighbors, suggesting selection favoring enhanced oxygen storage capacity over multiple generations.[19] This finding represents one example of recent human genetic adaptation to an environmental stressor. Whether Bajau individuals also exhibit enhanced splenic contractility during apnoea, or whether their constitutively larger spleens suffice to confer advantage, remains incompletely explored. However, the functional relevance of increased spleen size for actual diving performance in Bajau individuals remains to be directly demonstrated.

Haenyeo: lifespan diving practices

The Haenyeo ('sea women') of Jeju Island, South Korea, represent a culturally distinct but physiologically comparable group. Haenyeo, predominantly women, have harvested shellfish and seaweed through breath-hold diving for centuries. Recent comprehensive studies reveal these divers spend an average of 56% of their diving time underwater – up to five hours daily – the highest proportion documented in humans and exceeding some diving mammals.[20] They typically dive to 10–20 m, performing 15–31 dives per hour.[21] Studies of aging Haenyeo reveal sustained diving capacity into their 60s and 70s, suggesting that accumulated adaptations resist age-related declines in respiratory function.[22]

Recent genomic analyses demonstrate that Haenyeo possess both training-induced and genetic adaptations. Lee and colleagues (2017) identified gene variants associated with reduced diastolic blood pressure. An enhanced cold tolerance in Haenyeo compared to mainland Koreans has been described already earlier.[23] The blood pressure variants may represent selection for the protection of pregnancies, as Haenyeo historically dove throughout gestation. Physiological measurements revealed enhanced bradycardia during simulated dives – likely a training effect rather than genetic – with heart rates decreasing by 18.8 beats·min^{-1} compared to 12.6 beats·min^{-1} in non-diving controls.[24] These findings establish Haenyeo as the second documented population, after Bajau, exhibiting genetic adaptations for diving.

However, lifelong breath-hold diving imposes physiological costs. Haenyeo experience decompression sickness

(DCS) attributed to repetitive dives. A study of 527 active Haenyeo identified three symptom clusters: type 1 (musculoskeletal), type 2 (neurological and systemic), and mixed presentations.[21] Type 2 symptoms, affecting the central nervous system, were more severe in older divers with longer diving careers, raising concerns about cumulative neurological effects. Additionally, repetitive diving has been associated with chronic headaches and analgesic overuse.[25] These findings underscore the need to balance adaptations against potential long-term health consequences. This likely reflects cumulative inert gas uptake during repetitive dives with short surface intervals.

Cardiopulmonary adaptations

PULMONARY VASCULAR CONTROL

Elite divers demonstrate refined pulmonary vascular control that optimises gas exchange under hypoxic conditions. During apnoea, pulmonary vascular resistance increases, reducing blood flow to under-aerated alveoli – a phenomenon termed hypoxic pulmonary vasoconstriction (HPV).[26] Another study showed a significantly smaller increase in total pulmonary resistance compared to controls after isocapnic hypoxia.

The authors suggest that this blunted response is a beneficial adaptation that allows for increased cardiac output with reduced right heart work, thus reducing cardiac oxygen utilisation in hypoxaemic conditions. Both vascular remodeling and altered endothelial function may contribute to this adaptation.[27] While acute apnoea induces hypoxic pulmonary vasoconstriction, trained divers may exhibit a blunted or more finely regulated response.

Whether traditional diving populations exhibit similar adaptations remains unexplored, though their sustained diving across lifespans suggests comparable or enhanced vascular plasticity.

CARDIAC METABOLIC ADAPTATIONS

Cardiac tissue must maintain function despite severe systemic hypoxia during prolonged apnoeas. Kjeld and colleagues (2018) analysed skeletal muscle biopsies from elite divers, revealing increased mitochondrial density and enhanced expression of genes involved in fatty acid oxidation – substrates that generate more ATP per oxygen molecule than glucose. These metabolic shifts likely extend to cardiac muscle, enabling sustained contractile function under oxygen-limited conditions.[28] Furthermore, divers exhibit reduced oxidative stress markers post-apnoea compared to controls, suggesting upregulated antioxidant defenses that mitigate hypoxia-reoxygenation injury.[28,29] Such adaptations may confer cardioprotective effects analogous to ischaemic preconditioning observed in clinical settings.[29]

Neural adaptations and cognitive considerations

The brain, despite representing only 2% of body mass, consumes approximately 20% of total oxygen.[30] Severe hypoxia threatens neuronal viability, yet elite divers tolerate arterial saturations that would induce unconsciousness in untrained individuals. Preliminary neuroimaging suggests structural changes in divers' brains. Increased hippocampal gray matter volume has been reported in some athletes, potentially reflecting neuroplasticity comparable to that observed with exercise-induced changes in non-divers.[31] However, the functional significance and consistency of these findings remain debated. Some studies report subtle memory deficits among competitive divers, raising concerns about cumulative hypoxic exposure,[32] though the causality and clinical relevance remain unclear. Traditional divers like the Haenyeo, who maintain cognitive function into advanced age despite decades of hypoxic exposure,[33] provide counterevidence against inevitable neurological harm, yet controlled neuropsychological assessments in these populations are lacking. However, direct evidence linking apnoea training to these structural changes remains limited.

Molecular mechanisms: hypoxia-inducible factors and oxidative defenses

At the cellular level, hypoxia-inducible factors (HIFs) orchestrate transcriptional responses to low oxygen. HIF-1α and HIF-2α are stabilised under hypoxia, activating genes that govern angiogenesis, erythropoiesis, and metabolic reprogramming.[34] Whether chronic intermittent apnoea in divers induces sustained HIF activation or altered HIF signaling thresholds remains incompletely characterised. Repeated hypoxia-reoxygenation also generates reactive oxygen species (ROS), which can cause oxidative damage. Yet divers exhibit elevated baseline antioxidant enzyme activities, superoxide dismutase, catalase, glutathione peroxidase, suggesting adaptive upregulation that mitigates oxidative stress.[35] This hormetic response, wherein moderate oxidative stress triggers protective adaptations, may underlie observed cardioprotective and neuroprotective effects.

Individual variability and genetic factors

Variability exists in adaptive responses to apnoea training. Some individuals demonstrate substantial splenic hypertrophy, while others show minimal changes despite identical training.[13] Genetic polymorphisms in genes regulating hypoxic sensing (e.g., EPAS1, EGLN1) or vascular function (e.g., NOS3, ACE) may modulate individual adaptive capacity. The Bajau case demonstrates that population-level genetic selection can enhance diving-related traits, but within-population variability likely reflects polygenic architecture, in which numerous variants collectively influence the phenotype. Age, sex, and prior training history also contribute. Women generally exhibit

smaller baseline splenic volumes but comparable relative contraction percentages.[17] Whether sex differences in diving performance reflect physiological constraints or sociocultural factors remains debated, though Haenyeo divers demonstrate that women can sustain elite diving performance across lifespans.

Potential risks and maladaptive consequences

While adaptations generally enhance hypoxic tolerance, potential maladaptive consequences warrant consideration. As humans lack the ability of cognitive perception of hypoxia, severe hypoxic episodes can trigger loss of motor control ('blackout'), particularly during ascent when ambient pressure decreases, and arterial oxygen precipitously drops.[36] Competitive divers occasionally experience blackouts, requiring rescue to prevent drowning.[37] Whether repeated blackouts cause cumulative neural injury remains unknown. Additionally, concerns exist regarding the risk of arterial gas embolism (AGE);[38] rapid ascents from depth may generate intrapulmonary pressure gradients that force gas into the pulmonary veins.[39] Traditional divers, whose gradual ascents and conservative depth limits contrast with competitive extremes, appear to avoid such pathology, suggesting that dose-response thresholds distinguish beneficial adaptation from harmful exposure.

Clinical and evolutionary implications

Understanding diving adaptations holds clinical relevance beyond performance optimisation. Chronic obstructive pulmonary disease, obstructive sleep apnoea, and high-altitude illnesses involve chronic or intermittent hypoxia. Mechanisms enabling divers to tolerate severe hypoxia, enhanced antioxidant defenses, metabolic flexibility, cerebrovascular autoregulation, may inform therapeutic strategies for these conditions. Intermittent hypoxic training protocols, inspired by diving and altitude physiology, are being explored for cardiovascular rehabilitation.[40] Additionally, perioperative risk stratification might benefit from understanding individual hypoxic tolerance, potentially identifying patients at elevated risk for complications during anaesthesia.

From an evolutionary perspective, the Bajau provide a case study of recent human adaptation. Their genetic signatures demonstrate that within the last few thousand years, selection can reshape human physiology in response to environmental pressures. This challenges the notion that human evolution is solely ancient history, revealing ongoing adaptive processes in contemporary populations.[7] Whether other diving populations harbor similar genetic adaptations, or whether phenotypic plasticity alone suffices to explain their capabilities, remains an open question with implications for understanding the interplay between genes and environment in shaping human diversity.

Future directions and knowledge gaps

Gaps still persist in our understanding of diving adaptations. Some areas that could benefit from further investigation include:

» Longitudinal studies tracking adaptations from novice to elite status remain relatively uncommon, which would help strengthen causal inference regarding training effects.
» Molecular profiling of HIF pathway dynamics, mitochondrial function, and epigenetic modifications across training timescales could help elucidate underlying mechanisms.
» Comparative genomic studies of Moken, Haenyeo, and other traditional diving populations could help determine whether genetic adaptations analogous to those of the Bajau exist elsewhere, or whether the Bajau represent a unique evolutionary outcome.
» Neuroimaging studies combining structural MRI, functional MRI, and neuropsychological testing could help clarify whether cognitive changes in divers reflect detrimental hypoxic injury or benign plasticity.
» Safety thresholds distinguishing adaptive from maladaptive hypoxic exposure remain to be fully defined; establishing evidence-based guidelines for training intensity and recovery could help reduce injury risk while optimising performance.
» Translational research exploring whether diving-derived adaptations can be pharmacologically or behaviorally induced in non-divers could potentially yield novel therapeutic interventions for hypoxic diseases.

Conclusions

Breath-hold diving appears to elicit physiological adaptations spanning hematological, cardiopulmonary, metabolic, and neural systems. Elite athletes can develop these adaptations through intensive training, while traditional populations such as the Bajau and Haenyeo have sustained diving practices across generations, potentially embedding these adaptations into their genomes. The interplay between phenotypic plasticity and genetic selection in shaping human diving capacity may offer insights into principles of adaptive physiology and evolutionary medicine. Continued research integrating exercise physiology, genomics, and clinical medicine has the potential to reveal therapeutic applications of these adaptive mechanisms while enriching our understanding of human biological diversity and resilience to environmental challenges. This should be interpreted cautiously, as such findings rather suggest potential heritable genetic adaptations rather than fully established genomic embedding.

References

1 Andersson JPA, Schagatay E. Repeated apneas do not affect the hypercapnic ventilatory response in the short term. Eur J Appl Physiol. 2009;105:569–74. doi: 10.1007/s00421-008-0936-y. PMID: 19018556.

2 Bailey DM, Willie CK, Hoiland RL, Bain AR, MacLeod DB, Santoro MA, et al. Surviving without oxygen: How low can the human brain go? High Alt Med Biol. 2017;18:73–9. doi: 10.1089/ham.2016.0081. PMID: 28002687.

3 Ferretti G. Extreme human breath-hold diving. Eur J Appl Physiol. 2001;84:254–71. doi: 10.1007/s004210000377. PMID: 11374109.

4 Teruoka G. Die Ama und ihre Arbeit: Vorläufiger Bericht. Eine Untersuchung über die japanischen Taucherinnen. Arbeitsphysiologie. 1932;5:239–51. doi: 10.1007/BF02009115.

5 Hong SK, Rahn H, Kang DH, Song SH, Kang BS. Diving pattern, lung volumes, and alveolar gas of the Korean diving woman (ama). J Appl Physiol (1985). 1963;18:457–65. doi: 10.1152/jappl.1963.18.3.457. PMID: 31083859.

6 Schagatay E, Lodin-Sundstrom A, Abrahamsson E. Underwater working times in two groups of traditional apnea divers in Asia: the Ama and the Bajau. Diving Hyperb Med. 2011;41:27–30. PMID: 21560982. [cited 2026 Apr 30]. Available from: https://dhmjournal.com/images/IndividArticles/41March/Schagatay_dhm.41.1.27-30.pdf.

7 Ilardo M, Nielsen R. Human adaptation to extreme environmental conditions. Curr Opin Genet Dev. 2018;53:77–82. doi: 10.1016/j.gde.2018.07.003. PMID: 30077046. PMCID: PMC7193766.

8 Scholander PF, Hammel HT, Lemessurier H, Hemmingsen E, Garey W. Circulatory adjustment in pearl divers. J Appl Physiol. 1962;17:184–90. doi: 10.1152/jappl.1962.17.2.184. PMID: 13909130.

9 Foster GE, Sheel AW. The human diving response, its function, and its control. Scand J Med Sci Sports. 2005;15:3–12. doi: 10.1111/j.1600-0838.2005.00440.x. PMID: 15679566.

10 Gooden BA. Mechanism of the human diving response. Integr Physiol Behav Sci. 1994;29:6–16. PMID: 8018553.

11 Elia A, Gennser M, Harlow PS, Lees MJ. Physiology, pathophysiology and (mal)adaptations to chronic apnoeic training: a state-of-the-art review. Eur J Appl Physiol. 2021;121:1543–66. doi: 10.1007/s00421-021-04664-x. PMID: 33791844. PMCID: PMC8144079.

12 Pernett F, Bergenhed P, Holmström P, Mulder E, Schagatay E. Effects of hyperventilation on oxygenation, apnea breaking points, diving response, and spleen contraction during serial static apneas. Eur J Appl Physiol. 2023;123:1809–24. doi: 10.1007/s00421-023-05202-7. PMID: 37060440. PMCID: PMC10363065.

13 Schagatay E, Richardson MX, Lodin-Sundstrom A. Size matters: spleen and lung volumes predict performance in human apneic divers. Front Physiol. 2012;3:173. doi: 10.3389/fphys.2012.00173. PMID: 22719729. PMCID: PMC3376424.

14 Bakovic D, Valic Z, Eterovic D, Vukovic I, Obad A, Marinovic-Terzic I, et al. Spleen volume and blood flow response to repeated breath-hold apneas. J Appl Physiol (1985). 2003;95:1460–6. doi: 10.1152/japplphysiol.00221.2003. PMID: 12819225.

15 Bouten J, Caen K, Stautemas J, Lefevere F, Derave W, Lootens L, et al. Eight weeks of static apnea training increases spleen volume but not acute spleen contraction. Respir Physiol Neurobiol. 2019;266:144–9. doi: 10.1016/j.resp.2019.04.002. PMID: 31009754.

16 Schagatay E, Holmström P, Mulder E, Limbu P, Schagatay FS, Engan H, et al. Spleen volume and contraction during apnea in Mt. Everest climbers and everest base camp trekkers. High Alt Med Biol. 2020;21:84–91. doi: 10.1089/ham.2019.0028. PMID: 32182148.

17 Espersen K, Frandsen H, Lorentzen T, Kanstrup IL, Christensen NJ. The human spleen as an erythrocyte reservoir in diving-related interventions. J Appl Physiol (1985). 2002;92:2071–9. doi: 10.1152/japplphysiol.00055.2001. PMID: 11960959.

18 University of Cambridge. Genetic adaptations to diving discovered in humans for the first time. PHYS.ORG; 2018. [cited 2026 Apr 30]. Available from: https://phys.org/news/2018-04-genetic-humans.html.

19 Ilardo MA, Moltke I, Korneliussen TS, Cheng J, Stern AJ, Racimo F, et al. Physiological and genetic adaptations to diving in sea nomads. Cell. 2018;173:569–580.e15. doi: 10.1016/j.cell.2018.03.054. PMID: 29677510.

20 McKnight JC, Solms B, Jensen M, Turnbull J, Balfour S, Laagland M, et al. Diving behaviour and physiology of the Korean Haenyeo. Curr Biol. 2025;35(16):R797–8. doi: 10.1016/j.cub.2025.06.066. PMID: 40829560.

21 Kim DJ, Han JW. Latent class analysis of decompression sickness symptoms of women divers. Healthcare (Basel). 2022;10(7):1246. doi: 10.3390/healthcare10071246. PMID: 35885774. PMCID: PMC9317702.

22 Hong SK, Henderson J, Olszowka A, Hurford WE, Falke KJ, Qvist J, et al. Daily diving pattern of Korean and Japanese breath-hold divers (ama). Undersea Biomed Res. 1991;18(5–6):433–43. PMID: 1746069.

23 Lee JY, Park J, Kim S. Cold adaptation, aging, and Korean women divers haenyeo. J Physiol Anthropol. 2017;36(1):33. doi: 10.1186/s40101-017-0146-6. PMID: 28789677. PMCID: PMC5549283.

24 Aguilar-Gómez D, Bejder J, Graae J, Ko Y, Vaughn A, Clement K, et al. Genetic and training adaptations in the Haenyeo divers of Jeju, Korea. Cell Reports. 2025;44:115577. doi: 10.1016/j.celrep.2025.115577. PMID: 40318638. PMCID: PMC12129667.

25 Choi JC, Lee JS, Kang S, Kang J, Bae J. Chronic daily headache with analgesics overuse in professional women breath-hold divers. Headache. 2008;48:1037–43. doi: 10.1111/j.1526-4610.2007.01017.x. PMID: 18194291.

26 Kjeld T, Isbrand AB, Arendrup HC, Højberg J, Bejder J, Krag TO, et al. Pulmonary vascular adaptations to hypoxia in elite breath-hold divers. Front Physiol. 2024;15:1296537. doi: 10.3389/fphys.2024.1296537. PMID: 39135706. PMCID: PMC11318387.

27 Kelly T, Brown C, Bryant-Ekstrand M, Lord R, Dawkins T, Drane A, et al. Blunted hypoxic pulmonary vasoconstriction in apnoea divers. Exp Physiol. 2022;107:1225–40. doi: 10.1113/EP090326. PMID: 35993480.

28 Kjeld T, Stride N, Gudiksen A, Hansen EG, Arendrup HC, Horstmann PF, et al. Oxygen conserving mitochondrial adaptations in the skeletal muscles of breath hold divers. PLoS ONE. 2018;13(9):e0201401. doi: 10.1371/journal.pone.0201401. PMID: 30231055. PMCID: PMC6145504.

29 Heusch G, Rassaf T. Time to give up on cardioprotection?: A critical appraisal of clinical studies on ischemic pre-, post-,

and remote conditioning. Circ Res. 2016;119:676–95. doi: 10.1161/CIRCRESAHA.116.308736. PMID: 27539973.
30 Clark DD, Sokoloff L. Circulation and energy metabolism in the brain. Chemistry Faculty Publications 81 [Internet]. 1999. [cited 2026 Jan 18]. Available from: https://fordham.bepress.com/chem_facultypubs.
31 Erickson KI, Voss MW, Prakash RS, Basak C, Szabo A, Chaddock L, et al. Exercise training increases size of hippocampus and improves memory. Proc Natl Acad Sci USA. 2011;108:3017–22. doi: 10.1073/pnas.1015950108. PMID: 21282661. PMCID: PMC3041121.
32 Billaut F, Gueit P, Faure S, Costalat G, Lemaitre F. Do elite breath-hold divers suffer from mild short-term memory impairments? Appl Physiol Nutr Metab. 2018;43:247–51. doi: 10.1139/apnm-2017-0245. PMID: 29053942.
33 Park JH, Kim KW. Research on Haenyeo: insights into human adaptations to extreme environments. Psychiatry Investig. 2025;22:737–40. doi: 10.30773/pi.2024.0394. PMID: 40633579. PMCID: PMC12301677.
34 Heyman SN, Leibowitz D, Mor-Yosef Levi I, Liberman A, Eisenkraft A, Alcalai R, et al. Adaptive response to hypoxia and remote ischaemia pre-conditioning: a new hypoxia-inducible factors era in clinical medicine. Acta Physiol (Oxf). 2016;216:395–406. doi: 10.1111/apha.12613. PMID: 26449466.
35 Solich-Talanda M, Żebrowska A, Mikołajczyk R, Kostorz-Nosal S, Ziora D, Jastrzębski D, et al. Effect of apnea-induced hypoxia on cardiovascular adaptation and circulating biomarkers of oxidative stress in elite breath-hold divers. Front Physiol. 2021;12:726434. doi: 10.3389/fphys.2021.726434. PMID: 34566688. PMCID: PMC8458773.
36 Mulder E, Staunton C, Sieber A, Schagatay E. Unlocking the depths: multiple factors contribute to risk for hypoxic blackout during deep freediving. Eur J Appl Physiol. 2023;123:2483–93. doi: 10.1007/s00421-023-05250-z. PMID: 37300699. PMCID: PMC10615935.
37 Fitz-Clarke JR. Breath-hold diving. Compr Physiol. 2018;8:585–630. doi: 10.1002/cphy.c160008. PMID: 29687909.
38 Blogg SL, Tillmans F, Lindholm P. The risk of decompression illness in breath-hold divers: a systematic review. Diving Hyperb Med. 2023;53:31–41. doi: 10.28920/dhm53.1.31-41. PMID: 36966520. PMCID: PMC10106275.
39 Walker JR III, Hexdall EJ, Cooper JS, Murphy-Lavoie HM. Diving gas embolism. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025. PMID: 29493946.
40 Lavie L. Oxidative stress in obstructive sleep apnea and intermittent hypoxia – Revisited – The bad ugly and good: Implications to the heart and brain. Sleep Med Rev. 2015;20:27–45. doi: 10.1016/j.smrv.2014.07.003. PMID: 25155182.

Conflicts of interest and funding: nil

Submitted: 1 March 2026
Accepted after revision: 27 June 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

Case reports

Bilateral sudden sensorineural hearing loss following indirect lightning exposure successfully treated with hyperbaric oxygen therapy: a case report

Recep Özkan[1], Osman Türkmen[2], Kübra Yapici[3], Zülküf Kaya[3]

[1] *Department of Hyperbaric and Diving Medicine, Ataturk University Erzurum, Türkiye*
[2] *Department of Hyperbaric and Diving Medicine, Samsun Training and Research Hospital, Samsun, Türkiye*
[3] *Department of Otorhinolaryngology, Atatürk University Faculty of Medicine, Erzurum, Türkiye*

Corresponding author: *Dr Recep Özkan, Department of Hyperbaric and Diving Medicine, Ataturk University, 25240 Erzurum, Türkiye*
ORCiD: *0000-0001-7342-6473*
recepzkn@hotmail.com

Keywords
Audiometry; Cochlear injury; Deafness; Lightning injury

Abstract
(Özkan R, Türkmen O, Yapici K, Kaya Z. Bilateral sudden sensorineural hearing loss following indirect lightning exposure successfully treated with hyperbaric oxygen therapy: a case report. Diving and Hyperbaric Medicine. 2026 30 September;56(3):371–375. doi: 10.28920/dhm56.3.371-375. PMID: 42743579.)
Introduction: Lightning injuries may result in a wide spectrum of neurological and otological complications. Sudden sensorineural hearing loss (SSNHL) following indirect lightning exposure is rare, and evidence regarding the role of hyperbaric oxygen therapy (HBOT) remains limited. We report a case of severe bilateral SSNHL following indirect lightning exposure with substantial recovery after HBOT.
Case report: A 26-year-old man developed sudden bilateral hearing loss after a lightning strike hit a lightning rod approximately 20 m from his location. He experienced transient syncope and limb weakness consistent with keraunoparalysis. Audiometry demonstrated severe bilateral sensorineural hearing loss, with pure-tone averages (PTA) of 70 dB HL in the right ear and 75 dB HL in the left ear. Because of persistent hearing loss despite systemic corticosteroid treatment, HBOT was initiated approximately 105 days after injury. A total of 30 HBOT sessions were administered together with intratympanic steroid injections. After 20 sessions, PTAs improved to 39 dB HL and 42 dB HL. Following completion of treatment, PTAs improved to 15 dB HL and 12 dB HL, with speech discrimination scores of 96% bilaterally.
Conclusions: Severe bilateral SSNHL following indirect lightning exposure is rarely reported. Proposed mechanisms include cochlear injury, microvascular compromise, and oxidative damage to inner-ear structures. This case demonstrates that indirect lightning exposure may result in both keraunoparalysis and severe bilateral SSNHL. Substantial hearing recovery was observed despite initiation of HBOT more than three months after injury, suggesting a potential role for HBOT as part of a multidisciplinary treatment approach in selected cases.

Introduction

Injuries due to lightning strikes are uncommon but potentially devastating, associated with substantial morbidity and mortality. Although cardiac and respiratory arrest remain the principal immediate causes of lightning-related death, survivors may experience a wide range of neurological, cardiovascular, dermatological, and otological sequelae.[1,2] The auditory system is particularly vulnerable because lightning can cause direct electrical injury, blast effects, thermal damage, and microvascular compromise affecting the structures of the middle and inner ear.[2,3]

Otologic manifestations of lightning injuries include tympanic membrane perforation, tinnitus, vertigo, conductive hearing loss, and sensorineural hearing loss. Tympanic membrane rupture is considered the most common otologic injury due to the intense shock wave generated by lightning strikes.[3,4] However, sudden sensorineural hearing loss (SSNHL) following lightning exposure is rarely reported, and its underlying pathophysiological mechanisms remain incompletely understood. Proposed mechanisms include cochlear ischaemia, disruption of hair-cell function, microvascular injury, and direct damage to auditory pathways.[2,3,5] Another neurological manifestation of lightning injury is keraunoparalysis, a transient syndrome characterised by motor weakness, sensory disturbances, and peripheral vascular symptoms that typically resolves spontaneously within hours after the event.[6,7] Most lightning injuries result from indirect mechanisms such as ground

current, side flash, or blast effects, and auditory injury may occur even in the absence of direct contact with the lightning discharge.[8,9]

Hyperbaric oxygen therapy (HBOT) is an established adjunctive treatment for idiopathic SSNHL and is believed to enhance oxygen delivery to the ischaemic cochlea, improve inner-ear oxygen tension, and facilitate recovery of injured auditory structures.[10–12] Although HBOT has been investigated in idiopathic SSNHL, data regarding its use in lightning-associated hearing loss remain limited. We report a case of severe bilateral SSNHL that developed after indirect lightning injury following a nearby lightning strike to a lightning rod and demonstrated near-complete audiometric recovery after a substantially delayed course of HBOT.

Case report

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

A 26-year-old previously healthy, non-smoking man was exposed to a lightning-related incident when a lightning strike hit a lightning rod located approximately 20 metres from his position. Immediately following the event, he experienced syncope and was transported to the emergency department for evaluation. After regaining consciousness, he reported bilateral hearing loss. He had no previous history of hearing impairment, chronic medical illness, or tobacco use.

In addition to hearing loss, the patient developed transient weakness involving both the upper and lower extremities immediately after the incident. The weakness gradually improved approximately three hours after the event and resolved completely within four hours. No residual motor deficits were identified on subsequent neurological examinations. This transient neurological presentation was considered consistent with keraunoparalysis, a well-recognised and typically reversible neurological manifestation of lightning injury.

Radiological investigations showed no intracranial or otological abnormalities. Otoscopic examination demonstrated intact tympanic membranes bilaterally without perforation.

The patient was subsequently referred to the otolaryngology department because of persistent bilateral hearing loss. Oral corticosteroid therapy (Deltacortril) was initiated eight days after the incident for bilateral SSNHL. Despite treatment, severe bilateral hearing loss persisted. Pure-tone audiometry performed prior to HBOT demonstrated pure-tone averages (PTA) of 70 dB HL in the right ear and 75 dB HL in the left ear (Figure 1). The patient was referred to our hyperbaric medicine unit approximately 105 days after the lightning-related incident.

Figure 1

Initial pure-tone audiometry demonstrating severe bilateral sensorineural hearing loss after lightning exposure. Pure-tone averages were 70 dB in the right ear and 75 dB in the left ear

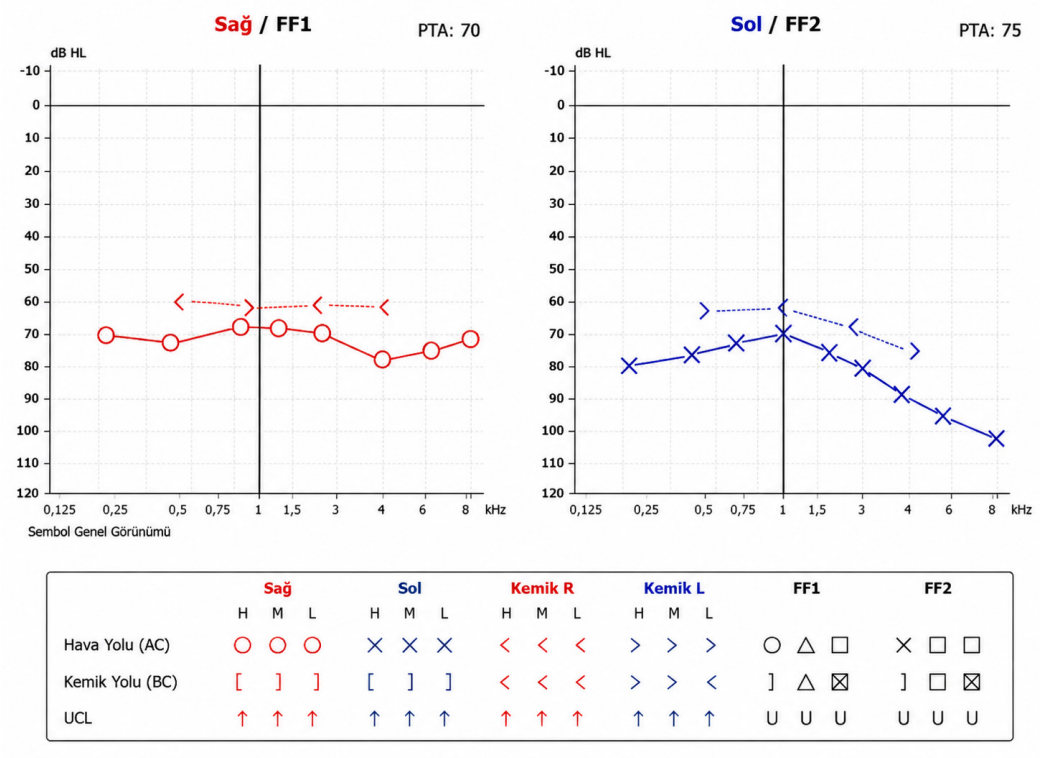


Figure 2
Pure-tone audiometry obtained after 20 sessions of hyperbaric oxygen therapy demonstrating partial recovery of hearing thresholds. Pure-tone averages improved to 39 dB in the right ear and 42 dB in the left ear, although speech discrimination scores remained limited (41% and 30%, respectively)

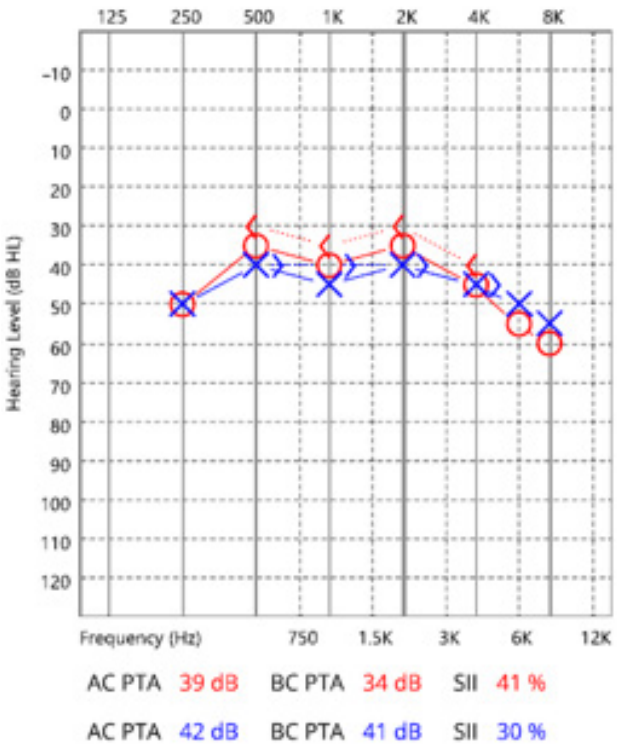
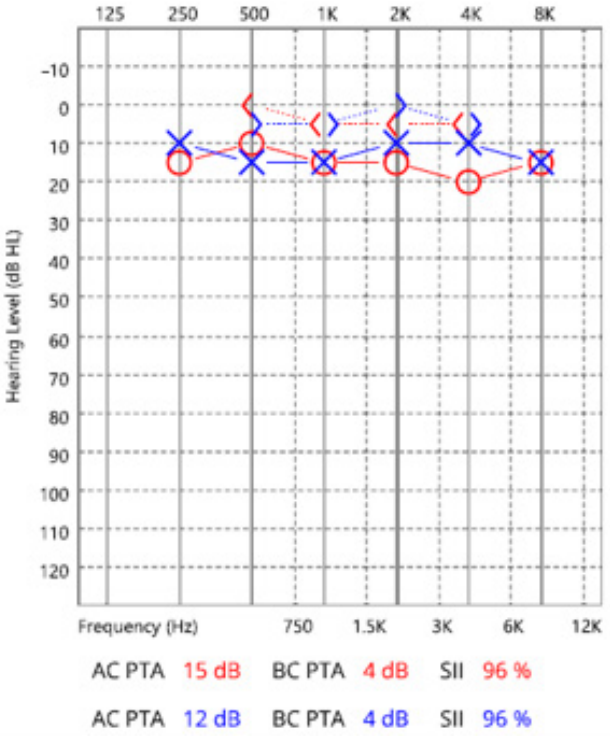


Figure 3
Final pure-tone audiometry obtained after completion of 30 hyperbaric oxygen therapy sessions and five bilateral intratympanic steroid injections demonstrating near-complete recovery. Pure-tone averages improved to 15 dB in the right ear and 12 dB in the left ear, with speech discrimination scores of 96% bilaterally



HBOT was initiated and administered in a multiplace hyperbaric chamber according to the institutional protocol for SSNHL. A total of 30 HBOT sessions were completed. In addition, bilateral intratympanic steroid injections were administered concurrently with HBOT at two-day intervals for a total of five injections in each ear.

Repeat audiometric evaluation after 20 HBOT sessions demonstrated substantial improvement in hearing thresholds. Pure-tone averages improved to 39 dB HL in the right ear and 42 dB HL in the left ear. Although hearing thresholds improved significantly, speech discrimination remained limited (41% in the right ear and 30% in the left ear) at this stage of treatment (Figure 2).

Following completion of 30 HBOT sessions and five bilateral intratympanic steroid injections, further audiometric recovery was observed. Final pure-tone audiometry demonstrated near-complete recovery, with pure-tone averages of 15 dB HL in the right ear and 12 dB HL in the left ear. Speech discrimination scores improved to 96% bilaterally (Figure 3). The patient reported marked subjective improvement in hearing and successfully returned to his normal daily activities.

The diagnosis of lightning-associated bilateral sudden sensorineural hearing loss was supported by the temporal relationship between the lightning incident and symptom onset, the presence of transient keraunoparalysis immediately after exposure, the absence of previous otological disease, the presence of severe bilateral sensorineural hearing loss on audiometric evaluation, and the exclusion of alternative neurological and otological causes. Although the patient had previously received systemic corticosteroid therapy without measurable benefit, substantial hearing recovery was observed following treatment with HBOT combined with intratympanic steroid administration.

Discussion

Lightning injuries are associated with a broad spectrum of neurological and otological complications. Otological manifestations most commonly include tympanic membrane perforation, tinnitus, vertigo, and hearing loss. Among these, tympanic membrane rupture is considered the most frequent otologic finding because of the intense blast wave generated during a lightning strike.[3,4] In contrast, SSNHL following lightning exposure is uncommon, particularly in the absence of direct lightning strike or structural ear injury.[2–4] Most lightning injuries result from indirect mechanisms such

as ground current, side flash, or blast effects rather than direct strikes.[8,9] The occurrence of severe bilateral SSNHL highlights the potential for significant auditory injury even after indirect exposure.

Isolated sensorineural hearing loss following lightning injury has been reported only rarely in the literature.[4,13] In the present case, severe bilateral SSNHL developed after indirect exposure to a nearby lightning rod strike, without tympanic membrane rupture, temporal bone injury, or radiologically detectable abnormalities. These findings suggest that inner-ear injury may occur even in the absence of overt structural damage.

The patient developed transient weakness involving both upper and lower extremities immediately after the event. The neurological deficits resolved completely within four hours and were consistent with keraunoparalysis, a characteristic but typically reversible neurological manifestation of lightning injury.[6,7] Keraunoparalysis is believed to result from transient autonomic dysfunction and intense vasospasm induced by the electrical discharge.[6,7] The presence of this phenomenon further supports a causal relationship between the lightning exposure and the subsequent auditory injury observed in our patient.

The precise mechanism responsible for hearing loss in lightning injuries remains uncertain. Several pathophysiological pathways have been proposed, including cochlear ischaemia, microvascular compromise, disruption of hair-cell function, oxidative stress, and direct neural injury.[2,3,5] In the present case, the absence of tympanic membrane perforation, temporal bone injury, or intracranial abnormalities suggests that cochlear injury and microvascular compromise may have contributed to the development of hearing loss.

HBOT has become an accepted adjunctive treatment for idiopathic SSNHL and is recommended by contemporary clinical practice guidelines.[10] HBOT increases dissolved oxygen content in plasma, improves oxygen delivery to the cochlea, and may enhance recovery of ischaemic inner-ear structures.[11,12] Although evidence supporting HBOT in idiopathic SSNHL has grown, data regarding its use in lightning-associated hearing loss remain extremely limited.

Hyperbaric oxygen therapy has also been associated with hearing improvement in conditions other than idiopathic sudden sensorineural hearing loss, including acute acoustic trauma. Studies of both acute acoustic trauma and idiopathic sudden sensorineural hearing loss have generally reported better outcomes when treatment is initiated early.[10–14] In contrast, HBOT in our patient was initiated approximately 105 days after the lightning-related injury, well beyond the conventionally accepted therapeutic window. Despite this prolonged delay, substantial audiometric recovery was observed, making the favorable outcome in the present case particularly noteworthy.

Substantial audiometric recovery was observed following treatment, with near-complete normalisation of hearing thresholds and restoration of speech discrimination scores. Oral corticosteroid therapy failed to produce measurable clinical improvement and HBOT was initiated approximately 105 days after the lightning-related injury, well beyond the conventionally accepted therapeutic window for SSNHL. Although causality cannot be established from a single case, the progressive improvement documented on serial audiometric examinations and temporal relationship between HBOT and recovery after a long treatment latency suggests a potential therapeutic benefit of HBOT-based treatment strategies in selected patients with lightning-associated SSNHL.

This case has several limitations. The combined use of HBOT and intratympanic steroid therapy precludes assessment of the independent effect of either intervention. Nevertheless, the temporal relationship between lightning exposure, bilateral SSNHL, keraunoparalysis, and subsequent audiometric recovery provides valuable clinical observations. To our knowledge, reports describing successful treatment of lightning-associated bilateral SSNHL with HBOT remain exceedingly limited.

Conclusions

Severe bilateral SSNHL may occur following indirect lightning exposure even in the absence of direct lightning strike or radiologically detectable injury. Clinicians should be aware of this uncommon presentation and consider early audiological assessment in symptomatic patients. HBOT, particularly when incorporated into a multidisciplinary treatment approach, may represent a useful therapeutic option in selected cases of lightning-associated SSNHL.

References

1 Jensen JD, Thurman J, Vincent AL. Lightning Injuries. 2023. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026. PMID: 28722949.

2 Ritenour AE, Morton MJ, McManus JG, Barillo DJ, Cancio LC. Lightning injury: a review. Burns. 2008;34:585–94. doi: 10.1016/j.burns.2007.11.006. PMID: 18395987.

3 Ogren FP, Edmunds AL. Neuro-otologic findings in the lightning-injured patient. Semin Neurol. 1995;15:256–62. doi: 10.1055/s-2008-1041030. PMID: 8570927.

4 Kiliç E, Genç H, Aydin Ü, Aşik B, Satar B. Variations in otological presentation of lightning strike victims: Clinical report of 3 patients. Ulus Travma Acil Cerrahi Derg. 2017;23:163–6. doi: 10.5505/tjtes.2016.88580. PMID: 28467585.

5 Wong AC, Ryan AF. Mechanisms of sensorineural cell damage, death and survival in the cochlea. Front Aging Neurosci. 2015;7:58. doi: 10.3389/fnagi.2015.00058. PMID: 25954196. PMCID: PMC4404918.

6 Jacob NE, Rashika M. Keraunoparalysis: fleeting paralysis following a lightning flash. Cureus. 2024;16(10):e70773. doi: 10.7759/cureus.70773. PMID: 39493204. PMCID: PMC11531350.

7 Cherington M, Yarnell PR, London SF. Neurologic complications of lightning injuries. West J Med. 1995;162:413–7. PMID: 7785254. PMCID: PMC1022790.
8 Cooper MA, Holle RL. Mechanisms of lightning injury should affect lightning safety messages. In: Preprints of the International Lightning Detection Conference. Tucson (AZ); 2008.
9 van Ruler R, Eikendal T, Kooij FO, Tan ECTH. A shocking injury: A clinical review of lightning injuries highlighting pitfalls and a treatment protocol. Injury. 2022;53:3070–7. doi: 10.1016/j.injury.2022.08.024. PMID: 36038387.
10 Chandrasekhar SS, Tsai Do BS, Schwartz SR, Bontempo LJ, Faucett EA, Finestone SA, et al. Clinical practice guideline: Sudden hearing loss (update). Otolaryngol Head Neck Surg. 2019;161(1_suppl):S1–S45. doi: 10.1177/0194599819859885. PMID: 31369359.
11 Murphy-Lavoie H, Piper S, Moon RE, Legros T. Hyperbaric oxygen therapy for idiopathic sudden sensorineural hearing loss. Undersea Hyperb Med. 2012;39:777–92. PMID: 22670557.
12 Newth A, Perleth M, Sherlock S, Romero L, Bennett MH. Hyperbaric oxygen therapy for acute idiopathic sudden sensorineural hearing loss; a systematic review with meta-analysis. Diving Hyperb Med. 2025;55:398–406. doi: 10.28920/dhm55.4.398-406. PMID: 41364864. PMCID: PMC13092557.
13 Turan M, Kalkan F, Bozan N, Özçalimli İ, Zeki Erdem M, Yalınkılıç A, et al. Isolated sensorineural hearing loss as a sequela after lightning strike. Case Rep Otolaryngol. 2015;2015:738416. doi: 10.1155/2015/738416. PMID: 26161278. PMCID: PMC4487271.
14 Manheim M, Mogilevsky L, Geva A, Zehavi G, Knoll O, Gur I. Effects of hyperbaric oxygen therapy initiation latency on auditory outcomes following acute acoustic trauma. Diving Hyperb Med. 2025;55:126–35. doi: 10.28920/dhm55.2.126-135. PMID: 40544140. PMCID: PMC12520991.

Conflicts of interest and funding: nil

Submitted: 9 June 2026
Accepted after revision: 27 June 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

A case of severe multi-system decompression sickness requiring critical care, aggressive fluid resuscitation and hyperbaric oxygen therapy

Alice Palmer[1,2], Craig Holdstock[1,2], Doug Watts[3], Rosanna J Stokes[3], Jack Whiteley[2]

[1] *Derriford Hospital, Plymouth, United Kingdom*
[2] *University Hospitals Plymouth NHS Trust, United Kingdom*
[3] *DDRC Healthcare, Plymouth, United Kingdom*

Corresponding author: *Dr Alice Palmer, Derriford Hospital, Plymouth, United Kingdom*
ORCiD: *0009-0008-1483-3384*
palmera4808@gmail.com

Keywords
Case reports; Diving medicine; Intensive care medicine; Recompression; Treatment; Venous gas embolism

Abstract

(Palmer A, Holdstock C, Watts D, Stokes RJ, Whiteley J. A case of severe multi-system decompression sickness requiring critical care, aggressive fluid resuscitation and hyperbaric oxygen therapy. Diving and Hyperbaric Medicine. 2026 30 September;56(3):376–380. doi: 10.28920/dhm56.3.376-380. PMID: 42743580.)
An experienced recreational diver developed a severe multisystem life-threatening decompression sickness following a series of eight dives over four days, the profiles of which were all within acceptable limits. The diver had marked haemoconcentration and required large volume intravenous fluid replacement during initial treatment. Similar presentations have previously only been described following deep dives, fast ascents or where mandatory decompression had been missed. The management of this case, including the involvement of a hyperbaric unit co-located to an intensive care department, and the underlying pathophysiology are discussed.

Introduction

Severe multisystemic decompression sickness (DCS) involving cardiopulmonary compromise is rare, affecting < 1% of DCS cases.[1] It typically presents with combinations of hypoxia, hypotension, quadriplegia, rash, shock, coagulopathy and haemoconcentration. DCS of this severity is typically a consequence of deep air dives, rapid uncontrolled ascents, or technical multiday decompression diving.[1]

This report describes a recreational diving incident following an unprovocative dive profile resulting in severe multisystemic DCS associated with capillary leak syndrome. This case highlights the requirement for large volume fluid resuscitation, early recompression, and the ability of the hyperbaric unit to care for an intubated patient in such cases.

Case summary

The patient has fully consented to the reporting of their case.

Diver-X (47-year-old male) was an experienced recreational diver with 355 lifetime dives and instructor qualification. He had a raised body mass index (30 kg·m^{-2}) but no underlying medical conditions or previous diving incidents.

He dived on open circuit using gradient factors of 55/85 over four consecutive days (Table 1). On the final day, he completed two boat dives with a 1.5-hour surface interval. The first (Dive 7) was to a maximum depth of 33 metres of seawater (msw) for 45 minutes breathing Nitrox 31%. A three-minute decompression stop was undertaken at 6 msw and 3 msw. The second dive (Dive 8 – incident dive) was to a maximum depth of 27 msw (average 21 msw) for 35 minutes with a safety stop at 6 msw, resulting in a total dive time of 42 minutes (Figure 1). He was breathing Nitrox 28%.

The dive went as planned with no unusual events. He surfaced with his buddy in a controlled manner, appeared well and was able to exit the water without assistance. Five minutes after this, he reported tingling in his hands and feet. This rapidly progressed, and within 15 minutes he was unresponsive to pain stimuli with laboured breathing, dilated pupils and shaking legs. Surface oxygen was administered by boat crew.

The coastguard facilitated evacuation to the nearest emergency department, arriving 1.5 hours after leaving the water. During transfer, intravenous fluids and antiemetics were administered. Despite normobaric oxygen he continued to deteriorate, developing complete paralysis below the cervical spine by the time of arrival in the emergency department. A provisional diagnosis of severe neurological DCS was made.

Examination by the duty diving physician found truncal cutis marmorata, quadriplegia, focal seizures, and pupillary

Table 1
Summary of eight dives over four consecutive days

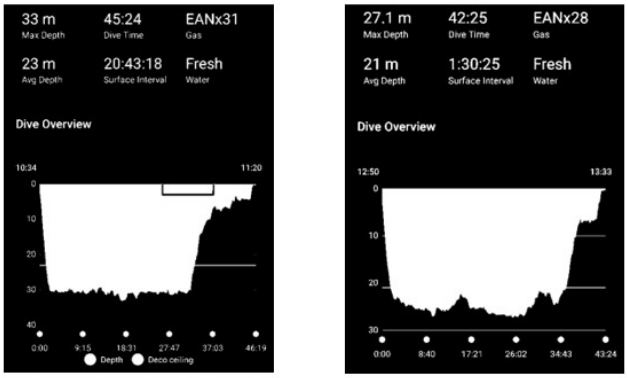
Day	Dive	Max depth (msw)	Dive duration minutes)	Pre-dive surface interval
1	1	21.5	53	–
	2	23.1	48	1 hour 19 mins
2	3	25.0	49	21 hours 12 mins
	4	11.5	49	1 hours 47 mins
3	5	31.8	35	20 hours 41 mins
	6	22.7	53	1 hour 57 mins
4	7	33	45	20 hours 43 mins
	8	27.1	42	1 hour 30 mins

nystagmus bilaterally with right sided pupillary deviation. His airway was patent, achieving saturations of 93% whilst breathing oxygen delivered at 15 L·min^{-1} via a non-rebreather mask with a respiratory rate of 20–25 breaths·min^{-1}. It should be noted that whilst the oxygen delivery was set at 100%, the non-rebreather mask would have provided the patient with an FiO_2 (fraction of inspired oxygen) of around 60-80%. This was the highest percentage that was able to be given, as it is unlikely that with the tachypnoea that a demand valve device would have been tolerated or effective. Consideration of non-invasive ventilation would have likely delayed the requirement for intubation and ventilation, which was ultimately the most effective method of providing 100% oxygen in this patient.

Diver-X was cold peripherally, displaying signs of shock with a heart rate of 125 beats·min^{-1} and blood pressure of 129/107 mmHg. A 12-lead ECG showed sinus tachycardia. A chest x-ray demonstrated bilateral lower-lobe reticular infiltrates but no pneumothorax or mediastinal gas. Further intravenous access was gained, and fluid resuscitation continued. Seizure management involved lorazepam and a levetiracetam infusion. Initial Glasgow Coma Scale (GCS) was 14/15; he was able to identify his location, follow commands and open his eyes to voice. There was no movement below the cervical spine to painful stimuli.

Serial arterial blood gas (ABG) measurements via an arterial line showed significant haemoconcentration with a haematocrit of 68%, haemoglobin (Hb) of 222 g·L^{-1} and a developing lactic acidosis (Table 2). A computed

Figure 1
Profiles of dives 7 and 8, the latter being the incident dive



Dive 7 *Dive 8*

tomography (CT) head and thorax showed significant portal venous gas and mild interstitial oedema at lung bases but no acute intracranial abnormality or evidence of pulmonary barotrauma.

Diver-X continued to deteriorate with progressing bulbar weakness, reduced GCS, tachycardia, hypotension, tachypnoea, and hypoxia with saturations of 89% on high flow oxygen via non-rebreather mask. Due to reduced consciousness, focal seizure activity and risk of airway compromise he was intubated in the emergency department and early intensive care support was sought. Immediately following intubation, he became increasingly tachycardic, hypoxic, and hypotensive, requiring vasopressor support and high FiO_2. He was transferred to the hyperbaric facility by a critical care transfer team. This was a 5-minute journey to an adjacent building via ambulance.

Recompression with a fully extended Royal Navy (RN) Table 62 to 2.8 atmospheres absolute (atm abs) started 5.5 hours after surfacing. The table included 127 minutes at 2.8 atm abs and 300 minutes at 1.9 atm abs. A RN62 table was chosen as it is a treatment table deemed to be effective for neurological decompression sickness whilst reducing the risk to the chamber personnel of mandatory decompression if the patient became unstable and the recompression had to be aborted (for example when compared a heliox table like a Comex 30).

During recompression, Diver-X was significantly fluid deplete and oliguric requiring 9 L of crystalloid fluid resuscitation. His ABG showed only a marginal improvement in haematocrit and Hb levels with a stable lactate (Table2).

Diver-X was transferred to the intensive care unit (ICU). During the first 72 hours, he was increasingly hypoxic despite a high FiO_2, accompanied by an initial increase in noradrenaline requirement which then stabilised. He was laid prone for 48 hours to optimise ventilation and oxygenation. Further hyperbaric treatment was considered within this time however it was deemed to be too high risk given the transfer required by land ambulance (albeit short) and the relative

Table 2
Haematological and biochemical parameters within 12 hours of surfacing; ^^^above detectable limits; atm abs – pressure in atmospheres absolute BE – base excess; Hb –haemoglobin; Hct –haematocrit

Parameter	Hours after surfacing							
	2	4	6	8	9	10	11	12
FiO_2			1.0	1.0	1.0	1.0	1.0	1.0
Atm abs	1.0	1.0	2.8	2.8	1.9	1.9	1.9	1.9
pH	7.35	7.32	7.26	7.30	7.32	7.32	7.37	7.34
PaO_2 kPa mmHg			 ^^^ ^^^	 39.6 297	 53.9 404	 42.5 319		
$PaCO_2$ kPa mmHg	 6.66 50	 5.24 39	 5.96 45	 5.84 44	 5.21 39	 4.85 36	 5.2	
HCO_3^- (mmol·L^{-1})	27.3	20.2	20.1	21.4	20.2	18.6	22	
BE (mmol·L^{-1})			-7	-5	-5.9	-7.5		
Hct	68.0%	65.3%	63.0%	66.0%	63.9%	61.9%		
Hb (g·L^{-1})	222	213	206	215	208	202	201	212
Lactate (mmol·L^{-1})	2.6	2.3	3.0	2.4	2.4	2.6	2.4	2.4

remoteness of a hyperbaric facility off-site from ICU. Whilst it may have been achievable it was felt to be unsafe until the patient had improved clinically.

During early sedation holds Diver-X exhibited poor neurological recovery (only able to stick out tongue on command), prompting re-sedation. A CT head scan showed evolving ischaemic changes, and chest imaging demonstrated the development of aspiration pneumonia with patchy airspace consolidation. He was started on intravenous antibiotics.

After five days in ICU, Diver-X was stable enough to be transferred to the hyperbaric-chamber. He had three repeat treatments with un-extended RN62 tables over days 5, 7 and 9 while sedated and ventilated. Sedation holds during this time showed neurological improvement with some new leg movements.

Successful extubation was achieved on Day 12. Significant neurology persisted with hyperreflexia, hypotonia and low power throughout. Over days 14, 15 and 16 he had three further treatments with RN66 tables. Neurological assessment after each treatment showed improvement until a plateau following seven treatments. At this stage Diver-X was alert and orientated with marked improvement in global power and spontaneous movement in all four limbs. Some residual weakness remained, predominantly in the left lower limb. He was transferred to a stroke rehabilitation ward where physiotherapy continued for 10 weeks.

Magnetic resonance imaging revealed an overall improvement in intracranial appearances and no convincing abnormal cord signal. His clinical improvement mirrored this with increasing strength in all limbs and improving sensation. He began to mobilise using a wheelchair and continued to improve following transfer to a hospital local to his family with intensive physiotherapy input. He was discharged home able to walk independently.

Diver-X was advised to have bubble contrast echocardiogram to screen for a patent foramen ovale (PFO) to investigate underlying risk factors for severe DCS. Although the cardiac centre he attended did not use full diving protocol, they were able to detect a Grade 2 PFO on Valsalva. He does not wish to return to diving.

PATIENT PERSPECTIVE

"*I only remember snippets between getting back on the dive-boat and the emergency room. The dive went to plan, and I didn't feel unwell until I was back on the boat. It took around a week for me to grasp the timeline of events. I still find all a bit surreal with hindsight I really had no idea what was going for the first few days.*

I had no movement on my left and was extremely weak on my right. After a stay on the stroke ward, I was transferred to a neuro-centre closer to family, still being hoisted. I was discharged two weeks later, walking independently, and followed five weeks of daily physiotherapy and rehabilitation at home. I now walk relatively long distances, commute on the tube, regularly swim 500 m and row 2 km daily alongside my daily regime and I'm largely independent. My left side is almost on par with the right, I have not regained full sensation in some of my fingers. I started back at work in the city nine weeks later, I found some of the more complex topics hard to conceptualise to begin with. Cognitively as far as I'm aware there aren't any issues. All in all, I'm in a good place.

Table 3

A summary of haemoconcentration and precipitating incident dives reported in current literature;[5–10] Hb – haemoglobin; Hct –haematocrit; msw –metres of seawater

Reference	Hct	Hb g·L^{-1}	Dive features
5		254	Depth: 110 msw, uncontrolled ascent from 24 msw
6	58%		Depth: 66 msw, 6.5 h omitted decompression
7	63%	209	Depth: 55 msw, uncontrolled ascent from 55msw
8	58%	206	Depth: 50 msw + five deep dives within four days prior
9	65%	221	Dive 1: 54 msw, dive 2: 48 msw, partial omitted decompression
10	55%		Dive 1: 60 msw, dive 2: 40 msw

I think about what happened a lot and what could have been. I owe so much to the staff in ICU and the hyperbaric chamber".

Discussion

In this case, a benign dive profile led to severe, life threatening, multisystem DCS. Multiday diving to within table/computer limits is common practice and yet there are no similar reports of such severe DCS.

The presence of a patent foramen ovale (PFO) was presumably the major risk factor in this case allowing a arterialised venous bubbles to contact saturated tissues, creating a bubble cascade effect throughout the vascular endothelium. Diver-X reported no unusual activity such as excessive Valsalva or exertion during the dive therefore it is unclear why he had such a large bubble cascade on this occasion. Widespread endothelial injury compromising endothelial integrity, and or activation of inflammatory cascades leading to capillary leak syndrome would provide an explanation for the huge fluid depletion seen.

Haemoconcentration demonstrated by raised haematocrit, has been well described in divers and animal research models secondary to vascular leakage due to increased permeability across damaged vascular endothelium.[2–5] In this case the haematocrit and haemoglobin values were high compared to the literature. Cases of DCS of similar severity involving dives to depths of at least 40 msw and including omitted decompression or rapid ascents had an average haematocrit of 59.8% and haemoglobin of 222 g·L^{-1} (Table 3).[6–10]

During the initial treatment, Diver-X received 9 L of crystalloid fluid. Despite the large volume of fluid resuscitation there was only marginal improvement in haematocrit (Table 2), demonstrating potential for a larger fluid resuscitation requirement, a feature consistent with severe DCS described in case studies[6–8,10] and summative literature.[1,11]

Conclusions

This case report demonstrates that life-threatening severe multisystem DCI can occur following benign dive profiles, likely a result of an underlying contributing factor, such as a PFO. The facility to provide early recompression and aggressive fluid resuscitation in ventilated patients is critical. A co-located ICU and hyperbaric unit equipped with suitably trained staff is vital and it is likely that this case of DCI would have proved fatal if this had not been available.

References

1 Mitchell SJ, Bennett MH, Moon RE. Decompression sickness and arterial gas embolism. N Eng J Med. 2022;386:1254–64. doi: 10.1056/NEJMra2116554. PMID: 35353963.

2 Nossum V, Hjelde A, Brubakk AO. Small amounts of venous gas embolism cause delayed impairment of endothelial function and increase polymorphonuclear neutrophil infiltration. Eur J Appl Physiol. 2002;86:209–14. doi: 10.1007/s00421-001-0531-y. PMID: 11990728.

3 Fahlman A, Dromsky DM. Dehydration effects on the risk of severe decompression sickness in a swine model. Aviat Space Environ Med. 2006;77:102–6. PMID: 16491576.

4 Boussuges A, Blanc P, Molenat F, Bergmann E, Sainty JM. Haemoconcentration in neurological decompression illness. Int J Sports Med. 1996;17:351–5. doi: 10.1055/s-2007-972859. PMID: 8858406.

5 Newton HB, Burkart J, Pearl D, Padilla W. Neurological decompression illness and hematocrit: analysis of a consecutive series of 200 recreational scuba divers. Undersea Hyperb Med. 2008;35:99–106. PMID: 18500074.

6 Trytko B, Mitchell S. Extreme survival: a serious technical diving accident. South Pacific Underwater Medicine Society (SPUMS) Journal. 2005;35:23–7. [cited 2026 Mar 20]. Available from: https://dhmjournal.com/images/IndividArticles/35March/Trytko_dhm.35.1.23.27.pdf.

7 Arjomand A, Holm JR, Gerbino AJ. Severe decompression sickness associated with shock and acute respiratory failure. Case Rep Crit Care. 2020;2020:8855060. doi: 10.1155/2020/8855060. PMID: 33204543. PMCID: PMC7661127.

8 Homsy M, Bounds C, Glover M, Castledine B, Martindale T. Case report: Physiological changes due to a rapid ascent from 55 metres underwater. J Intensive Care Soc. 2021;22:88–91. doi: 10.1177/1751143719892789. PMID: 33643438. PMCID: PMC7890754.

9 Gempp E, Lacroix G, Cournac JM, Louge P. Severe capillary leak syndrome after inner ear decompression sickness in a recreational scuba diver. J Emerg Med. 2013;45:70–3. doi: 10.1016/j.jemermed.2012.11.101. PMID: 23602149.

10 Klapa S, Meyne J, Kähler W, Tillmans F, Werr H, Binder A, et al. Decompression illness with hypovolemic shock and neurological failure symptoms after two risky dives: a case report. Physiol Rep. 2017;5(6):e13094. doi: 10.14814/phy2.13094. PMID: 28325788. PMCID: PMC5371546.

11 Mitchell SJ. Decompression illness: a comprehensive overview. Diving Hyperb Med. 2024;54(1 Supp):1–53. doi: 10.28920/dhm53.4.supp.1-53. PMID: 38537300. PMCID: PMC11168797.

Conflicts of interest and funding: nil

Submitted: 21 March 2026
Accepted after revision: 16 May 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

Decompression sickness or ciguatera intoxication: a case report

Robert A van Hulst[1]

[1] *Departments of Hyperbaric Medicine and Anesthesiology, Academic Medical Centre, University of Amsterdam, The Netherlands*

Corresponding author: *Professor Robert van Hulst, Departments of Hyperbaric Medicine and Anesthesiology, Academic Medical Centre, University of Amsterdam, Meibergdreef 9, 1100 DD, Amsterdam, the Netherlands*
ORCiD: *0009-0002-6945-5508*
r.a.vanhulst@amsterdamumc.nl

Keywords
Decompression sickness; Ecology; Fish; Intoxication; Neurology

Abstract

(van Hulst RA. Decompression sickness or ciguatera intoxication: a case report. Diving and Hyperbaric Medicine. 2026 30 September;56(3):381–383. doi: 10.28920/dhm56.3.381-383. PMID: 42743581.)
This report presents the case of a diver who suffered from ciguatera intoxication (CTX). After three days of consecutive diving, he arrived at hospital with symptoms of chest pain, shortness of breath, nausea, and had sensory abnormalities across his body, arms and legs. Although his clinical signs and symptoms may have suggested cardiac involvement or decompression sickness (DCS), ultimately his condition was caused by CTX after eating fish. This case illustrates the similarities between DCS and food intoxication. The common pathology of CTX is described and its differential diagnosis with DCS is discussed.

Introduction

Decompression sickness (DCS) can present with a large variety of clinical signs and symptoms, for example, joint pain, skin marbling, cardiorespiratory, and neurological deficits.[1] Decompression sickness treatment is performed based on the diver's history of dive time, dive depth, and the latency of presenting symptoms.

This report describes a case of an experienced diver who presented with cardiac symptoms, gastro-intestinal complaints, and subtle neurological signs after three days of consecutive diving. Based on his additional medical history, as well as medical and neurological evaluation, it was finally concluded that he had a ciguatera intoxication (CTX). This diagnosis was made by *par exclusion*. This report discusses the importance of having a broad differential diagnosis in a diver with subtle neurological signs.

Case report

The diver provided written consent for publication of his case details.

An experienced 63-year-old male diver (> 200 dives) attended the emergency room (ER) in Bonaire with chest pain, shortness of breath, gastrointestinal complaints, nausea and vomiting, and tingling in his arms and legs. In the past three days, he had completed six dives, each lasting a maximum of 50 minutes, with a minimum of an hour surface interval between each dive. All dive times were less than 30 minutes at a maximum depth of 24 m, with slow ascents and safety stops at 3–5 m for 5–10 minutes.

At 17:00, he finished his final dive and then at 18:00 went to a restaurant with his family. At 19:00, after eating his fish dish, the diver experienced nausea and vomiting, along with shortness of breath. Shortly after, the family decided to go to the hospital as he was experiencing some weakness and tingling in his chest, arms, and legs.

At 20:00 he attended the ER and had a heartrate of 102 beats·min^{-1}, his breathing rate was 24 breaths·min^{-1}, oxygen saturation 98%, ECG was normal, and a chest X-ray was also normal. In addition, his troponins were normal, and in his repeated measurements three hours later, his blood pressure was 150/95, and temperature 37.1°C. The initial physical examination showed the patient to be in no acute stress, he was alert and oriented to time, place, and person. There were no cardiac or neurological disorders, or prescription/over-the-counter medications present in his health history. Physical examination of his heart and lungs was normal.

Neurological examination revealed normal cranial nerves, symmetrical reflexes in the extremities, and strength in the arms and legs was normal. His coordination was normal, though there were subjective sensory abnormalities across his body, arms, legs, and lower abdominal region and these changes were not in a dermatomal distribution. More specifically, he had temperature related dysesthesias that

were more pronounced in the hands and feet. When exposed to cold water his hands and feet experienced an unpleasant burning sensation.

After a thorough examination and based on his dysesthesias, the patient was determined to have a mild CTX poisoning due to eating a barracuda steak. Decompression sickness was reasonably ruled out given his conservative dives and he was treated symptomatically with intravenous fluids and anti-emetics. During his 24-hour stay at the hospital, he slowly recovered from his symptoms and made the decision to return to his island apartment prior to departing for the USA.

Lab testing for CTX is complicated as it uses a bioassay designed Neuro-2a assay, and this was not available in the island's hospital.[2]

Discussion

Neurological signs and symptoms presenting acutely after diving are most likely manifestations of DCS and require oxygen and hyperbaric treatment.[1] However, neurological diseases and neurological DCS can share similarities in their presentations and thus may be mistaken for one another.[3–5] After reviewing the literature, this appears to be the first case report to consider a diver with the diagnostic dilemma of distinguishing between DCS and CTX. However, a previous publication presented a case series of eight divers who had CTX while diving from a liveaboard. This group of divers presented a variety of signs and symptoms that lasted from days to weeks. The authors warned that divers should be aware of ciguatera and the similarity of symptoms of DCS.[6]

Ciguatera fish poisoning is the world's most frequently reported seafood poisoning, with around 50,000 cases reported annually.[7] Ciguatera is a neurotoxin and unaffected by heat, freeze drying, or gastric acid. The presence of this toxin is not detected by taste or appearance when consuming fish.[8] The toxins are potent, lipophilic sodium channel activators that bind to the voltage sensitive sodium channel on the cell membranes of all excitable tissues.[9]

The full syndrome of CTX involves gastro-intestinal, neurological musculoskeletal, dermatological, and psychological symptoms.[9] Neurological features may include peripheral sensory or motor symptoms, while central symptoms may present as severe headache or autonomic features. At the onset, the most prevalent symptoms were paraesthesia, dysesthesia, myalgia, and arthalgia.[10] The paraesthesia may last for a minimum of several days and in severe cases, can persist for many weeks. Ciguatoxic dysesthesia is classically referred to as a reversal of thermal sensation. According to reports cited here, cold or room-temperature objects cause a burning sensation that is unpleasant when touched, while warm objects are described by patients as being ice-burned, cold, or chilled.[9,10]

Treatment of CTX is symptomatic. In the old literature, hyperosmotic mannitol infusions were advised, although the pharmacological basis for use remains speculative. The mannitol effect is thought to be due to osmotic reduction on neuronal oedema and a scavenger property of the molecule has been suggested.[9] However, other small case studies have suggested that this approach may not be particularly efficacious, and a double blind randomised controlled trial found no significant differences in the mannitol group compared to the control population.[11] More recently, other studies disagreed with the data collection and methods in the latter clinical trial and thereby questioned their conclusions; thus the use of mannitol to treat CTX symptoms perhaps remains equivocal.[12,13]

Informal conversations with chefs from restaurants on the island of Bonaire revealed a belief that large barracudas (> 90 cm) had a higher chance of being poisoned with CTX. In contrast, researchers in French Polynesia found that fish size is not an effective indicator of fish toxicity.[14]

This case report has some limitations; first of all, due to the lack of a CTX bioassay kit on the island it was not possible to confirm the intoxication by means of a lab test. Secondly, it was only possible to observe the patient for 36 hours because he decided to return to the USA with his mild complaints. In the case presented here, it is concludes that the diver had CTX based on his health status before the dive, his conservative dive-profiles, and exclusion of other cardiac and neurological diseases. Further indictors include his neurological complaints following his history of eating fish and his response to conservative therapy. Physicians treating divers must be alert to subjects' dive history and in addition, also include recent dietary intake in their history-taking questions.

References

1 Mitchell SJ, Bennett MH, Moon RE. Decompression sickness and arterial gas embolism. N Eng J Med. 2022;386:1254–64. doi: 10.1056/NEJMra2116554. PMID:35353963.

2 Loeffler CR, Spielmeyer A. Faster ciguatoxin extraction methods for toxicity screening. Sci Rep. 2024;14:21715. doi: 10.1038/s41598-024-72708-1. PMID: 39289443. PMCID: PMC11408646.

3 Butler FK Jr, Pinto CV. Progressive ulnary palsy as a late complication of decompression sickness. Ann Emerg Med. 1986;15:738–41. doi: 10.1016/s0196-0644(86)80439-5. PMID: 3706868.

4 Jan MH, Jankosky CJ. Multiple sclerosis presenting a neurological decompression sickness in a US Navy diver. Aviation Space and Environ Med. 2003;74:184–6. PMID: 12602452.

5 van Hulst RA, van der Kamp W. Decompression sickness in a vegetarian diver: are vegetarian divers at risk? A case report. Undersea Hyperb Med. 2010;37:181–4. PMID: 20568548.

6 Adams MJ. An outbreak of ciguatera poisoning in a group of scuba divers. Journal of Wilderness Medicine. 1993;4:304–11. doi: 10.1580/0953-9859-4.3.304.

7 Traylor J, Murray BP, Singhal M. Ciguatera toxicity. [Updated 2024 Mar 19]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 Jan. PMID: 29494117.
8 Strickler J, Kellish A. Seafood poisoning. Nursing 2024;54(8):34–7. doi: 10.1097/NSG0041. PMID: 39051956.
9 Pearn J. Neurology of ciguatera. J Neurol Neurosurg Psychiatry 2001;70:4–8. doi: 10.1136/jnnp.70.1.4. PMID: 11118239. PMCID: PMC1763481.
10 Chateau-Degat ML, Beuter A, Vauterin G, Nguyen NL, Chinain M, Darius T, et al. Neurological signs of ciguatera disease: evidence of their persistence. Am J Trop Med Hyg. 2007;77:1170–75. PMID: 18165542.
11 Schnorf H, Taurarii M, Cundy T. Ciguatera fish poisoning: a double-blind randomized trial of mannitol therapy. Neurology. 2002;58:873–80. doi: 10.1212/wnl58.6.873. PMID:11914401.
12 Mullins ME, Hoffman RS. Is mannitol the treatment of choice for patients with ciguatera fish poisoning? Clin Toxicol (Phila). 2017;55:947–55. doi: 10.1080/15563650.2017.1327664. PMID: 28535116.
13 Friedman MA, Fernadez M, Backer LC, Dickey RW, Bernstein J, et al. An updated review of ciguatera fish poisoning: clinical, epidemiological, environmental, and public health management. Mar drugs. 2017;15:72. doi: 10.3390/md15030072. PMID: 28335428.
14 Gaboriau M, Ponton D, Daruis HT, Chinain M. Ciguatera fish toxicity in French Polynesia: size does not always matter. Toxicon. 2014;84:41–50. doi: 10.1016/j.toxicon.2014.03.006. PMID: 24699216.

Conflicts of interest and funding: nil

Submitted: 9 March 2026
Accepted after revision: 30 May 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

In-water recompression for severe decompression illness in competitive freediving: a case report

Jake Johnston[1], Juan Valdivia[2,3], Leigh Baker[4], Kelly Heape[1,5]

[1] *Department of Anesthesiology, Pharmacology and Therapeutics, Faculty of Medicine, University of British Columbia, Vancouver, BC, Canada*
[2] *Department of Neurosurgery, St. Joseph's Hospital, Tampa, FL, USA*
[3] *International Association for Development of Apnea (AIDA International), Medical and Science Committee, Zurich, Switzerland*
[4] *Virginia Mason Franciscan Health, Seattle, WA, USA*
[5] *Department of Anesthesiology and Perioperative Medicine, Royal Columbian Hospital, New Westminster, BC, Canada*

Corresponding author: *Dr Jake Johnston, Department of Anesthesiology, Pharmacology and Therapeutics, University of British Columbia, Room 11228, 11th Floor, 2775 Laurel Street, Vancouver, BC, Canada V5Z 1M9*
ORCiD: *0000-0003-3847-9216*
jakejohnstonmd@gmail.com

Keywords
Arterial gas embolism; Breath-hold diving; Decompression sickness; Diving medicine; Hyperbaric oxygen; Remote locations; Risk assessment

Abstract
(Johnston J, Valdivia J, Baker L, Heape K. In-water recompression for severe decompression illness in competitive freediving: a case report. Diving and Hyperbaric Medicine. 2026 30 September;56(3):384–387. doi: 10.28920/dhm56.3.384-387. PMID: 42743582.)
Introduction: Decompression illness (DCI) is a recognised risk of freediving. When timely hyperbaric oxygen therapy (HBOT) is unavailable, oxygen-based in-water recompression (IWR) may be considered.
Case report: A 49-year-old competitive freediver developed severe neurological DCI within minutes of surfacing from a dive to 117 metres of seawater at a sanctioned competition, with altered mental status, right facial droop, right hemiparesis and aphasia. With an anticipated 24–48 h delay to HBOT, emergency IWR was initiated with a full-face mask and 100% oxygen. Incremental ascent from 18 msw over 14.5 min produced substantial recovery. Residual gait instability and pronator drift prompted a second oxygen-IWR 5.5 h later from 15 msw over 41 min, achieving complete resolution.
Discussion: While this case had an excellent outcome, IWR carries significant risks including oxygen toxicity and drowning, and review identified serious safety and ethical failures. Patient selection is key; perceived benefit must be weighed carefully against risk.
Conclusions: IWR may be considered for DCI when HBOT is not promptly accessible. No IWR guidelines exist specifically for freedivers. The success of this case does not establish that deeper exposures were necessary or superior to shallower protocols. IWR is high-risk, requiring proper selection, trained personnel and favorable conditions.

Introduction

Decompression illness (DCI) encompasses decompression sickness (DCS) and arterial gas embolism (AGE).[1] It is an increasingly recognised complication of freediving, particularly after repeated dives or single dives beyond 100 metres of seawater (msw).[2–4] Definitive treatment is hyperbaric oxygen therapy (HBOT),[5] which is often unavailable within a clinically useful timeframe from the remote locations where deep freediving competitions are held.

In-water recompression (IWR), returning a symptomatic diver to depth to breathe oxygen on a defined schedule, has been used when timely HBOT is not feasible.[5,6] Expert guidance supports oxygen-IWR for moderate-to-severe or progressive DCS where trained personnel, equipment and conditions permit, and lists altered consciousness, vertigo, vomiting, shock and physical incapacity among contraindications.[5] The principal published schedules, the Australian and US Navy protocols, recompress to 9 msw.[6,7] Both derive from compressed-gas diving; none is validated for freediving. Appropriate patient selection is therefore a paradox: those with significant DCI most likely to benefit from very early recompression may also be riskiest to manage underwater.

A recent case series described 13 DCI incidents among six competitive freedivers, six treated with IWR at 5–25 msw with no protocol consistency. Only one was medically supervised, the rest self-diagnosed and self-treated. The authors concluded that freediving-specific IWR guidance should be developed.[8]

Table 1

Timeline of events, clinical findings and interventions; time is given from surfacing. CWTB – constant weight with bi-fins; DCI – decompression illness; IWR – in-water recompression; NRB – non-rebreather mask

Time (h:min)	Event and findings	Intervention
0:00	Surfaced from 117 msw CWTB dive; initially well	On-site medical oversight available
0:08	Began breathing oxygen at 3 msw (standard practice after dives deeper than 80 msw)	100% oxygen by second-stage regulator at 3 msw
0:10	Difficulty maintaining position on line; signalled distress	Assisted to beach by safety and medical team
0:14	Unresponsive to voice; right facial droop; right hemiparesis; aphasia	High-flow oxygen by NRB at > 15 L·min^{-1}; severe neurological DCI suspected
0:15–0:32	Emergency oxygen-IWR #1; improved mentation within one minute at 18 msw	100% oxygen via full-face mask; 18 msw with incremental ascent over 14.5 min
0:32–0:48	Post-IWR #1 on surface	100% oxygen via NRB at 15 L·min^{-1}
0:48	Ambulated with assistance to medical vehicle; vital signs stable	Monitoring and supportive care
1:10–1:20	Fingertip paresthesia; abnormal sensation right leg	Re-examination by neurosurgeon: paresis and aphasia resolved; residual gait instability and right pronator drift
6:20	Non-emergency oxygen-IWR #2	100% oxygen by second-stage regulator; 15 msw with incremental ascent over 41 min
7:05	Complete resolution of pronator drift and gait instability; vital signs stable	Ongoing monitoring

We report a physician-supervised case of severe neurological DCI treated with staged oxygen-IWR at a sanctioned competition, describing the decision-making, schedules, outcome, and the failures identified on review.

Case report

The patient provided written informed consent for anonymous publication of his clinical details.

A 49-year-old male competitive freediver (172 cm, 67 kg) presented with acute neurological symptoms after a competition dive. He was a non-smoker with no medical comorbidities or medications. He had a prior history of DCI self-treated with IWR, and no baseline neurological deficit.

The incident occurred on day six of a competitive diving program. Seas were calm, visibility exceeded 20 m and water temperature was 29°C. His competition dives were: day one, constant weight (CWT) 111 msw (3:04 min:s); day two, constant weight with bi-fins (CWTB) 108 msw (3:08); day three, rest; day four, CWTB 114 msw (3:18); day five, rest; day six, CWTB 117 msw (3:23), the incident dive. All prior dives were completed successfully without lung squeeze or DCI.

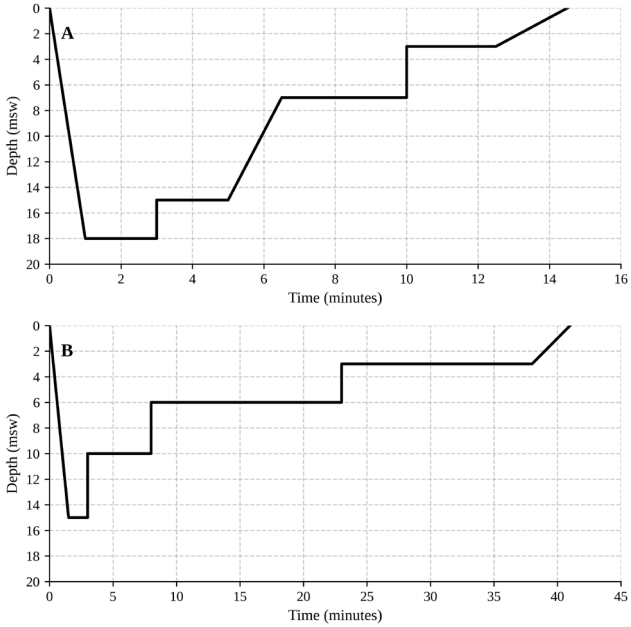
Table 1 summarises the timeline. On surfacing at 10:40 he was alert and oriented without focal deficit, self-extracted onto the platform, and examined normally. He conversed in full sentences with normal speech and no facial asymmetry, showed intact fine motor function, and ambulated normally. He was not cyanotic and there was no clinical evidence of hypoxaemia. He later reported right upper limb paresthesia beginning three to four minutes after leaving the water, which he did not disclose at the time.

At 10:48 he moved to an underwater line to breathe 100% oxygen at 3 msw for five minutes, routine practice after dives deeper than 80 msw. He swam there unaided, was handed to the in-water medic, and descended without difficulty. At approximately 10:50, after two minutes breathing oxygen, he was seen struggling to hold position and signaling distress. He was recovered to the beach by safety divers, placed supine at the water's edge, and given oxygen by non-rebreather mask at > 15 L·min^{-1}. By 10:54 he was unresponsive to loud voice with right facial droop, right upper and lower limb hemiparesis and aphasia. Severe neurological DCI was suspected.

Given rapidly evolving focal deficits and an anticipated 24–48 h delay to a chamber, the medical team, safety diving team and event organiser jointly decided on emergency oxygen-IWR. The patient could not consent because of his altered mental status. He was known to both teams and had openly advocated IWR in emergencies. The decision rested on the high probability of permanent deficit or death given the delay, and his previously expressed wishes.

On-site personnel included certified scuba divers, an anesthesiologist/intensivist, a neurosurgeon, a respiratory therapist and a paramedic with equipment for advanced resuscitation. Safety divers capable of repeated 18 msw

Figure 1
In-water recompression (IWR) depth profiles; depth scales are identical, time scales differ. (A) Emergency oxygen-IWR following the incident dive, total duration 14.5 min. (B) Second, non-emergent IWR performed approximately 5.5 h later, total duration 41 min



breath-hold dives observed the patient. An escorting scuba diver accompanied the patient throughout, breathing 100% oxygen because no air cylinder was available.

The first IWR ran from 10:55 to 11:12. A full-face mask (Guardian, Ocean Technology Systems, Santa Ana, CA) delivering 100% oxygen was fitted, allowing facial visualisation. Safety divers floated him the short distance from shore to the decompression line with continuous hands-on support and dived him to 18 msw. He then ascended incrementally to the surface over 14.5 min, head-up throughout (Figure 1A). Within a minute at 18 msw he was audibly humming on exhalation, could grip the line, and later reported feeling "*a lot better straight away*".

He resumed oxygen by non-rebreather at 15 L·min^{-1}. At 11:28 he walked with assistance to the medical vehicle; vital signs were stable (SpO_2 100%, blood pressure 125/73 mmHg, respiratory rate 14 breaths·min^{-1}, heart rate 55 beats·min^{-1}). At 11:50 he described fingertip paresthesia and an abnormal sensation in the right leg. Examination at 12:00 showed resolution of hemiparesis and aphasia but residual gait instability and right pronator drift. Now competent, he declined evacuation despite strong recommendation and requested a second IWR.

After verbal consent and a time-out, a second oxygen-IWR was performed at 17:00 under physician supervision with two alternating safety divers. He self-descended to 15 msw and ascended incrementally over 41 min (Figure 1B), head-down, breathing oxygen by second-stage regulator. Pronator drift and gait instability resolved completely, with no oxygen-related complications. Re-examination after this session found him at his neurological baseline, with complete resolution of all symptoms. He was withdrawn from the competition and advised not to dive until assessed by a diving physician.

Discussion

A left hemisphere syndrome with dense right-sided deficits evolved over 14 minutes after surfacing, improved promptly with IWR and resolved entirely after a second session. This lateralised cortical presentation suggested AGE. In breath-hold diving this has been proposed to occur without pulmonary barotrauma, through right-to-left shunting of decompression-derived bubbles across a patent foramen ovale or intrapulmonary anastomoses, though the pathophysiology remains debated.[9]

No consensus defines a 'reasonable timeframe' to HBOT before consideration of IWR; clinical deterioration is suggested beyond delays of two to six hours,[6,10] while the US Navy Diving Manual considers IWR when anticipated chamber delays exceed 12–24 h.[7] This patient met several prerequisites for attempting IWR: severe, rapidly evolving deficits; prolonged anticipated delay to HBOT; warm calm water with established safety infrastructure; full-face oxygen delivery; and personnel able to observe continuously and abort. Nonetheless, the presentation included altered consciousness and physical incapacity, both plausible contraindications due to drowning risk. Mitigation comprised a full-face mask, continuous hands-on control, escorting safety divers and physician supervision, but a full-face mask neither guarantees prevention of aspiration nor permits assisted ventilation should breathing cease. Taking an incapacitated patient underwater remains high-risk and requires extreme caution. IWR also trades the controlled conditions of a chamber for immediacy, defensible only where the benefit of early recompression clearly outweighs the added hazard.

The first schedule began at 18 msw, deeper than the 9 msw of commonly cited protocols;[6,7] the second at 15 msw. Both were selected pragmatically. Reasoning from Boyle's law, the team expected the greater ambient pressure at 18 msw to produce greater bubble volume reduction than a 9 msw schedule, and judged that this might promote faster reversal of a presumed cerebral arterial gas embolism. The 18 msw schedule was devised by the event organiser and had reportedly been used in unpublished freediving cases; the second reflected milder residual symptoms and stability.

The outcome was excellent, but recovery does not establish that the deeper exposures were necessary, safer or superior to established shallower protocols. Optimal IWR strategy for freediving is unknown; reported depths span 5–25 msw with no consistency between protocols.[8] A deeper schedule produces greater bubble volume reduction and may promote

more rapid redistribution of bubbles occupying vessels on the arterial side of the cerebral microcirculation, though this is unproven. These potential benefits come at the cost of a greater risk of oxygen toxicity seizure and consequent drowning, plus, because of the greater distance to the surface, a longer ascent to reach the surface medical team should IWR need to be aborted. The risk of an oxygen toxicity seizure is substantially greater in water than in a dry chamber, where a convulsion can be managed without risk of drowning, and an irritable injured brain probably adds further to that risk. To be clear, we are not recommending the tables used in this case over published alternatives; we share them to add to a sparse literature.[11,12]

Review identified several failures. There was no pre-established IWR plan despite the known delay. The escorting diver breathed 100% oxygen at 18 msw because compressed air was unavailable, an avoidable toxicity risk in a diver managing a patient; escorting divers should breathe compressed air. This was a major shortcoming. A full-face mask was not used for the second session; now competent, the patient chose a mouthpiece for comfort and familiarity. In retrospect we would recommend a full-face mask for both sessions, since it offers better airway protection should an oxygen toxicity seizure occur. Treatment proceeded without consent in a patient unable to give it, justified as necessary to prevent death or serious harm, but this remains a concern a favorable outcome does not resolve.

For future remote events, improvements include pre-emptive IWR discussion and protocol selection with athletes and staff, a possible pre-consent form where evacuation is not timely, equipment checks, role assignment and abort criteria. We recommend a full-face mask and a pre-procedure timeout before any attempt at IWR. An aspirational goal for teams intending to use IWR is specific training in the use of 100% oxygen in water, together with rehearsal of IWR scenarios; preparation of this kind would avoid situations such as the unavailability of compressed air for the escorting diver that occurred here. Documented, supervised cases remain rare; most reported freediving IWR is self-administered without oversight.[8] We include our shortcomings to help others prepare better and avoid making the same mistakes.

Conclusions

Severe neurological DCI can occur in competitive freediving, often far from hyperbaric facilities. Where timely transport to HBOT is not possible, oxygen-IWR may be considered in carefully selected cases. Here, complete recovery followed two physician-supervised IWR sessions from 18 msw and 15 msw, though this does not establish that deeper exposures were necessary or superior. IWR remains high-risk, warranted only when anticipated benefits clearly outweigh risks, with proper training, equipment and safety protocols in place. This case adds to the limited literature on IWR and provides insight into its potential application in competitive freediving emergencies.

References

1 Mitchell SJ, Bennett MH, Moon RE. Decompression sickness and arterial gas embolism. N Engl J Med. 2022;386:1254–64. doi: 10.1056/NEJMra2116554. PMID: 35353963.

2 Blogg SL, Tillmans F, Lindholm P. The risk of decompression illness in breath-hold divers: a systematic review. Diving Hyperb Med. 2023;53:31–41. doi: 10.28920/dhm53.1.31-41. PMID: 36966520. PMCID: PMC10106275.

3 Fitz-Clarke JR. Risk of decompression sickness in extreme human breath-hold diving. Undersea Hyperb Med. 2009;36:83–91. PMID: 19462748.

4 Tetzlaff K, Schöppenthau H, Schipke JD. Risk of neurological insult in competitive deep breath-hold diving. Int J Sports Physiol Perform. 2017;12:268–71. doi: 10.1123/ijspp.2016-0042. PMID: 27248853.

5 Mitchell SJ, Bennett MH, Bryson P, Butler FK, Doolette DJ, Holm JR, et al. Pre-hospital management of decompression illness: expert review of key principles and controversies. Diving Hyperb Med. 2018;48:45–55. doi: 10.28920/dhm48.1.45-55. PMID: 29557102. PMCID: PMC6467826.

6 Doolette DJ, Mitchell SJ. In-water recompression. Diving Hyperb Med. 2018;48:84–95. doi: 10.28920/dhm48.2.84-95. PMID: 29888380. PMCID: PMC6156824.

7 Naval Sea Systems Command. U.S. Navy diving manual, revision 7. SS521-AG-PRO-010. Washington (DC): U.S. Navy; 2016 Dec 1. [cited 2026 Jul 19]. Available from: https://www.navsea.navy.mil/Portals/103/Documents/SUPSALV/Diving/US%20DIVING%20MANUAL_REV7.pdf.

8 Lin N, Yu E, Lussier A, Gouin E, Lindholm P. Use of in-water recompression for decompression illness after deep freediving: a case series. Diving Hyperb Med. 2025;55:376–83. doi: 10.28920/dhm55.4.376-383. PMID: 41364861. PMCID: PMC12831604.

9 Kohshi K, Denoble PJ, Tamaki H, Morimatsu Y, Ishitake T, Lemaître F. Decompression illness in repetitive breath-hold diving: why ischemic lesions involve the brain? Front Physiol. 2021;12:711850. doi: 10.3389/fphys.2021.711850. PMID: 34539434. PMCID: PMC8446421.

10 Blatteau JE, 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.

11 Pyle RL, Youngblood D. In-water recompression as an emergency field treatment of decompression illness. SPUMS Journal. 1997;27:154–69. [cited 2026 Jul 19]. Available from: https://www.dhmjournal.com/images/IndividArticles/27Sept/Pyle_SPUMSJ.27.3.154-169.pdf.

12 Edmonds C. Underwater oxygen treatment of decompression sickness. SPUMS Journal. 1995;25:122–8. [cited 2026 Jul 19]. Available from: https://dhmjournal.com/images/IndividArticles/25Sept/Edmonds_SPUMSJ.25.3.122-128.pdf.

Conflicts of interest and funding: nil

Submitted: 26 July 2026
Accepted after revision: 10 August 2026

Copyright: This article is the copyright of the authors who grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in electronic and other forms.

Erratum

Measuring whole-body inert gas uptake and washout during submersion

Oscar Plogmark, Kristian Soltesz, Carl Hjelte, Oskar Frånberg

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.

An error was identified in Figure 3 of the published article. The images in the figure were incorrectly placed, resulting in the same image being shown for each part of the figure.

The online version of the article has been corrected, and the corrected figure will also be included in the version deposited in PubMed Central. However, three versions of the article had already been distributed before the error was identified and cannot be corrected retrospectively.

The error is limited to Figure 3 and does not affect the text, data, results or conclusions of the article.

The journal apologises for the error.

doi: 10.28920/dhm56.3.388.
PMID: 42743583.

Nicky Telles
Editorial Manager

Diving and Hyperbaric Medicine Journal copyright statement 2026

All articles in *Diving and Hyperbaric Medicine* are published under licence from the authors. Copyright to these articles remains with these authors. Any distribution, apart from for limited educational purposes, is in breach of copyright.

HBOE
HBOEvidence

HBOEvidence

HBOEvidence is seeking an interested person/group to continue the HBOEvidence site. The database of randomised controlled trials in diving and hyperbaric medicine. The HBOEvidence is in the process of being integrated into the SPUMS website.

Those interested in participating in this project can contact:
Stephan Roehr president@spums.org.au

Back issues of DHM

After a one-year embargo, individual articles from *Diving and Hyperbaric Medicine* are freely available on our website https://www.dhmjournal.com/index.php/full-journals-embargoed/full-journals

They are also available on PubMed Central as full articles after one year embargo dating back to 2017. These are searchable via their doi, PMID or PMCID number.

Embargoed articles are available via the DHM website for single use purchase.
Please follow the link below if you would like more information:
https://www.dhmjournal.com/index.php/purchase-single-articles

Or email Nicky Telles our Editorial Manager:
editorialassist@dhmjournal.com

DHM Journal Facebook

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

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

Welcome all, as I continue to settle into the role of SPUMS President capably assisted by my trusted ExCom members without any regrets so far.

A few surprise emails, and business propositions potentially targeted at SPUMS after searching for 'Bubbles and Docs' landing in the presidential inbox provided some light-hearted relief on occasions and expedited filing in the SPAM filing cabinet.

Having said that, national and international collaboration will be the way forward for us and I am delighted that we have been cordially approached via Dr Phil Bryson Vice President UHMS and the new UHMS President Dr Sandra Wainwright to explore and facilitate a closer collaboration and sharing of ideas and experiences and missions as societies and experts in the field. Stronger together it shall be.

I am reliably informed that the August AHDMA ASM in Singapore again was as expected a well-attended and successful meeting with sizeable SPUMS input and commitment (Thank you Simon Mitchell!). With a fair wind I would very much like to attend next year's meeting in person.

In early August Lizzie Elliot and Ian Gawthrope attended and lectured at the recent DAN/UHMS Fall 2026 CME meeting in the Philippines, many thanks for the hard work and time spent in addition to doing a recon for the 2027 SPUMS ASM! On that note: A definite date entry must for the calendar "*Sunday 16 May to Saturday 22 May 2027 SPUMS ASM Dumaguete, Philippines*". The theme is "*Innovations in Diving and Hyperbaric Medicine*" and there will be a position Statement workshop: "*Mental Health and neurodevelopmental factors relevant to recreational fitness to dive assessment*".

Preparations are progressing well, and the registration will open shortly, please check the SPUMS website for up-to-date information. The Convenors Lizzie Elliot and Cathy Meehan have already arranged for a very impressive lineup of Keynote and expert speakers and a great underwater workshop itinerary. Dive this Space!

Another exciting meeting will be the EUBS 50th anniversary Annual Scientific meeting in Geneva, Switzerland, 14–18 September. Regrettably, I will not be able to go but I am reassured by the sizeable portion of ExCom members attending. Any questions or suggestions or collaboration requests or ideas please feel free to find them either at the conference or at the social functions that I am sure they will attend as worthy SPUMS Flagbearers and representatives (I know who you are.) and start a conversation.

I myself am looking forward to attending the 33rd HTNA ASM "*Safety, it doesn't' Happen by Accident*" Fremantle WA this 28–30 October 2026, where there might be a change of the old guard I've been told and will keep you posted.

Fremantle again, The ANZHMG Introductory Couse in Diving and Hyperbaric Medicine course in Fremantle 2027 has moved from its traditional February slot to an 26 April to 7 May 2027 window, again anyone interested should engage early as it always oversubscribed and a waitlist in place (https://spums.au/index.php/education/spums-approved-courses-for-doctors).

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

The DHM Journal continues to go from strength to strength, and I would like to acknowledge the tireless efforts of Nicky Telles our Editorial and Web manager, Simon Mitchell Editor and all of the ExCom for their hard work and support. Such consistent and high-quality work does not go unnoticed.

Wishing you all a safe and happy Spring or Autum on either side of the planet.

Dr Stephan Roehr
SPUMS President

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*

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.

SPUMS Facebook page
Find us at:
SPUMS on Facebook

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



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


SPUMS
South Pacific Underwater Medicine Society
SAVE THE DATE.
SPUMS ASM
16th–22nd May 2027
Atmosphere Resorts & Spa
Dumaguete, Philippines
Theme:
Innovations in Diving and Hyperbaric Medicine.
With Position Statement Workshop:
Mental health and neurodevelopmental factors relevant to recreational fitness to dive assessment.
Keynote Speakers:
Dr Virginie Papadopoulou & Dr Calina Ouliaris
Guest Speakers:
Dr Bram Querido & Dr Thijs Wingelaar
Convenors:
Dr Lizzie Elliott & Dr Catherine Meehan
Please send EOI for talks or poster presentations to
convenor@spums.org.au

Notices and news

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

EUBS Member-at-Large elections

The EUBS Elections again held electronically, using the ElectionRunner software. 50% of our members voted, which is above average compared to previous years – however it could still be improved.

We had to elect a Member-at-Large for the four-year period 2026–2030. There were two candidates: Dr Pedro Barata from Portugal and Dr Evangelos Papoutsidakis from Spain. The candidate who scored best was Dr Evangelos Papoutsidakis , and he will become the Member-at-Large 2026; he will take office as from our General Assembly on 18 September. We will be saying goodbye to our 2022 Member-at-Large, Dr Anne Räisänen-Sokolowski, for having served faithfully on the ExCom for four years. We thank Anne for her efforts for our Society and hope be able to count on her in the future.

Thanks to all EUBS members who have voted, and please, if you have any comments on the voting process or software used, send us an e-mail (secretary@eubs.org).

EUBS2026 Annual Scientific Meeting, Geneva, Switzerland, from 14–18 September 2026

As this issue of Diving and Hyperbaric Medicine Journal is published, we will have had the pleasure to unite again in Geneva, from 14–18 September 2026. While this text is written before the meeting, we are certain it will have been a great pleasure to see our friends again, and we are confident that the 50th Annual Scientific Meeting of EUBS will have been a great success.

During this meeting, another very important event will be happening – actually, two:

- We will hold the 11th ECHM Consensus Conference on the Indications for HBO therapy. The European Committee for Hyperbaric Medicine (ECHM) has in its objectives the continuous improvement in the quality of care and the safety in hyperbaric medicine. One of the tools used to reach this target is the organisation of consensus conference to issue widely accepted guidelines. Three such consensus conferences have been organised in 1994, 2004 and 2016. Ten years after the last one, it was now time to review and update these guidelines according to the advance in medical knowledge and the experience gained in clinical practice during that period. In 1994, the guidelines were elaborated by a jury from expert reports and discussion with the conference audience. In 2004, report of the guidelines was improved in grading the recommendations both by the level of evidence supporting the recommendation and the importance for practice of that recommendation. In 2016, ECHM reported not only recommendations with their confidence level but also the evidence supporting the recommendation and how confident jury members are in that recommendation (GRADE method). At the forthcoming edition, ECHM will update the list by asking selected well recognized experts in each field to update a report on a topic with an exhaustive literature survey, a synthesis of the evidence and a proposal for recommendations on that topic with inclusion of the 'PICO for synthesis' method (PICO = Population, Intervention, Comparator, Outcome). The special attention will be dedicated to new, emerging indications as well as re-evaluation of the non-approved indications. All the reports will circulate between the expert group and each expert will be asked to weight his agreement or disagreement with the proposed recommendations. During the conference, reports and expert opinions will be presented, discussed and final consensual recommendations with weighted evidence and jury confidence will be issued. Some new practical aspects will be included, namely contraindications for HBOT, cost analysis for hyperbaric treatment, oxygen dosing, as well as choosing the appropriate recompression schedule (non-standard HBOT) in case of decompression illness and gas embolism.
- After this meeting, ECHM will officially be integrated in the EUBS structure as a Committee, according to the changes in the Constitution and Bylaws of both organisations, voted and approved in 2025.

Whether you read this during or after the EUBS-ECHM meeting, we are confident that all participants will enjoy the Swiss hospitality and an excellent scientific and social program. For those who have planned to have some days off before and after the conference to enjoy the Geneva area – extra beautiful that time of the year – we wish you a marvellous stay.

The report of the General Assembly of EUBS, held on Friday 18th at the end of the conference, is available to EUBS members in their Members Area; the Abstract Book

electronic file will be available for download to EUBS members there as well.

EUBS website

As always, please visit the EUBS website (www.eubs.org) for the latest news and updates. Do not forget to renew your membership annually – each member will receive a personal renewal invitation one month before expiry; even if your membership has expired, you can easily renew it when trying to log in again. In case of problems, do not hesitate to contact the EUBS secretary at secretary@eubs.org.

EUBS Website and OXYNET

Occasionally, we can use the EUBS website newsletter as a tool to seek help for our members, as it is a perfect way to reach all of the EUBS membership and because communication, networking and interaction are prime goals of our Society.

The OXYNET database of hyperbaric centres is presented as an interactive map page on the EUBS website. ExCom is looking for members in each country help us to keep the database up to date - let us know if you are willing to help.

A Help Requests page on our EUBS website has been created (EUBS Members Help Requests, under the "Activities" menu on the homepage). Please check this page and try to help out. In case you need help as well and would like to use this service, please contact the webmaster (webmaster@eubs.org).You should also consult the page (http://www.eubs.org/?page_id=284) where research projects seeking collaborators and international participation are presented.

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.

Events, courses & news

Announcement DMP/HMP curriculum

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 members 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.

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

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).

- **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.

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.

DISCLAIMER

Opinions expressed in this publication are given in good faith and in all cases represent the views of the authors and are not necessarily representative of the policies or views of SPUMS, EUBS or the Editor and Editorial Board.