Diving and Hyperbaric Medicine

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

SPUMS *Volume 55 No. 3 September 2025* EUBS

Revised CNS limit for 1.3 atmospheres of inspired oxygen

HBOT for femoral head avascular necrosis

Bacterial survival in hyperbaric air

Vestibular rehabilitation after inner ear DCS

The role of ECGs in assessing fitness for military diving

Diving with autism spectrum disorder

Serial chest CT after freediving pulmonary syndrome

HBOT for dropped head syndrome after radiotherapy

Periorbital emphysema complicating HBOT

Treating neonates with HBOT

Tissue necrosis after marine sting

E-ISSN 2209-1491 ABN 29 299 823 713

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

EDITORIAL OFFICE

PURPOSES OF THE SOCIETIES

To promote and facilitate the study of all aspects of underwater and hyperbaric medicine
To provide information on underwater and hyperbaric medicine
To publish a journal and to convene members of each Society annually at a scientific conference

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

CONTENTS

Diving and Hyperbaric Medicine Volume 55 No. 3 September 2025

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 cover photo for this issue shows technical divers Sami Paarkarinen (deeper) and Dr Laura Tuominen lighting a vertical shaft for a grateful photographer (myself) in the famous Ojamo flooded mine outside Helsinki, Finland. The photo is significant for two reasons. First, I have just returned from the EUBS meeting in Helsinki, where both Sami and Laura spoke. I offer my congratulations to Dr Anne Räisänen-Sokolowski and her team for organising this fabulous event. I was lucky enough to get three days of diving in Ojamo prior to the meeting. Second, technical divers worldwide will be fascinated to read the new guideline for managing oxygen exposure (and oxygen toxicity risk) at an inspired PO_2 of 1.3 atmospheres that appears in this issue. The guideline, authored by Joseph Hoyt and colleagues, was based on a synthesis of available evidence and refined at a workshop held in conjunction with the American Academy of Underwater Sciences at Seattle in April. The paper describes the essential absence of relevant evidence underpinning the old recommendations and provides an evidence base for modifying the 'central nervous system' limit for a 1.3 atmosphere oxygen exposure; a limit that is frequently being ignored or broken. This will be a very consequential paper in the world of technical diving.

Also in this issue, Mehmet Ekici and colleagues publish a substantial series of avascular necrosis cases treated at Kayseri City Training and Research Hospital, Turkey. Both objective (MRI scans) and subjective (pain) improvements were associated with hyperbaric oxygen treatment. This complements a number of other series, including one from the Perth (Australia) group recently published in this journal.

Laetitia Hendier and colleagues from the University of Geneva Hospitals report a study of the effect of hyperbaric air on bacterial growth on common surfaces found in hyperbaric chambers. This is of particular relevance to multiplace chambers where the presence of multiple patients simultaneously creates an environmental contamination and cross infection risk.

Rosanna Stokes and her colleagues from DDRC report a comprehensive evaluation of the effect of vestibular rehabilitation strategies on recovery after inner ear decompression sickness. Although some of the interventions are probably beyond the resources available to many hyperbaric units, the paper also provides some pragmatic advice for simple interventions. This outstanding work is part of Rosanna's PhD programme, and was presented at the EUBS meeting.

Bas van der Kooi and colleagues from the Royal Netherlands Navy provide another contribution thematically aligned with rationalizing the testing / investigation that is routinely undertaken as part of occupational diving medicals (with a military focus here). In this case, they put routine ECG recordings under the spotlight. This work is extremely important in providing an evidence base for changing some of the conservative approaches that have been taken over a long period.

Bram Querido and Thijs Wingelaar address another topic to complement Dr Querido's recent series of reviews of diving with psychiatric conditions such as attention deficit hyperactivity disorder and psychoses. On this occasion they review the issues around diving with autism spectrum disorder. These are among the most controversial and debated issues in fitness for diving, and although most decisions devolve to an 'every case on its own merits' approach, the provision of a framework for approaching the subject is extremely valuable.

There is one case series and four case reports in this issue. Gizem Kavram and colleagues from Turkey describe treatment of three neonates with hyperbaric oxygen; two for distal thrombotic emergencies and one for non-healing and infection in a surgical wound. This complements a recent larger series, also from Turkey, of older infants treated with hyperbaric oxygen and involving one of the present authors. Most hyperbaric practitioners have a degree of nervousness around the unfamiliarity of offering treatment for neonates, and this series provides valuable reassurance that it can be done safely. Madeleine Wagner and colleagues report serial CT scans in a freediver who suffered freediving induced pulmonary syndrome. Ping Wu and colleagues describe a case of severe periorbital emphysema arising in a patient who had undergone recent surgery in the related tissue area. Shane Rosenweig and colleagues describe a case of dropped head syndrome following mantle radiotherapy that exhibited significant and sustained improvement after hyperbaric oxygen treatment. Finally, Deon Viljoen and Jessica Andrews provide a timely reminder of the potential for seemingly minor marine stings to produce significant tissue damage.

At a personal level, it is with great sadness that I write an obituary for Prof Des Gorman who died in July this year. Des was a profoundly influential mentor to me and a massive contributor to diving medicine science. He will be missed.

Professor Simon J Mitchell
Editor, Diving and Hyperbaric Medicine

Cover photo: Technical divers Sami Paarkarinen (deeper) and Dr Laura Tuominen lighting a vertical shaft for a grateful photographer (myself) in the famous Ojamo flooded mine outside Helsinki, Finland.

Original articles

The effect of hyperbaric oxygen therapy on lesion size in early-stage femoral head avascular necrosis

Mehmet Ekici[1], Eyüp Can Mazlum[2], Mahmut Burak Laçin[3], Mehmet Emin Akçin[4], Ali Eray Günay[5], Firat Ozan[6]

[1] *Yozgat City Hospital, Orthopaedic and Traumatology Clinic, Yozgat, Türkiye*
[2] *Medical Park Gebze Hospital, Orthopaedic and Traumatology Clinic, Kocaeli, Türkiye*
[3] *Besni State Hospital, Radiology Clinic, Adıyaman, Türkiye*
[4] *Oxigap Hyperbaric Oxygen Center, Undersea and Hyperbaric Medicine Clinic, Gaziantep, Türkiye*
[5] *Private Erciyes Hospital, Orthopaedic and Traumatology Clinic, Kayseri, Türkiye*
[6] *Kayseri City Education and Research Hospital, Orthopaedic and Traumatology Clinic, Kayseri, Türkiye*

Corresponding author: *Dr Mehmet Ekici, Yozgat City Hospital, Orthopaedic and Traumatology Clinic, Yozgat, Türkiye*
ORCiD: *0000-0003-4336-8596*
dr.mehmetekici.ortopedi@gmail.com

Keywords
Bone necrosis; Hyperbaric medicine; Hyperbaric research; Orthopaedics

Abstract
(Ekici M, Mazlum EC, Laçin MB, Akçin ME, Günay AE, Ozan F. The effect of hyperbaric oxygen therapy on lesion size in early-stage femoral head avascular necrosis. Diving and Hyperbaric Medicine. 2025 30 September;55(3):226–230. doi: 10.28920/dhm55.3.226-230. PMID: 40986917.)
Introduction: Femoral head avascular necrosis (AVN) is a common orthopaedic condition that occurs when intraosseous microcirculation is compromised. Hyperbaric oxygen therapy (HBOT) increases tissue oxygen concentration, reduces oedema, stimulates angiogenesis, lowers intraosseous pressure, and enhances microcirculation. The aim of this study was to evaluate the effectiveness of HBOT in early femoral head AVN based on magnetic resonance imaging (MRI) findings.
Methods: A total of 37 hips from 25 patients with Ficat Stage 1–2 femoral head AVN, followed between 2018 and 2021 and receiving HBOT at Kayseri City Training and Research Hospital, were retrospectively included. Thirty HBOT sessions of 90 minutes each were administered at 243 kPa pressure (2.4 atmospheres absolute) with 100% oxygen breathing, along with a weight-bearing restriction protocol.
Results: There were 20 females and five males. The mean (standard deviation) age was 46.9 (9.5). In pre-treatment MRI imaging, the mean lesion size was 29.87 (22.64) cm^3 in 20 right hips and 28.84 (14.95) cm^3 in 17 left hips ($P = 0.183$). At the second month after treatment, the lesion size was 12.39 (11.26) cm^3 in 20 right hips and 21.81 (13.56) cm^3 in 17 left hips ($P < 0.001$). The mean pre-post differences for the right and left hips was 17.48 (21.15) cm^3 and 7.02 (5.95) cm^3 respectively (both $P < 0.001$).
Conclusions: Femoral head AVN is a progressive disease, with femoral head collapse exceeding 40% in a five-year follow-up. This study demonstrated a reduction in lesion size associated with HBOT in early stage femoral head AVN. In our opinion, HBOT is an integral part of the treatment for early-stage femoral avascular necrosis.

Introduction

Femoral head osteonecrosis is a commonly encountered orthopaedic condition in the general population, manifesting when intraosseous microcirculation is disrupted.[1,2] Although increased use of corticosteroids, alcoholism, haemoglobinopathies, pregnancy, autoimmune disease, and trauma are recognised as risk factors, the aetiology remains unknown in most cases.[3] Early diagnosis and preservation of the shape of the femoral head are fundamental principles in treatment. Conservative approaches such as non-steroidal anti-inflammatory (NSAID) use, restricted weight-bearing, and physical therapy protocols often prove unsuccessful.[4] Spontaneous healing occurs in only a small percentage of osteonecrotic femoral heads. Approximately 67% of asymptomatic patients and 85% of symptomatic patients progress to femoral head collapse.[5]

Surgical treatment is an option for symptomatic hips, but the type of surgery varies based on the severity of the disease. Procedures that preserve the femoral head, such as vascularised or non-vascularised bone grafts with core decompression, pedicled muscle grafts, and derotation osteotomies, may be preferred in the early stages of avascular necrosis. In advanced stages of femoral head osteonecrosis, total hip arthroplasty is an effective treatment. Young patients

undergoing hip arthroplasty have a higher likelihood of revision throughout their lives compared to elderly patients. Therefore, joint-preserving treatments play a crucial role in femoral head osteonecrosis.

Hyperbaric oxygen therapy (HBOT) increases tissue oxygenation, reduces oedema, stimulates angiogenesis, lowers intraosseous pressure, and enhances microcirculation.[6] There is evidence supporting the effectiveness of HBOT in the early stages of the femoral head osteonecrosis.[7] The aim of this study was to evaluate the effectiveness of HBOT in early femoral head avascular necrosis (AVN) based on magnetic resonance imaging (MRI) assessments.

Methods

The protocol used in this study was approved by the ethics committee of Kayseri City Hospital. (no: 343 01.04.2021).

A total of 37 hips from 25 patients who were retrospectively followed between 2018 and 2021 due to Ficat Stage 1-2 femoral head AVN and received HBOT at Kayseri City Training and Research Hospital were included in the study.

Inclusion criteria were, age > 18 years, Ficat stage 1–2 femoral head AVN, patients followed up in the orthopaedic outpatient clinic, and completed 30 sessions of HBOT. Exclusion criteria were rheumatoid arthritis, septic arthritis, previous surgery on the relevant femoral head, malignancy, pregnancy.

The included patients underwent HBOT at 243 kPa with 100% oxygen breathing for 90 minutes per session. A total of 30 sessions were administered, along with a protocol restricting weight-bearing. The 30-session HBOT protocol was completed in our center over a total of six weeks, with five sessions per week. Patients who were undergoing HBOT and were smokers were encouraged to quit or reduce smoking by our hyperbaric and underwater medicine team. No additional medical therapy was applied. All patients who met the criteria and completed 30 sessions of HBOT were included in the study. Two patients who met the inclusion criteria during the relevant period but did not complete 30 sessions were not included in the study.

MRI images of the affected femoral head(s) taken at the 2nd month before and after HBOT were examined. Additionally, visual analogue score (VAS) measurements were compared for the patients before and six months after treatment. In the VAS scaling, the absence of pain was rated as zero (0), while the most severe pain experienced is rated as ten (10).

Hip MRIs were obtained using a 3T magnetic resonance imaging device (Magnetom Skyra; Siemens Healthcare, Erlangen, Germany) with a 13-channel body coil. The images were acquired in axial and coronal planes using T2-weighted turbo spin echo fat saturated (time to repetition – 3750 ms, time to echo – 60 ms, slice thickness – 3.5 mm, field of view – 320 mm, matrix: 256 x 256) and T1-weighted turbo spin echo (time to repetition – 400 ms, time to echo – 15 ms, slice thickness – 3.5 mm, field of view – 320 mm, matrix: 256 x 256) sequences. The avascular necrotic areas in the obtained images were quantitatively measured in volumetric values using the syngo.via software (Siemens Healthineers, Forchheim, Germany).

The data were transferred to electronic media and analysed using SPSS 22.0 software. Normality was tested with the Shapiro-Wilk test. Data were normally distributed and are therefore reported as mean (standard deviation [SD]). The Chi-square test was used for the analysis of categorical data, and the Kruskal-Wallis test was used for the analysis of independent variables. Results with a P-value < 0.05 were considered statistically significant.

Results

The study included 20 female and five male patients, with a mean age of 46.9 (SD 9.5). The demographic data of the patients are summarised in Table 1. The study included 11 smokers. Two patients had a history of steroid use, while the remaining 12 patients had idiopathic AVN.

Lesion sizes before and after HBOT are given for right and left hips separately in Table 2.

When patients were grouped according to the Ficat classification, in Stage 1, the mean difference in lesion

Table 1
Demographic data; F – female; M – male; SD – standard deviation

Parameter	Value
Number of Patients	25
Age, mean (SD)	46.9 (9.5)
Gender (F/M)	20/5
Smoking	11
Steroid use	2
Side	
- Right	8
- Left	5
- Bilateral	12
Ficat stage	
- Stage 1	12
- Stage 2	13
Dominant lower extremity	
- Right	21
- Left	4

Table 2
Lesion sizes before and after treatment in patients

Treatment stage	Lesion size, cm^3 Mean (SD)	
	Right hip	Left hip
Before	29.87 (22.64)	28.84 (14.95)
After	12.39 (11.26)	21.81 (13.56)
Mean difference	17.48 (21.5)	7.02 (5.95)
P-value	< 0.001	< 0.001

size before and after treatment was 2.71 (SD 1.62) cm^3 in the right hip, while in Stage 2, this difference was 25.32 (11.23) cm^3 ($P < 0.001$). In Stage 1, the difference in the left hip before and after treatment was 3.11 (0.60) cm^3, while in Stage 2, this difference was 9.77 (3.01) cm^3 ($P < 0.001$). The difference between stage I and II was statistically significant. A representative MRI image of a bilateral Stage 2 patient is shown in Figure 1.

In the group of patients who smoked, the mean difference between lesions in pre- and post-treatment MRI images was 6.35 (1.33) cm^3, while in the non-smoking group, this difference was 19.64 (4.05) cm^3 ($P = 0.03$).

The mean pre-treatment VAS score was 7.8 (0.7), and after six months of treatment this had decreased to 2.9 (0.9) ($P < 0.001$).

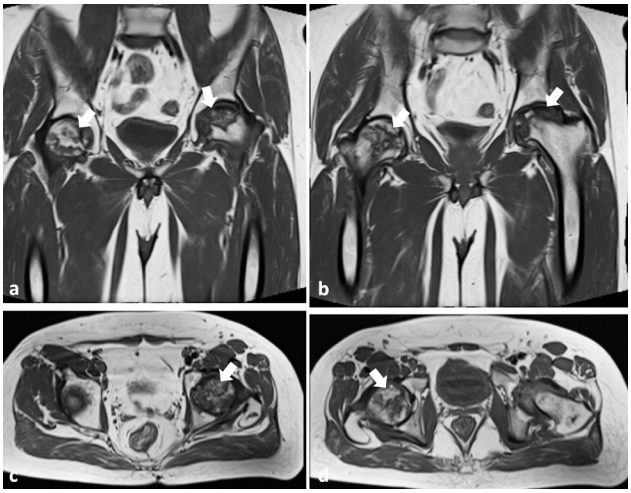
Discussion

Femoral AVN is an extremely progressive disease. Eighty percent of untreated cases progress to femoral head collapse and hip arthritis.[8] Historically, various treatment methods have been attempted, including both surgical methods such as core decompression, osteotomies, and bone grafting, as well as non-surgical methods.[9]

There are several classification systems for femoral AVN.[10–12] The classification system developed by Ficat and Arlet is highly useful in terms of clinical relevance.[13] Early intervention before the collapse of the cartilaginous roof is crucial for hip joint survival. In our study, Ficat-Arlet stages 1 and 2 patients were included.

Steinberg and colleagues emphasised the importance of joint-preserving treatments in early-stage (1–2) patients and demonstrated the potential benefits of HBOT in the early stages.[10] The exact mechanisms by which HBO facilitates

Figure 1
Coronal (a, b) and axial (c, d) T1-weighted magnetic resonance images of the same patient showing irregularly demarcated hypointense avascular necrosis areas in both femoral heads, indicated by white arrows



healing in AVN are unproven, though based on its known effects in other settings[14] it is possible to speculate. Elevated pressures of dissolved oxygen in the arterial blood create a standing osmotic gradient that might reduce bone marrow oedema. Higher dissolved oxygen pressures increase diffusion distance and improve oxygen supply to poorly perfused tissue where it is required to support oxygen-dependent healing processes. Intermittent hyperbaric oxygen has also been shown to stimulate angiogenesis in healing tissue. There is evidence that it modulates osteoclast and osteoblast functions.[15]

In a large series of patients with HBO treatment for femoral AVN, Steinberg et al. reported clinical results for 54 patients (58 hip joints) with idiopathic, traumatic, and secondary AVN in stages I and II. All patients received 90-minute HBO sessions six times a week, with an average of 80 sessions per patient. Over an average follow-up period of eleven years, four joints (7%), all of which had secondary AVN, underwent hip arthroplasty. This resulted in a 100% hip 'survival' rate for idiopathic and traumatic cases.[16] In a 30-year follow-up study by Currie et al., objective improvement was demonstrated in 71% of patients after HBOT according to MRI image results. It was also shown that subjective changes were good in 93% of patients.[17] These results are highly consistent with those reported here where the patients also reported subjective improvements that paralleled the observed reduction in lesion size.

Moghamis et al. reported one year follow-up data showing that femoral AVN patients receiving HBOT had equivalent Oxford Hip Score outcomes to patients receiving core decompression.[18] In another study, Oxford Hip Scores were found to be high in 73–86% of femoral AVN patients who received HBOT. In this patient group, a 92% improvement

in VAS scores was also reported.[19] In our study, consistent with the literature, pain scores decreased to satisfactory levels in 24/25 patients (96%).

In a recent review the hip survival in Ficat Stage 2 hips after HBOT was found to be 100%.[20] The amount of lesion regression was statistically higher in Stage 2 compared to Stage 1 in 13 patients. However, this may be related to the larger lesion size in advanced-stage patients.

Hirota et al. reported that smokers and individuals who have a smoking history of 10 pack-years have an increased risk of femoral head avascular necrosis.[21] This negatively affects healing due to microvascular circulation disorders. In our study, the reduction in lesion size in 11 patients who smoked was calculated to be significantly lower ($P = 0.03$). Based on this result, we think that more effective results can be obtained by encouraging patients with femoral AVN to quit smoking.

The difference in lesion sizes measured by MRI imaging before and after treatment in 25 patients included in the present study objectively demonstrated a beneficial change associated with HBOT. However, the limitation of this study is the small sample size and lack of a formal control group. The latter implies that conclusions must be drawn cautiously. One strength of the present study is the volumetric measurement method used which is superior to other studies. This allows for the most accurate measurement of progression, remission, and reactivation, guiding the course of treatment. It enables us to obtain more reliable data compared to two-dimensional measurements in other studies.

Conclusions

This study objectively demonstrated reduction in lesion size in early-stage femoral avascular necrosis patients associated with HBOT. The decrease in bone marrow oedema in the avascular necrosis area positively reflects on clinical outcomes. In our opinion, HBOT is an integral part of the treatment for early-stage femoral avascular necrosis. We hope that larger sample sizes and long-term prospective studies with controls will shed further light on this subject.

References

1 Meyers MH. Osteonecrosis of the femoral head: Pathogenesis and long-term results of treatment. Clin Orthop Relat Res. 1988;231:51–61. PMID: 3286078.

2 Desforges JF, Mankin HJ. Nontraumatic necrosis of bone (osteonecrosis). New Eng J Med. 1992;326(22):1473–9. doi: 10.1056/NEJM199205283262206. PMID: 1574093.

3 Min BW, Song KS, Cho CH, Lee SM, Lee KJ. Untreated asymptomatic hips in patients with osteonecrosis of the femoral head. Clin Orthop Relat Res. 2008;466:1087–92. doi: 10.1007/s11999-008-0191-x. PMID: 18327630. PMCID: PMC2311457.

4 Mont MA, Jones LC, Hungerford DS. Nontraumatic osteonecrosis of the femoral head: ten years later. J Bone Joint Surg Am. 2006;88:1117–32. doi: 10.2106/JBJS.E.01041. PMID: 16651589.

5 Pepke W, Kasten P, Beckmann N, Janicki P, Egermann M. Core decompression and autologous bone marrow concentrate for treatment of femoral head osteonecrosis: a randomized prospective study. Orthop Rev (Pavia). 2016;8:6162. doi: 10.4081/or.2016.6162. PMID: 27114808. PMCID: PMC4821226.

6 Nylander G, Lewis D, Nordström H, Larsson J. Reduction of postischemic edema with hyperbaric oxygen. Plastic Reconst Surg. 1985;76:596–603. doi: 10.1097/00006534-198510000-00021. PMID: 4034778.

7 Reis ND, Schwartz O, Militianu D, Ramon Y, Levin D, Norman D, et al. Hyperbaric oxygen therapy as a treatment for stage-I avascular necrosis of the femoral head. J Bone Joint Surg Br. 2003;85:371–5. doi: 10.1302/0301-620x.85b3.13237. PMID: 12729112.

8 Konarski W, Poboży T, Śliwczyński A, Kotela I, Krakowiak J, Hordowicz M, et al. Avascular necrosis of femoral head-overview and current state of the art. Int J Environ Res Public Health. 2022;19(12):7348. doi: 10.3390/ijerph19127348. PMID: 35742595. PMCID: PMC9223442.

9 Camporesi EM, Vezzani G, Bosco G, Mangar D, Bernasek TL. Hyperbaric oxygen therapy in femoral head necrosis. J Arthroplasty. 2010;25(6 Suppl):118–23. doi: 10.1016/j.arth.2010.05.005. PMID: 20637561.

10 Steinberg ME. The conservative management of avascular necrosis of the femoral head. In: Arlet J, Ficat RP, Hungerford DS, editors. Bone circulation. Baltimore: William and Wilkins; 1984. p. 334.

11 Marcus ND, Enneking WF, Massam RA. The silent hip in idiopathic aseptic necrosis: Treatment by bone grafting. J Bone Joint Surg Am. 1973;55:1351–66. PMID: 4758708.

12 Gardeniers JWM. ARCO (Association Research Circulation Osseous): Committee on terminology and classification. ARCO News 4; 1992. p. 41–6. Available from: https://scholar.google.com/scholar_lookup?journal=ARCO%20News&title=Committee%20on%20terminology%20and%20classification&volume=4&publication_year=1992&pages=41-46&.

13 Ficat RP, Arlet J. Functional investigation of bone under normal conditions. In: Hungerford DS, editor. Ischemia and necrosis of bone. Baltimore: Williams and Wilkins; 1980. p. 29.

14 Mitchell SJ, Moon RE. Hyperbaric and diving medicine. In: Longo D, Fauci A, Kasper D, Hauser S, Jameson JL, Loscalzo J, editors. Harrison's principles of internal medicine. New York: McGraw Hill; 2025.

15 Vezzani G, Quartesan S, Cancellara P, Camporesi E, Mangar D, Bernasek T, et al. Hyperbaric oxygen therapy modulates serum OPG/RANKL in femoral head necrosis patients. J Enzyme Inhib Med Chem. 2017;32(1):707–11. doi: 10.1080/14756366.2017.1302440. PMID: 28385082. PMCID: PMC6009909.

16 Koren L, Ginesin E, Melamed Y, Norman D, Levin D, Peled E. Hyperbaric oxygen for stage I and II femoral head osteonecrosis. Orthopedics. 2015;38:e200–5. doi: 10.3928/01477447-20150305-57. PMID: 25760507.

17 Currie JRB, Gawthrope IC, Banham ND. The use of hyperbaric oxygen for avascular necrosis of the femoral head and femoral condyle: a single centre's experience over

30 years. Diving Hyperb Med. 2024;54:92–6. doi: 10.28920/dhm54.2.92-96. PMID: 38870950. PMCID: PMC11444917.

18 Moghamis I, Alhammoud AA, Kokash O, Alhaneedi GA. The outcome of hyperbaric oxygen therapy versus core decompression in the non-traumatic avascular necrosis of the femoral head: Retrospective cohort study. Ann Med Surg (Lond). 2021;62:450–4. doi: 10.1016/j.amsu.2021.01.084. PMID: 33643644. PMCID: PMC7889431.

19 Salameh M, Moghamis IS, Kokash O, Ahmed GO. Hyperbaric oxygen therapy for the treatment of Steinberg I and II avascular necrosis of the femoral head: a report of fifteen cases and literature review. Int Orthop. 2021;45:2519–23. doi: 10.1007/s00264-021-05120-3. PMID: 34218337. PMCID: PMC8514350.

20 Uzun G, Mutluoglu M, Ersen O, Yildiz S. Hyperbaric oxygen therapy in the treatment of osteonecrosis of the femoral head: A review of the current literature. Undersea Hyperb Med. 2016;43:189–99. PMID: 27416686.

21 Hirota Y, Hirohata T, Fukuda K, Mori M, Yanagawa H, Ohno Y, et al. Association of alcohol intake, cigarette smoking, and occupational status with the risk of idiopathic osteonecrosis of the femoral head. Am J Epidemiol. 1993;137:530–8. doi: 10.1093/oxfordjournals.aje.a116706. PMID: 8465804.

Conflicts of interest and funding: nil

Submitted: 28 October 2024
Accepted after revision: 26 June 2025

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: hboevidence wikis.unsw.edu.au. The HBOEvidence site in the process of being integrated into the SPUMS website.

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

Evaluation of bacterial survival on inert surfaces in a hyperbaric environment

Laetitia Hendier[1], Hervé Soule[2], Mohamed Abbas[2], Didier Pittet[2], Rodrigue Pignel[3], Sylvain Boet[3]

[1] *Internal Medicine Department, University of Geneva Hospitals, Switzerland*
[2] *Infection Control Programme and WHO Collaborating Centre on Antimicrobial Resistance and Infection Prevention and Control, University of Geneva Hospitals and Faculty of Medicine, Geneva, Switzerland*
[3] *Diving and Hyperbaric Medicine Unit, Division of Emergency Medicine, Department of Anaesthesiology, Clinical Pharmacology, Intensive Care and Emergency Medicine, Geneva University Hospitals and Faculty of Medicine, University of Geneva, Geneva, Switzerland*

Corresponding author: *Dr Laetitia Hendier, Internal Medicine Department, The University of Geneva Hospitals, rue Gabrielle-Perret-Gentil 4, 1202 Geneva, Switzerland*
ORCiD: *0009-0000-2276-4712*
laetitia.hendier@hcuge.ch

Keywords
Bacteria; Hyperbaric oxygen; Infection control; Microbiology; Pressure chamber; Safety

Abstract
(Hendier L, Soule H, Abbas M, Pittet D, Pignel R, Boet S. Evaluation of bacterial survival on inert surfaces in a hyperbaric environment. Diving and Hyperbaric Medicine. 2025 30 September;55(3):231–235. doi: 10.28920/dhm55.3.231-235. PMID: 40986918.)
Introduction: Surface cleaning and hand hygiene within hyperbaric chambers are challenging because of the risk of fire with currently used products containing alcohol or glycerine. This study aimed to investigate if hyperbaric conditions could have inhibitory effects on bacteria present on inert materials.
Methods: We deposited *Staphylococcus aureus (S. aureus)* and *Escherichia coli (E. coli)* on inert materials in an experimental chamber (Comex1200Alu) and compressed the chamber environment with air (253 kPa, 95 minutes) (referred to as indoor). The control was contaminated materials placed outside the chamber (referred to as outdoor). We chose inert materials including plastic, metal, and seat upholstery (imitation leather). We measured bacterial growth and survival and compared the groups using a Student's *t*-test.
Results: Regardless of the surface types tested, there were no significant differences in bacterial reduction between indoor and outdoor conditions for either *E. coli* or *S. aureus* and any of the materials ($P > 0.05$).
Conclusions: We found that pressurised air (253 kPa for 95 minutes) has neither proliferative nor bactericidal action on *S. aureus* and *E. coli* colonies deposited on inert surfaces compared to those present outside a hyperbaric chamber in normobaric air conditions.

Introduction

Hyperbaric oxygen therapy (HBOT) is treatment approved for multiple elective and urgent indications, including treating some infectious processes. To attain a hyperbaric condition, special compression chambers are used. There are two kinds of chambers: monoplace chambers that allow treatment of a single patient at a given time, and multiplace chamber that allow treatment of several patients simultaneously in the same chamber. Multiplace chambers are compressed with air while monoplace chambers are often compressed with oxygen.

For infection control, disinfection must be ensured between sessions for different patients to avoid nosocomial spread. In this regard, it is important to know if an environment has inherent bactericidal or bacteriostatic effect. The most important measures to reduce transmission of bacteria and the risk of healthcare associated infections are environment cleaning and hand hygiene with alcohol-based hand rubs.[1] However, many products recommended for disinfection such as alcohol-based hand rubs and soaps containing glycerine are flammable in the hyperbaric environment or are corrosive for the structure. Cleaning measures have therefore been put in place, avoiding any product likely to catch fire in the hyperbaric chamber, such as ultraviolet-C (UV-C). UV-C has germicidal properties as it is absorbed by nucleic acids in microorganisms, resulting in irreversible cell damage, and rapid cell death. There is a significant reduction in bioburden following adjunctive UV-C disinfection than with standard cleaning alone. However, this technique may fail to disinfect shadowed surfaces, and may be damaging to acrylic components of monoplace or other chambers.[2]

Previous studies have explored the effect of HBOT on bacteria in hyperbaric and hyperoxic environments relevant

to monoplace chambers. High tension of oxygen may be bacteriostatic and/or bactericidal.[3] The hypothesised mechanism is that a defect in nucleic acid or energy metabolism, or both, exists in hyperoxic cells.[4] Another possible antibacterial mechanisms of HBOT could be the absence of CO_2 increasing the bactericidal effect of the oxygen alone.[5] Some facultative anaerobic and obligate aerobic bacteria are resistant to hyperoxia but not at higher pressures of oxygen e.g., 100% O_2, 304 kPa (3 atmospheres absolute [atm abs]) over a prolonged exposure (24 hours). The bactericidal activity of hyperbaric oxygen against these species is potentiated by the absence of carbon dioxide.[6] The bactericidal and bacteriostatic effects of hyperbaric oxygen are more evident as the pressure increases and are not always dependent upon which oxygen tension is applied.[7] In biological tissues, HBOT has a bactericidal or bacteriostatic effect depending on the type of bacteria.[8]

While there are several studies concerning hyperbaric hyperoxic conditions applicable to the monoplace chamber environment, there are limited data on hyperbaric air. This is relevant to multiplace chambers compressed with hyperbaric air (not oxygen). The effect of hyperbaric air on bacteria present on inert surfaces of the chamber has been overlooked. It is unclear if hyperbaric air inhibits (or enhances) bacterial growth, and what the implications are for infection control in the hyperbaric environment. This is of clinical importance because multiplace hyperbaric chambers can accommodate several patients at once, in close proximity, which may allow for contact between patients who may be infected or colonised. This poses a risk for patient-to-patient transmission of pathogens, including multidrug resistant bacteria.

This study aimed to evaluate the effect of hyperbaric air on the viability of bacteria on different materials found in multiplace chambers in the absence of specific cleaning measures. We hypothesised that hyperbaric air has an inhibitory effect.

Methods

We evaluated the survival of two facultative anaerobic bacteria deposited on inert surfaces, *Staphylococcus aureus* and *Escherichia coli*. They were exposed to hyperbaric air for 95 minutes (a typical duration of hyperbaric oxygen therapy session).

The method used was inspired by the phase 2 / step 2 application standard NF EN 14561. In this bactericidal standard simulating real conditions, inoculated sheets artificially contaminated with bacteria are exposed to the disinfectant being tested.[9] In this work, the inoculated sheets were not exposed to a disinfectant, but to particular atmospheric conditions in order to study their effect on the survival of the bacteria. Ethics approval was not necessary because we did not use a chamber using for patients and the bacteria came from a collection.

CONTAMINATED TILE

We took three materials often found in clinical hyperbaric chambers: plastic flooring, metal and imitation leather seat coverings. Pieces of about 1 cm^2 were cut and autoclaved. Six to eight sterile Petri dishes, each containing a sprout carrier of each material, were prepared for each series of tests.

PREPARATION OF THE INTERFERING SUBSTANCE SOLUTION

The interfering substance (organic material that may inhibit antibacterial action) used was bovine albumin fraction V for biochemistry (SIGMA). A dilution of albumin with sterile water was performed to obtain a solution at a concentration of 0.6 $g{\cdot}L^{-1}$. This solution was filtered through a 0.45 µm syringe microfilter and stored in the refrigerator until used.

PREPARATION OF CONTAMINATION SUSPENSIONS

The reference strains used to perform the experiment were *E. coli* K12 NCTC 10536 and *S. aureus* NC 10788. Pure cultures of each of the two bacteria were prepared on tryptone-soy agar (TSA) (Biomérieux) for 18–24 h at 36 ± 1°C. Colonies collected on a second subculture were diluted in 100 mL of tryptone-soy broth (TSB) (Biomérieux) and incubated for 18–24 h at 36 ± 1°C. Finally, those cultures were diluted with sterile water to constitute homogeneous bacterial suspensions containing approximately 10^8 colonies forming units (CFU)$\cdot mL^{-1}$.

For each species, the bacterial test suspension used for the contamination of the inert surfaces was prepared by mixing this suspension with the interfering substance solution in equal parts, in order to obtain a final albumin concentration of 0.3 $g{\cdot}L^{-1}$. To accurately estimate bacterial counts within each suspension, serial dilutions were prepared and a 1 mL sample of each of the dilutions was spread over the surface of TSA for *E. coli* and ChromID *S. aureus* (Biomérieux) for *S. aureus*. These were incubated at 36 ± 1°C for 48 h. Bacterial CFU were quantified by visual inspection, adjusted for the corresponding dilution factor, and converted to $\log_{10}$.

The test suspensions were used within one hour of their preparation.

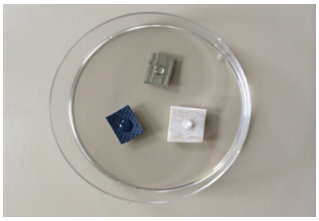
CONTAMINATION OF THE INOCULATED SHEETS

For each test, one of the two bacteria tested was used. Each of the three inoculated sheets was contaminated with 20 µl of the contamination suspensions, corresponding to approximately 10^6 CFU (Figure 1). They were allowed to dry at room temperature for 90 minutes before the actual test.

HYPERBARIC CHAMBER

The experimental hyperbaric chamber used was a Comex 1200Alu which is 1 m long and has a diameter of 1.2 m. It is

Figure 1
Three germ-supports: imitation leather metal, plastic, placed in a sterile Petri dish



equipped with pressure, temperature, and humidity sensors. Although not a clinical chamber, the tests reproduced typical pressurised air conditions of a multiplace HBOT session.

TEST METHOD

Three different inoculated sheets previously contaminated with one of the two contamination suspensions were placed in two sterile dishes. One dish was introduced inside the hyperbaric chamber, and the other one was left outside, close to the chamber.

The inoculated sheets were exposed for 95 minutes to hyperbaric air. Specifically, the hyperbaric exposure involved compressing the chamber with air to 253 kPa over 15 minutes, maintenance at 253 kPa for 65 minutes, followed by decompression to surface pressure over 15 minutes. Outside the chamber, the inoculated sheets remained for 95 minutes in normobaric air (101 kPa). At pressure, the internal chamber temperature was 24°C, 2°C higher than the external temperature. The humidity was 12% inside compared to 40% outside. A single compression cycle was performed for each bacteria tested. Once the cycle was completed, each inoculated sheet was immediately introduced into a vial containing 100 mL of tryptone soy mixture (Biomérieux) using sterile forceps. Each test was repeated ten times with each bacteria.

ENUMERATION OF RESIDUAL BACTERIA AFTER THE TEST (FINAL VALUE)

The vials were stored in a refrigerator. The analysis began immediately. Each vial was vigorously shaken for three minutes to remove the residual bacteria from their inoculated sheets. Three agar plates (TSA for tests with *E. coli*, and ChromID *S. aureus* for tests with *S. aureus)* were inoculated on the surface from each vial: one with 1 mL of undiluted liquid and the other two with 10^{-1} and 10^{-2} dilutions respectively. The agar plates were incubated at 36 ± 1°C. After 48 h, bacterial CFU were quantified by visual inspection, adjusted for the corresponding dilution factor and the total initial volume of broth used to recover bacteria from the inoculated sheets, and converted to $\log_{10}$.

STATISTICAL CALCULATION

For each inoculated sheet, the difference between the initial contamination, close to 10^6 CFU, and the final value was calculated. This is the logarithmic reduction expressed in $\log_{10}$. The average logarithmic reductions between inside and outside the chamber were compared, for each surface material and each bacteria. This difference was assessed using Student's *t*-test to evaluate whether the difference was statistically significant. A *P*-value of less than 0.05 was considered statistically significant.

Results

For each of the three surfaces (metal, seat coverings and plastic floor covering) and for each of the two bacteria tested, we obtained ten results under indoor conditions (i.e., within the hyperbaric chamber) and ten results under outdoor conditions (i.e., outside the chamber). The results are summarised in Table 1. Although the average initial logarithmic contamination of *E. coli* (6.46 CFU·mL^{-1}) was higher than for *S. aureus* (6.12 CFU·mL^{-1}) this was of no functional relevance to the experiment.

Both bacteria on all surfaces showed a reduction in CFU over the course of both inside and outside exposures, but there were no significant differences for either bacterial species between inside (hyperbaric) and outside (normobaric) environments. We showed a lower logarithmic reduction for *S. aureus* than for *E. coli* both inside and outside the chamber.

Discussion

This *in vitro* study assessed bacterial survival in hyperbaric and normobaric air environments and found that there was no difference in survival of *E. coli* and *S. aureus.* The hyperbaric air environment did not lead to a bacterial multiplication or bactericidal effect on either of these bacteria colonising inert materials, even though the membrane structure from the bacteria Gram-positive (*S. aureus*) and Gram-negative (*E. coli*) are different. In contrast, a previous study showed that gram-negative bacteria persisted longer than gram-positive bacteria in normobaric air conditions. This may be related to the humidity. Indeed, humid conditions have been showed to improve persistence for most types of bacteria including *E. coli* while *S. aureus* was found to persist longer at low humidity level.[10]

Our study evaluated bacterial survival on inert surfaces in the context of increased pressure. The results obtained are difficult to compare with other studies evaluating the effect of hyperbaric conditions on bacterial survival which focused on the effect of hyperbaric oxygen exposure on bacteria cultured in nutrient media. For example, in the study by Masure et al., the survival of bacteria in a liquid nutrient medium was assessed. The study described a bacteriostatic effect of

Table 1

Mean and standard deviation (SD) initial contamination and mean (SD) reduction inside and outside the hyperbaric chamber reported as $\log_{10}$ colony forming units per mL (CFU·mL^{-1}) for different bacteria and germ support; *P*-values are for comparisons between inside the chamber (hyperbaric air) and outside the chamber (normobaric air)

Surface	Inside the chamber		Outside the chamber		*P*-value
	Mean initial contamination (CFU·mL^{-1} $\log_{10}$)	Mean reduction (CFU·mL^{-1} $\log_{10}$)	Mean initial contamination (CFU·mL^{-1} $\log_{10}$)	Mean reduction (CFU·mL^{-1} $\log_{10}$)	
Bacteria 1: *Staphylococcus aureus*					
Metal (aluminium)	6.12 (0.12)	0.57 (0.18)	6.12 (0.12)	0.54 (0.23)	0.37
Seat (Imitation leather)	6.12 (0.12)	1.96 (0.56)	6.12 (0.12)	2.28 (0.37)	0.08
Floor (plastic)	6.12 (0.12)	1.31 (0.48)	6.12 (0.12)	1.39 (0.40)	0.34
Bacteria 2: *Escherichia coli*					
Metal (aluminium)	6.46 (0.11)	1.60 (0.21)	6.46 (0.11)	1.56 (0.19)	0.33
Seat (Imitation leather)	6.46 (0.11)	4.30 (0.27)	6.46 (0.11)	4.22 (0.41)	0.39
Floor (plastic)	6.46 (0.11)	2.46 (0.31)	6.46 (0.11)	2.57 (0.32)	0.22

hyperbaric oxygen for *E. coli* at 304 kPa (3 atm abs) and for *S. aureus* at 507 kPa (5 atm abs) for 24 hours.[8] We did not see this effect on inert surfaces with hyperbaric air exposure.

The average $\log_{10}$ reduction for *E. coli* on the imitation leather (seat) surface was higher inside and outside the chamber (4.30 and 4.22 CFU·mL^{-1} respectively) than on the metal surface (1.60 and 1.56 CFU·mL^{-1} respectively). These results for the different surfaces reveals the hypothesis, in a fortuitous way, that there is a difference in the adhesion of bacteria to materials. Bacteria deposited on the seat cover (imitation leather) seemed to adhere more strongly than those deposited on metal surfaces, regardless of the bacteria tested. Although the agitation time of the inoculated sheets in TSB bottles was the same for each bottle, fewer bacteria were collected from the seat cover than from the metal. Bacteria may have preferential attachment to certain materials such as imitation leather. This suggests that cleaning efforts may need to be adapted depending on the nature of the material.

Cleaning measures have been put in place avoiding any product likely to catch fire in the hyperbaric chamber with the introduction of ultraviolet C (UV-C) devices. A recent study was conducted in the context of the COVID-19 pandemic, focusing on the challenges of disinfecting the chamber and discussing the quarantine of patients or the closure of the hyperbaric centre under these conditions. Ultimately, it was demonstrated that, despite the varying conditions during the different phases of the pandemic, it was possible to ensure at least partial operation of the workplace by integrating UVC radiation into each disinfection procedure.[11] Another study evaluated the effectiveness of two UV-C devices in eradicating multidrug-resistant bacteria (*Clostridioides difficile* and methicillin-resistant *Staphylococcus aureus*). These bacteria were suspended and then inoculated onto sheets, which were placed in the hyperbaric chamber, while control plates were placed outside the chamber during the UV-C disinfection. This technique reduces contamination of high-touch clinical surfaces, but more studies are needed to test the comparative efficacy of UV-C devices in real-world clinical environments.[12]

Conclusions

The results of this *in vitro* study showed that the survival of bacteria *S. aureus* and *E. coli* on inert materials inside (hyperbaric air environment) and outside the chamber (normobaric air environment) were not different. From the point of view of the material used, a more pronounced adherence for the imitation leather (seat coverings) may be a key factor to consider. Therefore, a precautionary approach, particularly through the increased use of personal protective equipment such as gloves when patients are subject to contact isolation precautions for example, appears justified to reduce the risk of cross-contamination in multiplace hyperbaric chambers. Further studies on different bacterial species, the effect of several compressions on bacteria are required to broaden our knowledge of bacterial survival in hyperbaric chambers in order to better prevent infectious risks for patients. Moreover, to apply this study in clinical condition, it would be interesting to test bacterial growth after the disinfection of the chamber with our clinical disinfection protocol.

References

1 Huart D, Henckes A, Cochard G, Zahar JR, Baron R, Saliou P. Infection control in therapeutic hyperbaric chambers: Practical inventory in France. Infect Control Hosp Epidemiol. 2015;36:1375–7. doi: 10.1017/ice.2015.175. PMID: 26189343.

2 Browne K, Wood D, Clezy K, Lehm J, Walsh WR. Reduction of bacterial load with the addition of ultraviolet-C disinfection inside the hyperbaric chamber. Diving Hyperb Med. 2020;50:332–7. doi: 10.28920/dhm50.4.332-337. PMID: 33325012. PMCID: PMC8026220.

3 Mcallister TA, Stark JM, Norman JN, Ross RM. Inhibitory effects of hyperbaric oxygen on bacteria and fungi. Lancet. 1963;282:1040–2. doi: 10.1016/s0140-6736(63)90011-4. PMID: 14062728.

4 Clark JM, Pakman LM. Inhibition of Pseudomonas aeruginosa by hyperbaric oxygen. II. Ultrastructural changes. Infect Immun. 1971;4:488–91. doi: 10.1128/iai.4.4.488-491.1971. PMID: 5005305. PMCID: PMC416336.

5 Bornside GH, Pakman LM, Ordóñez AA. Inhibition of pathogenic enteric bacteria by hyperbaric oxygen: enhanced antibacterial activity in the absence of carbon dioxide. Antimicrob Agents Chemother. 1975;7:682–7. doi: 10.1128/AAC.7.5.682. PMID: 238466. PMCID: PMC429203.

6 Park MK, Myers RA, Marzella L. Oxygen tensions and infections: modulation of microbial growth, activity of antimicrobial agents, and immunologic responses. Clin Infect Dis. 1992;14:720–40. doi: 10.1093/clinids/14.3.720. PMID: 1562664.

7 Zanon V, Rossi L, Castellani E, Camporesi EM, Palù G, Bosco G. Oxybiotest project: microorganisms under pressure. Hyperbaric oxygen (HBO) and simple pressure interaction on selected bacteria. Med Gas Res. 2012;2:24. doi: 10.1186/2045-9912-2-24. PMID: 22963601. PMCID: PMC3517458.

8 Masure O, Le Foll C, Barthelemy L, Michaud A, Chastel C. Effets de l'oxygène hyperbare sur le développement in vitro de quatre bactéries pathogènes: E.coli, P.aeruginosa, S.aureus, et C.perfringens. Médecine et Maladies infectieuses; 1977. [cited 2024 May 10]. Available from: https://www.sciencedirect.com/science/article/pii/S0399077X77800607.

9 European Committee for Standardization. European Norm 14561: Chemical disinfectants and antiseptics. Quantitative carrier test for the evaluation of bactericidal activity for instruments used in the medical area. Test method and requirements (phase 2/step 2). Version 2007. [cited 2023 Nov 12]. Available from: https://standards.iteh.ai/catalog/standards/cen/2e84463c-3941-49c5-a664-25f0a76dee20/en-14561-2006.

10 Kramer A, Schwebke I, Kampf G. How long do nosocomial pathogens persist on inanimate surfaces? A systematic review. BMC Infect Dis. 2006;6:130. doi: 10.1186/1471-2334-6-130. PMID: 16914034. PMCID: PMC1564025.

11 Hájek M, Chmelař D, Rozložník M, Lochmanová A, Tlapák J, Vrbová T, et al. COVID-19 infection prevention and control management during hyperbaric oxygen therapy: a best practice implementation project. JBI Evid Implement. 2023;21(S1):S57–S63. doi: 10.1097/XEB.0000000000000400. PMID: 38037449.

12 Warren BG, Masker J, Brown G, Gamez I, Smith B, Anderson DJ, et al. Efficacy of UV-C disinfection in hyperbaric chambers. Infect Control Hosp Epidemiol. 2020;41:1080–3. doi: 10.1017/ice.2020.248. PMID: 32546289.

Conflicts of interest and funding: nil

Submitted: 12 July 2024
Accepted after revision: 26 June 2025

Vestibular rehabilitation and recovery in divers with inner ear decompression sickness: a case series

Rosanna J Stokes[1, 2], Doug Watts[2], Gary Smerdon[2], Stephen D Hall[1], Lisa Bunn[1], Jonathan Marsden[1]

[1] *Brain Research and Imaging Centre, University of Plymouth, Plymouth, UK*
[2] *DDRC Healthcare, Science Park, Plymouth, UK*

Corresponding author: *Dr Rosanna J Stokes, DDRC Healthcare, Science Park, Plymouth, UK*
ORCiD: *0009-0006-4501-0634*
rosanna.stokes@ddrc.org

Keywords
ENT; Fitness to dive; Scuba diving; Sharpened Romberg test; Vertigo

Abstract
(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 and Hyperbaric Medicine. 2025 30 September;55(3):236–245. doi: 10.28920/dhm55.3.236-245. PMID: 40986919.)
Introduction: The mechanism of injury and recovery of divers with inner ear decompression sickness (IEDCS) is not well understood and there is no consensus regarding management following recompression treatment. Given the rare occurrence, divers are not routinely offered the standard therapies that patients with other acute vestibular disorders may be offered such as vestibular rehabilitation.
Methods: This is an observational case series of 13 divers presenting acutely with IEDCS to DDRC Healthcare in Plymouth, UK between July 2021 and January 2024. Vestibular and balance tests were undertaken to aid the treating dive physician in the diagnosis and management of the divers with both hyperbaric oxygen therapy and customised vestibular rehabilitation.
Results: Average values for vertical perception, posturography, dynamic gait index and patient-reported outcomes measures improved by discharge and at the three month follow up despite 67% showing an ongoing positive head impulse test or nystagmus in the dark on videonystagmography at follow up.
Conclusions: Divers should be warned that despite symptom resolution or minimal residual symptoms post-IEDCS there is a high rate of deficit evident on vestibular testing, and this, alongside investigation for a right to left cardiac shunt, should be a major consideration when considering returning to diving. For the clinician, a stopwatch timed Sharpened Romberg's test appears to be a reasonable method for monitoring progress of balance stabilisation during the treatment period. Early initiation of vestibular rehabilitation exercises should be considered for all divers with IEDCS.

Introduction

Inner ear decompression sickness (IEDCS) represents around 20% of decompression sickness cases.[1] This may present with acute vestibular symptoms (vertigo, unsteadiness) and/or cochlear symptoms (loss of hearing, tinnitus), ranging in severity from a feeling of unsteadiness to severe vertigo and vomiting. These symptoms can be resistant to recompression therapy, requiring repeated treatments and many divers are left with permanent deficits in vestibular function.[2] The exact mechanism of injury and recovery is not well understood. Animal studies have shown that there is haemorrhage and precipitation of blood proteins into the perilymphatic and endolymphatic vestibular systems, as well as ectopic new bone growth in fluid spaces after one month.[3,4]

Diver case studies have described IEDCS after deep/technical diving where tissues are supersaturated with inert gas. In some cases, the event was associated with the switching of breathing gases causing local counter-diffusion of inert gases and exaggerated supersaturation which the inner ear appears particularly sensitive to.[5] Other studies describe conservative dive profiles well within table limits. In such cases, around 70% of divers are found to have an underlying right to left cardiac shunt such as a patent foramen ovale (PFO) suggesting a possible embolic pathology.[6,7]

IEDCS is treated acutely with hyperbaric oxygen recompression therapy with better outcomes seen with a shorter time to recompression.[8] Therapy is repeated daily until the symptoms improve or plateau. Diving physicians commonly use basic clinical bedside balance tests such as a heel-toe walk or a Romberg/Sharpened Romberg test to assess symptoms, however there is no standardised method for comprehensively assessing residual vestibular symptoms.[9] More detailed vestibular testing can be undertaken but this is often limited by access to specialised clinicians and equipment.

Previous studies have utilised detailed vestibular testing on divers presenting acutely with inner ear pathology. Results have shown the characteristics of a peripheral vestibular lesion but these studies have not provided a comprehensive insight into the specifics of the mechanism of injury or how the lesion/symptoms change over time in these divers.[2,10] Studies regarding IEDCS tend to focus on the acute phase, with little understanding of how function is affected over the longer term and what considerations should be made when assessing fitness to return to diving. There is also a paucity of qualitative information regarding the burden of residual symptoms.

Vestibular rehabilitation exercises are commonly used in other acute vestibular disorders such as vestibular hypofunction, but they have not been utilised in IEDCS recovery as standard.[11] Early initiation of vestibular rehabilitation has been shown to aid clinical recovery in these disorders and should be considered an important adjunct to hyperbaric therapy.[12]

The aim of this case series was to describe the short and long-term effects of IEDCS on the vestibular system in these divers to aid understanding of symptom burden, prognosis and allow a more informed assessment when considering fitness to return to diving.

Methods

Ethical approval for studying the divers with IEDCS was obtained through the Integrated Research Application System (Approval number 337421). Ethical approval for collection of normative data was obtained through Plymouth University. An observational study was performed involving all scuba divers presenting acutely with IEDCS to the DDRC Healthcare hyperbaric chamber in Plymouth between July 2021 and September 2024. Any alternative diagnosis for inner ear symptoms such as barotrauma or infection was an exclusion criterion and thorough history and otoscopic examination was undertaken on each diver presenting at the hyperbaric centre.

Divers were offered laboratory and clinical vestibular testing (as described below) as well as a personalised vestibular rehabilitation exercise program to aid their diagnosis and recovery. Laboratory based vestibular testing was offered after the initial recompression and once symptoms (e.g., vomiting/severe nausea) allowed. Testing was timed to not interfere with scheduled repeated recompression treatments, with results used to aid clinical assessment by the treating dive doctor. Testing was repeated on the day of discharge with advice to progress rehabilitation exercises at home and the option of telephone follow up. Testing was also repeated opportunistically when divers attended DDRC Healthcare for their return to diving medical examination which was usually scheduled for three months post injury.

Assessments included clinical tests (head impulse test, eye position with and without Frenzel's glasses and eye movement [smooth pursuit and saccades], timed Romberg's in tandem stance and the dynamic gait index [DGI]). The DGI assesses eight walking tasks on a 0–3 ordinal scale including head movements while walking. The maximum score is 24 with greater than 22 indicating safe ambulators and < 19 being predictive of falls.[13]

Laboratory vestibular tests included videonystagmography (VNG) and rotary chair assessment of nystagmus, eye movements (smooth pursuit, saccades and optokinetic reflex) and vestibulo-ocular reflex (VOR) (using sinusoidal and step rotations) as described previously.[14]

In addition, posturography measured postural sway over 30 seconds via force plates with a 1 kHz sampling rate (Moteklink, Motek, Netherlands or Kistler 9286AA Kistler, Instruments Ltd, Hampshire, UK) with eyes open/closed with feet 10 cm apart, feet together or in tandem. Signals were filtered (4th order, 5 Hz low pass Butterworth) and the centre of pressure velocity ($mm \cdot s^{-1}$) was calculated offline.[15] Subjective visual vertical was assessed in the dark with a static background and a dot array rotating at $30° \cdot s^{-1}$ clockwise or anti-clockwise using the Rod-and-Disc test.[16] Divers also completed a set of questionnaires validated for people with peripheral vestibular dysfunction comprising the Dizziness Handicap Inventory,[17] Vertigo Symptom Scale,[18] Situational Characteristics Questionnaire,[19] and the Activities-Specific Balance Confidence Scale (ABC-UK).[20] Open discussion regarding any issues highlighted on these questionnaires allowed for qualitative feedback of the divers' experience of their symptom burden and progress with vestibular rehabilitation.

Normative data for the posturography, Rod and Disc test and VNG/rotary chair testing was collected using members of the general population as controls (n = 15). Those who had a history of decompression illness or vestibular disorders were excluded. The IEDCS data was defined as abnormal if it was greater or lesser than the mean ± one standard deviation (SD) of the normative values.

Results

DIVER DEMOGRAPHICS AND PROVOKING FACTORS

Between July 2021 and September 2024 thirteen divers were seen with symptoms of inner ear disorder after diving. One diver was excluded due to an alternative diagnosis of inner ear barotrauma; this diagnosis was made after worsening of symptoms at pressure. Mean age of the 12 divers was 53 years (range 32–73), 58% were male and 42% female. Mean depth of the provoking dive was 27 metres of seawater (range 19–36); 10 divers were breathing air and two were breathing nitrox 32%. All dives were uneventful with no rapid ascents or known missed decompression

stops. In terms of contributing risk factors; six divers had been diving on consecutive days, two were overweight, one had dehydration secondary to gastroenteritis, one was fatigued from strong currents during the dive, one had type 2 diabetes mellitus and one had a history of previous IEDCS. Of the nine divers who went on to have a bubble contrast echocardiogram, six had an underlying PFO (67%).

SYMPTOM ONSET AND MANAGEMENT

Time from the end of the dive to the onset of symptoms showed considerable variation (mean four hours, range 0–19). The mean time from symptom onset to first recompression treatment was nine hours (range 5–15) with two outliers excluded (76 and 312 hours). These outliers were divers who did not recognise that their symptoms were decompression sickness and delayed seeking medical attention. Two divers had a delay of over 10 hours before recompression; one required imaging in the emergency department prior to reaching the chamber and the other had put his symptoms down to seasickness whilst he had been on the dive boat. All of the divers were treated with an initial Royal Navy Treatment Table 62 at 284 kPa (2.8 atmospheres absolute). Mean number of hyperbaric treatments was seven (range 1–12). All divers were given a balance retraining booklet[21] on the first day of their presentation and then more personalised exercises were given following vestibular and balance assessments.

VESTIBULAR TESTING

Of the 12 divers with IEDCS, eight were seen at all three test points; one was excluded as they were not seen in the acute phase, one missed testing due to equipment malfunction and two did not attend follow-up. Initial testing (referred to as VFT1) was performed at a mean of 2.13 days post symptom onset (SD 1.13), discharge testing (VFT2) was performed at 9.63 days (SD 4.81) and follow-up testing (3MFU) at 6.53 months (SD 5.19).

Although no baseline audiometry results were available for comparison, no objective signs of new hearing loss were seen on audiogram for any of the eight divers. All had signs of vestibular dysfunction; 63% had a right sided lesion whilst 38% had a left sided lesion (as determined by the head impulse test).

Table 1

Results of dynamic gait index and vestibular function testing at admission, discharge and three-month follow-up; VOR – vestibulo-ocular reflex

Measure	Admission (n = 8)	Discharge (n = 8)	Follow-up (n = 8)
Mean time of assessment after onset	2.13 days	9.63 days	6.53 months
Dynamic gait index, mean (range)	19 (16–23)	23 (20–24)	24 (23–24)
Optokinetic response	10% asymmetry towards side of lesion	3% asymmetry towards side of lesion	Symmetrical
Smooth pursuit gain 0.2 Hz–0.4 Hz	Normal	Normal	Normal
Nystagmus slow phase velocity			
Centre	Abnormal	Normal	Normal
Away from lesion	Abnormal	Normal	Normal
Towards lesion	Normal	Normal	Normal
Saccadic			
Accuracy away/towards lesion	Normal	Normal	Normal
Latency away/towards lesion	Normal	Normal	Normal
VOR sinusoidal rotation			
Gain 0.2 Hz (n = 8)	Normal	Normal	Normal
Asymmetry 0.2 Hz (n = 8)	Abnormal	Abnormal	Abnormal
Gain 0.32 Hz (n = 7)	Normal	Normal	Normal
Asymmetry 0.32 Hz (n = 7)	Abnormal	Normal	Abnormal
VOR step rotation			
Time constant away lesion (per)	Normal	Normal	Normal
Time constant towards lesion (per)	Normal	Normal	Normal

Videonystagmography (VNG)

Clinical outcomes are summarised in Table 1 with a * supplementary version providing the mean (SD) and normative data.* Nystagmus was present in all divers in the dark at initial presentation along with a positive horizontal head impulse test to the side of the lesion. Nystagmus in the dark (Table 1 and Figure 1A) showed higher slow phase velocity with gaze towards the contralesional side compared to the ipsilesional side, as expected with Alexander's law.[22] Nystagmus towards both sides decreased in velocity by the end of the treatment period (Figure 1A).

At initial presentation the optokinetic response showed a higher gain when the stimulus moved toward the ipsilesional side but there was a large standard deviation. By discharge and follow up the response was more symmetrical (Table 1). Smooth pursuit gain and phase along with saccadic latency and amplitude were within normal limits at initial and subsequent testing (Table 1).

The VOR was tested using sinusoidal harmonic acceleration in a rotary chair. With rotation at both 0.2 Hz and 0.32 Hz in the dark, gain was normal but low when compared to control participants and remained low over all testing points (Table 1 and Figure 1B), however there was large individual variation. Asymmetry improved over time (Table 1 and Figure 1C). Step rotation (140°·s^{-1} acceleration/deceleration with a 60°·s^{-1} fixed-chair velocity) showed a shorter time constant when rotating toward the ipsilesional side, reflecting an impaired VOR. Although not statistically significant, the time constant towards the ipsilateral side appeared to increase over the treatment period whereas the time constant on the contralateral side appeared to decrease, resulting in a more symmetrical VOR (Figure 1D).

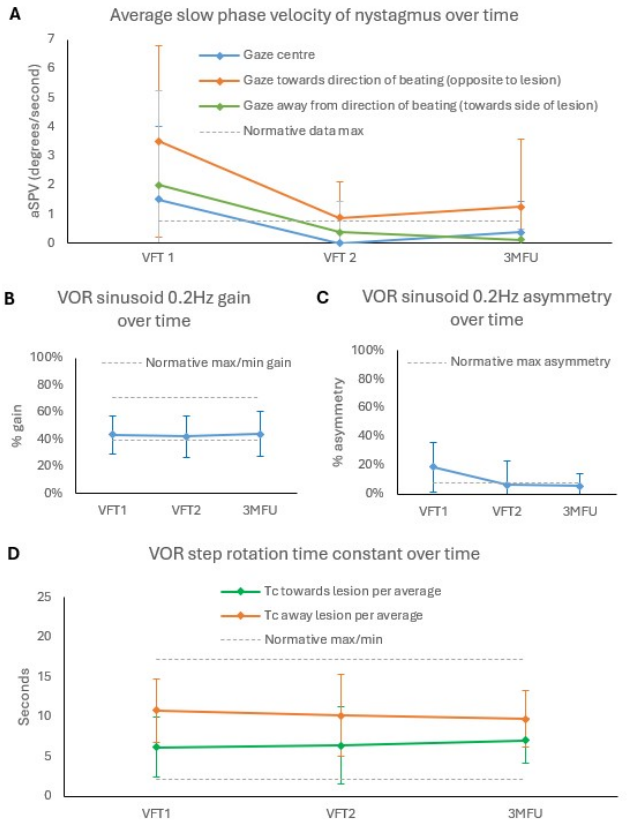
Five of the eight divers (62.5%) showed a persistent deficit on follow up testing with a positive head impulse test or presence of nystagmus with gaze in the dark.

Balance and walking

The velocity of sway (Posturography testing) with a 10 cm stance width (eyes open or closed) or with feet together and eyes open showed little variation over time. In contrast 'feet together, eyes closed' and 'tandem, eyes open' were the most sensitive tests, being increased at baseline and showing improvements over time (Figure 2A and B). 'Tandem, eyes closed' was very difficult for the divers to maintain and only two were able to hold the stance at all at initial presentation, and three at discharge and follow up. Those that did manage to hold tandem stance eyes closed showed improvement over the treatment period in both time in stance and measured sway (Figure 2A and B).

Figure 1

Videonystagmography results (mean and standard deviation) showing evidence of a peripheral nerve lesion; 3MFU – three-month follow-up; SPV – slow phase velocity; Tc – time constant; VFT1 – vestibular function testing at admission; VFT2 – vestibular function testing at discharge; VOR – vestibulo-ocular reflex



The DGI score improved over time with a mean score of 19/24 (IQR 4) at initial presentation, 23/24 (IQR 2) at discharge, and 24/24 (IQR 0) at follow up (Table 1 and Figure 2C).

Subjective visual vertical

Subjective visual vertical using the Rod & Disc test[16] showed little deviation with a static background. However, with a rotating background there was an asymmetric response with greater deviation when the background rotated towards the ipsilesional side. This improved over time (Figure 2D).

Patient reported outcomes (PROMS) and subjective report

Scores for all questionnaires improved over time with the minimal detectable change reached for the dizziness handicap inventory (> 18 points)[23] and the ABC-UK Scale (> 15%)[24] (See Table 2 and Figures 3A–D). At long term

***Footnote:** Supplementary version is available to download from https://www.dhmjournal.com/index.php/journals?id=361

Figure 2

Posturography (A+B) (note the difference in scale between A and B); dynamic gait index (C), and subjective visual vertical (D); data are shown as means and standard deviation. 3MFU – three month follow-up; FAEC – feet apart eyes closed; FAEO – feet apart eyes open; FTEC – feet together eyes closed; FTEO – feet together eyes open; TEC – tandem eyes closed; TEO – tandem eyes open; VFT1 – vestibular function testing at admission; VFT2 – vestibular function testing at discharge

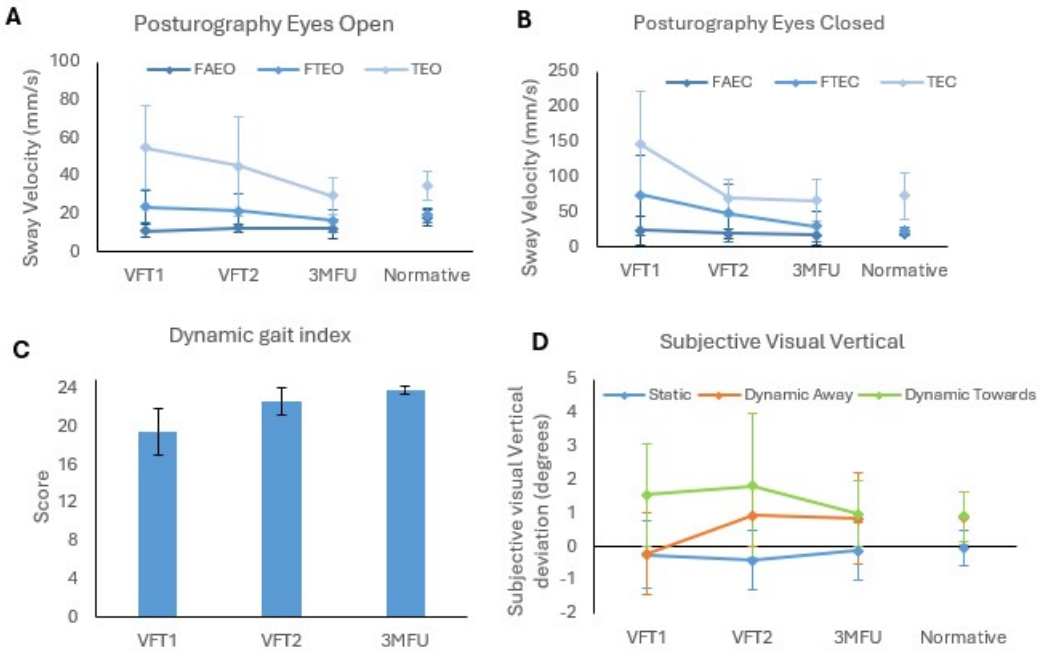


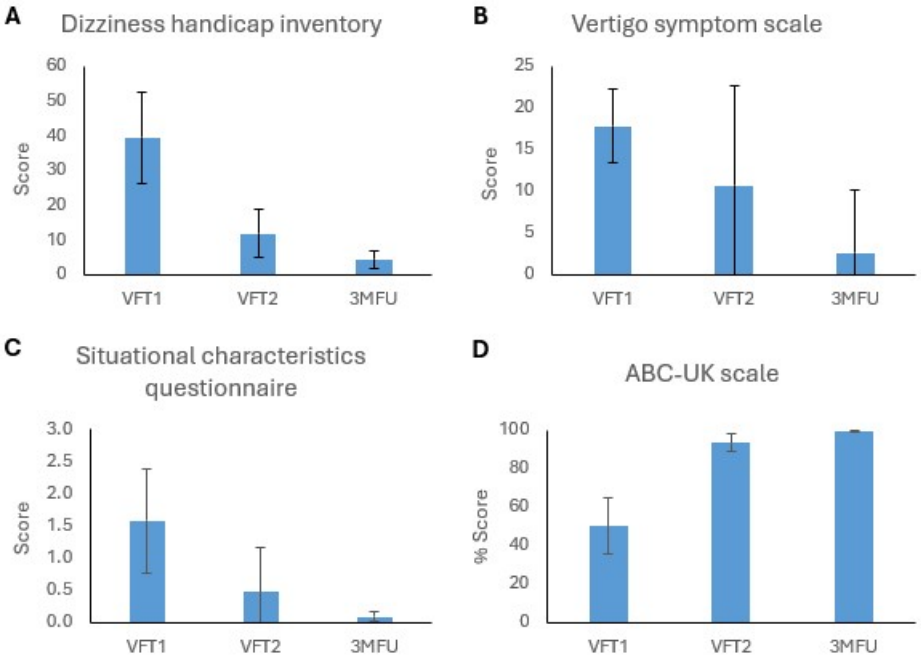
Table 2

Mean values for the patient reported outcome measures at admission, discharge and three-month follow-up

Measure	Admission (n = 8)	Discharge (n = 8)	Follow-up (n = 8)
Dizziness handicap inventory[17] > 10 = refer to balance specialist 16–34 = mild handicap 36–52 = moderate handicap ≥ 54 = severe handicap	39.50	11.75	4.29
Vertigo symptom scale Maximum = 60 > 12 = severe dizziness[36]	18.25	7.88	1.71
Situational Characteristic Questionnaire	1.58	0.47	0.08
Activities-specific balance confidence scale[37] Level of physical functioning, total = 100% 80% = high 50–80% = moderate < 50% = low	50.02%	93.46%	99.38%

Figure 3

Patient reported outcome measures (A-D) showing clinical improvement over time; data are shown as means and standard deviation. 3MFU – three-month follow-up; ABC-UK scale – activities-specific balance confidence scale; VFT1 – vestibular function testing at admission; VFT2 – vestibular function testing at discharge



follow up the divers anecdotally reported the return of some symptoms with fatigue, alcohol consumption, viral illness, and low light environments.

Personalised vestibular rehabilitation

All divers reported finding the rehabilitation exercises useful both in the acute phase and once discharged home. Customised exercises tended to focus on gaze stability, visual desensitisation and balance exercises in standing and walking in conditions of varying sensory information.[25,26] Complexity was increased by (a) increasing the speed of movement, (b) reducing the base of support and postural starting position, and (c) reducing the available sensory cues (e.g., eyes closed, removal of light touch support and standing on foam). Exercises targeted specific functional difficulties (e.g., standing at heights and painting) and were integrated in function task simulations/ activities. Exercises were prescribed for 5–10 minutes twice a day.[26] Visual dependence was determined through the Situational Characteristics Questionnaire, large (> 2 degree) visual vertical deviations with a moving visual scene and clinically, through a significant symptom increase when undertaking exercises when a moving visual scene was added. Visual dependence was addressed through visual desensitisation where exercises were performed in front of visual motion scenes of increasing complexity.[27]

Return to diving

Of interest, of the eight divers we have the information for, six returned to diving whilst two (with confirmed PFOs) were advised not to return to diving based on their residual subjective symptoms (including the diver who had a previous episode of IEDCS). Of those who returned to diving, four had confirmed PFOs; two had closures whilst the other two opted not have closures and returned to diving using recommended conservative strategies.[28] The two divers with negative bubble echocardiogram tests returned to diving with added conservatism. The six divers who returned to diving had no residual symptoms or signs of vestibular dysfunction on the standard dive medical examination. To date, we have not had reports of any further episodes of decompression illness.

Discussion

The mean age of the divers (53 years old) was not dissimilar to the average age of UK divers reported in diver surveys (~47 years old).[29] The gender of the divers was slightly more balanced than expected as there tends to be a larger male cohort in UK diving.[29] The average depth of the provocative dives was in line with previous case series of IEDCS[30] and the prevalence of an underlying PFO aligned with the average of 70% seen in other series.[31,32] The delay

to recompression (nine hours) was similar to cases in Israel (8.54 hours with those ≥ 72 hours excluded as outliers).[2]

At presentation all divers had evidence of a purely peripheral dysfunction with no central deficits seen on clinical or VNG testing. This was the case for divers presenting with purely vestibulo-cochlear symptoms as well as those with other symptoms of decompression sickness. Of the two divers with other symptoms, one had neurological signs (left leg weakness that rapidly resolved with oxygen administration), and one had cutaneous decompression sickness.

VOR gain following sinusoidal rotation increased by 7–8% over time. As rotary chair testing is expensive this test is not be used routinely in divers. Further, as rotation occurs at low velocities it is assessing both lateral canals and is therefore not as useful for diagnosing unilateral vestibular lesions compared to bilateral lesions, which are vanishingly rare in IEDCS. However, the step rotation test does allow an assessment of the time constant that involves both peripheral and central components and thus provides a possible window into central compensation processes. These showed a decrease when rotating to the side of the lesion and an increase when rotating away from the lesion over time suggestive of central compensatory changes.

Testing of visual perception with a static background showed little deviation. A deviation here may indicate deficits in utricle function.[33,34] Dynamic testing (with a moving background) did show changes with a greater visual vertical deviation when the background rotated towards the ipsilesional side. This reflects movement of the eyes in the direction of the VOR that drives the accompanying nystagmus. Whilst the dynamic results were as expected, the deviation was within the 2° error quoted in the literature and therefore should be interpreted with caution.[33,34]

Optokinetic nystagmus gain immediately after the lesion was higher when the stimulus moved towards the side of the lesion reflecting the asymmetry in the VOR response. However, as the optokinetic response requires a stimulus that covers the whole visual field, and the gain can be open to factors such as attention and instruction it is clinically less reliable.

Symptomatic improvement, as evidenced by the patient reported outcomes and posturography results, was seen in all divers despite an ongoing peripheral deficit in vestibular function seen in the head impulse test ± videonystagmography in five of the eight divers. This level of residual dysfunction (~70%) has been documented in other studies with divers with IEDCS[2,30] and should be a major consideration when assessing fitness to return to diving especially as the underwater environment can potentially contribute to disorientation. Poor visibility, swell, reduced visual reference and the potential for further insult to the inner ear (barotrauma, alternobaric vertigo, further IEDCS) could result in a hazardous situation.

Vestibular compensation is a well-documented phenomenon after a peripheral vestibular lesion with mechanisms involving adaptation, sensory substitution (central) and habituation identified.[27] VNG testing with step rotation showed a more symmetrical slow phase velocity time constant when comparing the ipsilateral and contralateral rotation over time. We hypothesise that over time central compensation focused on the brainstem structures involved in velocity storage (cerebellum, medial vestibular nuclei and prepositus hypoglossi nuclei) resulting in a shortened time constant on the contralateral side and reductions in left-right asymmetries that mediate occulomotor, perceptual and balance symptoms. We further hypothesise that targeted rehabilitation exercises could facilitate compensatory mechanisms to promote faster functional recovery.

Vestibular rehabilitation exercises should be considered for all divers presenting with IEDCS as a useful tool to aid recovery and in case of recurrence of symptoms after discharge. They are utilised in other unilateral vestibular diseases such as post neurectomy/schwannoma removal and vestibular neuritis with the mantra of 'the earlier the rehabilitation, the faster the recovery'. This reflects the presence of a critical period immediately after a lesion in animals where early engagement of the vestibular system leads to better recovery than delayed intervention (e.g., by 1–2 weeks).[27,35] In terms of the practicality of this, in the acute presentation of IEDCS we found that divers could be given the balance retraining booklet[21] to read during their first hyperbaric treatment in the chamber and to take away with them. This meant that they started the exercises on day one and were engaging in the rehabilitation process straight away. Once seen for the vestibular testing, they were given more personalised exercises including balance exercises and gaze stabilisation.

Dependence on vision was identified either clinically, via the Situational Characteristics Questionnaire or the vertical perception test. Very visually dependent people have a poorer spontaneous vestibular compensation as they tend to over rely on visual information for balance and orientation rather than using multi-sensory information (i.e., vision, somatosensory and remaining vestibular) and have difficulty resolving conflicts between vision and vestibulo-proprioceptive information. When using vision in isolation, errors can arise in interpreting self-motion from motion of the environment.[25,27,35] If visual dependence was identified, the strategy for rehabilitation included use of optokinetic videos and stimulus alongside the other exercises. Optokinetic training and stimulus videos are freely available online. We chose videos involving walking down a supermarket aisle or along a crowded street. These were displayed on a large

screen and the diver was initially asked to stand watching the video and describe their symptoms. If they were able to do this with ease, then the exercise progressed in difficulty with the diver asked to walk towards the screen whilst keeping fixated on the moving stimulus. This then progressed again by walking towards the screen and moving the head from side to side. This exercise was easy to recreate in the home environment and the divers could pick videos they found particularly provoked their symptoms.

Posturography demonstrated that use of the sharpened Romberg's (tandem eyes closed) was a reasonable method of assessing balance as a bedside test. Timing the duration of holding the stance using a stopwatch is recommended as a measurement of improvement (with consistency of self-selected leading foot and number of trials allowed).

The mean dynamic gait index (DGI) score at the initial testing point was 19 out of 24 which was better than expected as at initial presentation these divers often require a lot of support with standing and walking due to the vertigo and nausea (Table 1 and Figure 2A). However, it is plausible that this could be indicative of some improvement over the first day following initial hyperbaric recompression treatment as the testing session typically took place following this (to not delay treatment). As a diving physician, performing the DGI was a useful method of identifying movements that provoked symptoms and therefore formed a crude identification of initial exercises to suggest for rehabilitation. All divers scored 24 at follow-up which may represent a ceiling effect and a limitation in using the DGI. Going forward, the DGI pre- and post- the initial hyperbaric treatment will be checked to establish any improvement over this time. Over the three testing periods the overall change in DGI scores was five points, exceeding the minimal detectable change of four points quoted in the literature.[13]

The patient reported outcome measures were useful for identifying specific activities that the divers struggled with however, as they were designed for more chronic vestibular disorders, many of the questions were not applicable. A questionnaire designed for divers that covers symptoms occurring in both acute and chronic stages would provide better representation of this patient cohort.

This observational study of a case series documents real world practice therefore the number and timings of assessments was determined largely to target information for the clinical team rather than part of an investigative study. Choice of vestibular testing was based on available equipment rather than by design allowing only for investigation of the horizontal semicircular canals. Use of video head impulse testing and vestibular evoked myogenic potentials (VEMPS) will allow for assessment of the vertical semicircular canals and the otolith organs. Further research utilising these techniques is planned along with qualitative data collection.

Conclusions

Early initiation of vestibular rehabilitation exercises should be considered for all divers with IEDCS. Divers should be warned that there is a high rate of residual signs of vestibular deficit and that factors such as viral illness or alcohol consumption may interfere with the central compensation process even years after their injury. This deficit should also be a major consideration when considering returning to diving due to the risk of further decompression illness or barotrauma to the inner ear and poor environmental conditions inhibiting compensatory mechanisms. For the clinician, a stopwatch timed Sharpened Romberg's test (best of three with consistency of preferred leading leg) appears to be a reasonable method for monitoring progress of balance stabilisation during the treatment period. Clinical assessment should involve head impulse test and assessment of nystagmus with/without Frenzel's glasses. All divers should be educated regarding the possibility of an underling right to left cardiac shunt and offered referral to a cardiac specialist. Further investigation is needed to understand more about the mechanism of injury and recovery in IEDCS.

References

1 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.

2 Nachum Z, Shupak A, Spitzer O, Sharoni Z, Doweck I, Gordon CR. Inner ear decompression sickness in sport compressed-air diving. Laryngoscope. 2001;111:851–6. doi: 10.1097/00005537-200105000-00018. PMID: 11359165.

3 Landolt JP, Money KE, Topliff ED, Nicholas AD, Laufer J, Johnson WH. Pathophysiology of inner ear dysfunction in the squirrel monkey in rapid decompression. J Appl Physiol Respir Environ Exerc Physiol. 1980;49:1070–82. doi: 10.1152/jappl.1980.49.6.1070. PMID: 6969248.

4 Fraser WD, Landolt JP, Money KE. Semicircular canal fractures in squirrel monkeys resulting from rapid decompression. Interpretation and significance. Acta Otolaryngol. 1983;95:95–100. doi: 10.3109/00016488309130920. PMID: 6829307.

5 Doolette DJ, Mitchell SJ. Biophysical basis for inner ear decompression sickness. J Appl Physiol (1985). 2003;94:2145–50. doi: 10.1152/japplphysiol.01090.2002. PMID: 12562679.

6 Cantais E, Louge P, Suppini A, Foster PP, Palmier B. Right-to-left shunt and risk of decompression illness with cochleovestibular and cerebral symptoms in divers: case control study in 101 consecutive dive accidents. Crit Care Med. 2003;31:84–8. doi: 10.1097/00003246-200301000-00013. PMID: 12544998.

7 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/dhm53.3.243-250. PMID: 37718299. PMCID: PMC10735645.

8 Lee J, Kim K, Park S. Factors associated with residual symptoms after recompression in type I decompression sickness. Am J Emerg Med. 2015;33:363–6. doi: 10.1016/j.ajem.2014.12.011. PMID: 25636518.

9 Fitzgerald B. A review of the sharpened Romberg test in diving medicine. SPUMS Journal. 1996;26(3):142–6. PMID: 11539484. [cited 2025 Mar 17]. Available from: https://dhmjournal.com/images/IndividArticles/26Sept/Fitzgerald_SPUMSJ.26.3.142-146.pdf.

10 Shupak A, Doweck I, Greenberg E, Gordon CR, Spitzer O, Melamed Y, Meyer WS. Diving-related inner ear injuries. Laryngoscope. 1991;101:173–9. doi: 10.1288/00005537-199102000-00013. PMID: 1992269.

11 Cabrera Kang CM, Tusa RJ. Vestibular rehabilitation: rationale and indications. Semin Neurol. 2013;33:276–85. doi: 10.1055/s-0033-1354593. PMID: 24057831.

12 McDonnell MN, Hillier SL. Vestibular rehabilitation for unilateral peripheral vestibular dysfunction. Cochrane Database Syst Rev. 2015;1(1):CD005397. doi: 10.1002/14651858.CD005397.pub4. PMID: 25581507. PMCID: PMC11259236.

13 Shumway-Cook A, Woollacott M, Butler J, editors. Motor control, Theory and practical applications. 1st ed. Baltimore: Willaims & Wilkins; 1995.

14 Marsden J, Pavlou M, Dennett R, Gibbon A, Knight-Lozano R, Jeu L, et al. Vestibular rehabilitation in multiple sclerosis: study protocol for a randomised controlled trial and cost-effectiveness analysis comparing customised with booklet based vestibular rehabilitation for vestibulopathy and a 12 month observational cohort study of the symptom reduction and recurrence rate following treatment for benign paroxysmal positional vertigo. BMC Neurol. 2020;20(1):430. doi: 10.1186/s12883-020-01983-y. PMID: 33243182. PMCID: PMC7694922.

15 Marsden J, Stevenson V. Balance dysfunction in hereditary and spontaneous spastic paraparesis. Gait Posture. 2013;38:1048–50. doi: 10.1016/j.gaitpost.2013.03.001. PMID: 23587557.

16 Dichgans J, Held R, Young LR, Brandt T. Moving visual scenes influence the apparent direction of gravity. Science. 1972;178(4066):1217–9. doi: 10.1126/science.178.4066.1217. PMID: 4637810.

17 Jacobson GP, Newman CW. The development of the Dizziness Handicap Inventory. Arch Otolaryngol Head Neck Surg. 1990;116:424–7. doi: 10.1001/archotol.1990.01870040046011. PMID: 2317323.

18 Yardley L, Masson E, Verschuur C, Haacke N, Luxon L. Symptoms, anxiety and handicap in dizzy patients: development of the vertigo symptom scale. J Psychosom Res. 1992;36:731–41. doi: 10.1016/0022-3999(92)90131-k. PMID: 1432863.

19 Jacob RG, Redfern MS, Furman JM. Space and motion discomfort and abnormal balance control in patients with anxiety disorders. J Neurol Neurosurg Psychiatry. 2009;80:74–8. doi: 10.1136/jnnp.2007.136432. PMID: 18653552. PMCID: PMC4893779.

20 Powell LE, Myers AM. The Activities-specific Balance Confidence (ABC) Scale. J Gerontol A Biol Sci Med Sci. 1995;50A(1):M28–34. doi: 10.1093/gerona/50a.1.m28. PMID: 7814786.

21 Yardley L, Kirby S, Barker F, Little P, Raftery J, King D, et al. An evaluation of the cost-effectiveness of booklet-based self-management of dizziness in primary care, with and without expert telephone support. BMC Ear Nose Throat Disord. 2009;9:13. doi: 10.1186/1472-6815-9-13. PMID: 20098640. PMCID: PMC2810289.

22 Robinson DA, Zee DS, Hain TC, Holmes A, Rosenberg LF. Alexander's law: its behavior and origin in the human vestibulo-ocular reflex. Ann Neurol. 1984;16:714–22. doi: 10.1002/ana.410160614. PMID: 6441510.

23 Rizk HG, Velozo C, Shah S, Hum M, Sharon JD, Mcrackan TR. Item level psychometrics of the dizziness handicap inventory in vestibular migraine and Meniere's disease. Ear Hear. 2024;45:106–14. doi: 10.1097/AUD.0000000000001405. PMID: 37415269.

24 Shah G, Oates AR, Arora T, Lanovaz JL, Musselman KE. Measuring balance confidence after spinal cord injury: the reliability and validity of the Activities-specific Balance Confidence Scale. J Spinal Cord Med. 2017;40:768–76. doi: 10.1080/10790268.2017.1369212. PMID: 28875768. PMCID: PMC5778940.

25 Deveze A, Bernard-Demanze L, Xavier F, Lavieille JP, Elziere M. Vestibular compensation and vestibular rehabilitation. Current concepts and new trends. Neurophysiol Clin. 2014;44:49–57. doi: 10.1016/j.neucli.2013.10.138. PMID: 24502905.

26 Hall CD, Herdman SJ, Whitney SL, Cass SP, Clendaniel RA, Fife TD, et al. Vestibular rehabilitation for peripheral vestibular hypofunction: an evidence-based clinical practice guideline: From the American Physical Therapy Association neurology section. J Neurol Phys Ther. 2016;40:124–55. doi: 10.1097/NPT.0000000000000120. PMID: 26913496. PMCID: PMC4795094.

27 Pavlou M, Bronstein AM, Davies RA. Randomized trial of supervised versus unsupervised optokinetic exercise in persons with peripheral vestibular disorders. Neurorehabil Neural Repair. 2013;27:208–18. doi: 10.1177/1545968312461715. PMID: 23077146.

28 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.

29 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.

30 Lindfors OH, Lundell RV, Arola OJ, Hirvonen TP, Sinkkonen ST, Räisänen-Sokolowski AK. Inner ear decompression sickness in Finland: a retrospective 20-year multicenter study. Undersea Hyperb Med. 2021;48(4):399–408. PMID: 34847303.

31 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 2025 Mar 17]. Available from: https://dhmjournal.com/images/IndividArticles/45June/Mitchell_dhm.45.2.105-110.pdf.

32 Lacour M, Bernard-Demanze L. Interaction between vestibular compensation mechanisms and vestibular rehabilitation therapy: 10 recommendations for optimal functional recovery. Front Neurol. 2015;5:285. doi: 10.3389/fneur.2014.00285. PMID: 25610424. PMCID: PMC4285093.

33 Vibert D, Häusler R, Safran AB. Subjective visual vertical in peripheral unilateral vestibular diseases. J Vestib Res. 1999;9:145–52. Erratum in: J Vestib Res. 2000;10(1):57–8. PMID: 10378186.

34 Haijoub S, Hautefort C, Toupet M, Lacour M. Asymmetry and rehabilitation of the subjective visual vertical in unilateral vestibular hypofunction patients. Front Syst Neurosci. 2024;18:1454637. doi: 10.3389/fnsys.2024.1454637. PMID: 39318996. PMCID: PMC11419993.

35 Guerraz M, Yardley L, Bertholon P, Pollak L, Rudge P, Gresty MA, et al. Visual vertigo: symptom assessment, spatial orientation and postural control. Brain. 2001;124(Pt 8):1646–56. doi: 10.1093/brain/124.8.1646. PMID: 11459755.

36 Yardley L, Donovan-Hall M, Smith HE, Walsh BM, Mullee M, Bronstein AM. Effectiveness of primary care-based vestibular rehabilitation for chronic dizziness. Ann Intern Med. 2004;141:598–605. doi: 10.7326/0003-4819-141-8-200410190-00007. PMID: 15492339.

37 Myers AM, Fletcher PC, Myers AH, Sherk W. Discriminative and evaluative properties of the activities-specific balance confidence (ABC) scale. J Gerontol A Biol Sci Med Sci. 1998;53(4):M287–94. doi: 10.1093/gerona/53a.4.m287. PMID: 18314568.

Conflicts of interest and funding

This work was funded by the University of Plymouth and DDRC Healthcare as part of a PhD research programme. No conflicts of interest were declared.

Submitted: 18 March 2025
Accepted after revision: 22 June 2025

The role and efficacy of ECG screening in assessing fitness to dive in military divers: implications of sports medicine standards

Bas L van der Kooi[1], Alef DM Hoedemaeker[1], Lysette N Broekhuizen[2], Pieter-Jan AM van Ooij[1], Thijs T Wingelaar[1]

[1] *Royal Netherlands Navy, Diving and Submarine Medical Center, Rijkszee and Marinehaven, 1780 CA Den Helder, the Netherlands*
[2] *Ministry of Defense, Central Military Hospital, Utrecht, the Netherlands*

Corresponding author: *Dr Thijs T Wingelaar, Royal Netherlands Navy, Diving and Submarine Medical Center, Rijkszee and Marinehaven, 1780 CA Den Helder, the Netherlands*
ORCiD: *0000-0001-7740-7392*
tt.wingelaar@mindef.nl

Keywords
Cardiac; Cardiovascular; Diving; Electrocardiography; Health; Investigations

Abstract
(van der Kooi BL, Hoedemaeker ADM, Broekhuizen LN, van Ooij PJAM, Wingelaar TT. The role and efficacy of ECG screening in assessing fitness to dive in military divers: implications of sports medicine standards. Diving and Hyperbaric Medicine. 2025 30 September;55(3):246–253. doi: 10.28920/dhm55.3.246-253. PMID: 40986920.)
Introduction: Diving necessitates significant physiological adaptations, particularly within the cardiopulmonary system. Resting electrocardiograms (ECGs) are widely used in fitness to dive assessments, but their effectiveness in healthy young divers remains unclear. This study assessed the impact of applying sports medicine ECG criteria compared to traditional clinical standards, aiming to reduce (unnecessary) referrals to a cardiologist without compromising diver safety.
Methods: In this retrospective study covering 10 years, ECGs from Royal Netherlands Navy divers were analysed. Abnormal ECGs identified by clinical criteria between 2010 and 2019 were re-evaluated using international sports medicine ECG criteria. A control group of normal ECGs was matched based on demographic factors. Statistical analyses were performed using Pearson's chi-squared and Fisher's exact test, with significance set at $P < 0.05$.
Results: Of a total of 3,020 ECGs, 156 were classified as abnormal by clinical criteria. Reassessment using sports medicine standards reduced the number requiring further investigation by 85.9%. In the control group, 1.0% of previously unremarkable ECGs were identified as requiring further investigation upon reassessment. Conduction disorders and rhythm disturbances were the most common findings.
Conclusions: The findings of this study suggest that the application of sports medicine ECG interpretation criteria effectively reduces the number of ECGs requiring further investigation, thereby minimising referrals and associated costs. These results advocate for a re-evaluation of routine ECG screening practices in fitness to dive assessments in military divers, promoting a more tailored approach for this specific group.

Introduction

Immersion or submersion, such as in diving, necessitates significant physiological adaptations of the human body to maintain sufficient vital functions.[1] Pre-existing medical conditions, such as hypertension, cardiomyopathy, left ventricular hypertrophy (LVH) and valvular disease, can adversely affect these compensatory mechanisms, potentially leading to diving-related incidents and diseases.[2] While physiological changes are required across multiple systems, adaptations within the cardiopulmonary system are particularly critical. Specifically, central circulating blood volume may increase by 500 to 700 millilitres due to the redistribution of blood associated with submersion, resulting in heightened pressures within the atria and ventricles.[3,4] Additionally, exposure to cold water can induce peripheral vasoconstriction, further exacerbating cardiac pressures.[5] Such increases in cardiac stress during diving pose an increased risk of adverse cardiac events in individuals with underlying cardiac conditions, underscoring the necessity of fitness to dive assessments in preventing accidents.[6]

Sudden cardiac death (SCD) is a leading cause of mortality among young athletes during exercise. The majority of underlying conditions responsible for SCD can be identified through abnormalities on an electrocardiogram (ECG), including cardiomyopathies, electrical heart diseases and abnormal pathways.[7] In the field of diving medicine, the resting ECG is a widely utilised, cost-effective and non-invasive screening tool for identifying these overt electrical abnormalities indicative of cardiac pathology in fitness to dive assessments of both occupational and recreational

divers. However, the effectiveness of the resting ECG as a screening measure for cardiac issues among healthy, relatively young divers remains inadequately understood. Prior studies in general medicine suggest that screening asymptomatic young adults yields a low prevalence of abnormal ECG findings, with many of these categorised as normal variants, such as early depolarization.[8–10]

This uncertainty prompts critical questions regarding the ongoing utilisation of the ECG as a routine screening instrument in fitness to dive assessments, particularly given the substantial number of false-positive results when applying traditional ECG criteria to a young, usually sportive, population. Such outcomes can lead to unnecessary additional procedures and associated costs, while unjustly declaring military divers unfit for duty for extended periods, without clear benefits to diver safety.

Therefore, it is crucial to distinguish pathological abnormalities, such as cardiomyopathies and primary electrical disorders, which may manifest on an ECG as ventricular arrythmias or left bundle branch block (LBBB), from physiological adaptations to regular exercise or training, such as increased QRS voltage indicative of LVH, or (incomplete) right bundle branch block (RBBB). Recognising this need, an expert panel comprising specialists in sports cardiology, inherited cardiac disease and sports medicine convened in Washington in 2015 to revise the international criteria for interpreting ECGs in sports medicine. This update, based on emerging research, aimed to provide a better indication of commonly found ECG alterations in the young, asymptomatic and athletic population and may also be applicable to occupational divers.[11]

Currently, there is no international consensus on the criteria for interpreting resting ECGs in the context of fitness to dive assessments. In contrast to European and UK diving medical guidelines, Dutch guidelines mandate a resting ECG during fitness to dive assessments.[12,13] Despite this, research on this topic within the diving medicine literature remains limited. In aviation medicine, some evidence suggests that initial ECG screening for asymptomatic military aircrew leads to an extremely low number of individuals requiring further evaluation.[14] Possibly, selection bias may have contributed to this result. However, the relevance of these findings for military or occupational divers may be constrained, given the distinct physiological demands of diving compared to aviation and the differing criteria employed in ECG interpretation.

This study aimed to investigate the impact of modifying ECG interpretation criteria from clinical to sports medicine standards. We hypothesised that employing sports medicine criteria would reduce the number of ECGs requiring further investigation by a cardiologist, thereby decreasing unnecessary referrals.

Methods

The methods for handling medical information comply with national and European legislation and the guidelines of the Association of Universities in the Netherlands.

CONTEXT

The Royal Netherlands Navy Diving Medical Centre is responsible for the medical well-being of the Dutch armed forces' divers, submariners, and hyperbaric personnel. As mentioned in the introduction, the aforementioned group is subjected to annual medical assessments as part of national legislation.[13]

DATA COLLECTION

For this study, the ECGs from fitness to dive assessments, both initial screenings and revisions, of all divers between 1 January 2010 and 31 December 2019, were included. The conclusion of the assessment of the ECGs, conducted by the examining diving medicine physician on the day of the fitness to dive assessment, was recorded in a separate database. In the studied period, the Netherlands Armed Forces (including the Royal Netherlands Navy Diving and Submarine Medical Centre) classified ECGs according to the criteria of the AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram, I-VI, as published in 2007 and 2009.[15] These publications mention that ECG criteria may differ in several sub-groups, like young adults, but do not provide specific guidelines as to what may be considered normal in these groups. ECGs classified as abnormal according to these criteria were labelled as 'cases', and were matched by twice the number of normal ECGs from the entire dataset, labelled as 'controls'. This matching was based on criteria like initial screening or revision, smoking, age, height and weight. There was direct access of the examining diving medicine physicians to the cardiologists of the Central Military Hospital in Utrecht for consultation to discuss the interpretation of the ECGs. However, differences in ECG reading and interpretation skills among the examining diving medicine physicians may still have led to different interpretation and policy in similar cases.

The re-assessment for this study utilised the international criteria for interpreting ECGs in athletes, performed by two military diving medicine physicians (AH and TW) and a researcher (BK), who operated independently of each other.[10] In cases of differing interpretations, results were discussed until consensus was reached. If discrepancies arose among the evaluators, the ECGs were referred to a cardiologist (LB) for a final assessment. The results were coded in the database as 'normal variant' or 'further investigation required', as outlined in the aforementioned criteria. According to these criteria, certain findings may not be abnormal in isolation;

however, when considered alongside other findings, such as a right bundle branch block in conjunction with a right axis deviation, they may be deemed significant and necessitate further examination.

ANALYSIS

Data analysis was primarily descriptive. As the Kolmogorov-Smirnov test showed that none of the parameters were normally distributed, the median and interquartile range (IQR) were determined. All statistical analyses were performed with SPSS Statistics for Windows software (2022, version 29.0; IBM Corp; Armonk, NY). Differences were analysed with Pearson's Chi-squared test and Fisher's exact test where appropriate. Statistical significance was defined as $P < 0.05$.

Results

Between 1 January 2010 and 31 December 2019, a total of 3,020 ECGs were registered as part of fitness to dive assessments. Of these, 156 ECGs (5.2%) were labelled as abnormal. These ECGs were retrospectively re-examined using the international ECG criteria for athletes. From the 312 matched ECGs in the control group, a final total of 276 ECGs were analysed due to missing data. The baseline characteristics of the groups are presented in Table 1, with no significant differences observed between the groups.

The reassessment of the 'cases' and 'controls' revealed a total of 24 ECGs (0.8%) that required further investigation (see Figure 1). For the cases, this represents an 85.9% reduction in the number of ECGs requiring further investigation compared to the initial interpretation using clinical criteria. The majority of the 85.9% ECGs, which were identified as 'abnormal' at first assessment, were attributed to a complete right bundle branch block (cRBBB), left or right axis deviation and incomplete left bundle branch block (iLBBB) (Table 2). The rest of the abnormal ECGs were other findings such as ventricular extrasystoles, ST variations, intraventricular conduction disorders and sinus tachycardia. Despite two attempts at matching, data were unavailable for 36 individuals in the control group. No significant differences were found between the frequency of abnormalities of the 420 initial screenings and 2,600 revisions for the cases and controls ($P = 0.342$), which were assessed using the clinical criteria. These results are summarised in Table 3. Among the ECGs that required further investigation, the majority were classified as conduction disorders, including intra-ventricular conduction delay and bundle branch block (Table 4). The remaining ECGs predominantly exhibited rhythm disturbances.

In the control group, three ECGs were identified that required further investigation. Consequently, the application of the new criteria resulted in a 1.0% increase in abnormal ECG findings that were initially overlooked, disregarded or not reported by the examining physician during fitness to dive assessment. These abnormal findings included one instance of intra-ventricular conduction delay of 150 ms, a case with two premature ventricular ectopic beats per 10 s of tracing and a candidate with a prolonged corrected QT interval of 499 ms.

Discussion

This study provides evidence that the implementation of international criteria for interpreting ECGs in athletes appears to be effective in reducing the number of ECGs requiring further investigation and referrals to a cardiologist, while to date, none of the divers whose ECGs were reassessed have presented with any diving-related or other medical issues. This suggests a potential decrease in additional investigations, along with associated costs and

Table 1
Baseline characteristics of the included applicants for all applicants, cases and controls; IQR – interquartile range

Parameter	All applicants *n* = 3,020	Cases *n* = 156	Controls *n* = 312
Initial screening, *n*	420	25	54
Revision, *n*	2,600	131	258
Age (y), median (IQR)	31 (27–39)	30 (26–40)	30 (26–40)
Men, *n* (%)	2,977 (98.6)	156 (100)	312 (100)
Women, *n* (%)	43 (1.4)	–	–
Height (cm), median (IQR)	184 (179–188)	183 (179–189)	184 (180–189)
Weight (kg), median (IQR)	86 (80–92)	85 (79–94)	86 (81–92)
Smoking			
Yes, *n* (%)	472 (15.6)	31 (19.9)	62 (19.9)
No, *n* (%)	2,548 (84.2)	125 (80.1)	249 (80.1)

Figure 1

Classification of ECG findings for cases and controls showing the percentages of cases and controls within the total group, followed by the proportions of normal ECGs, those requiring further investigation, and missing ECGs within the cases and controls

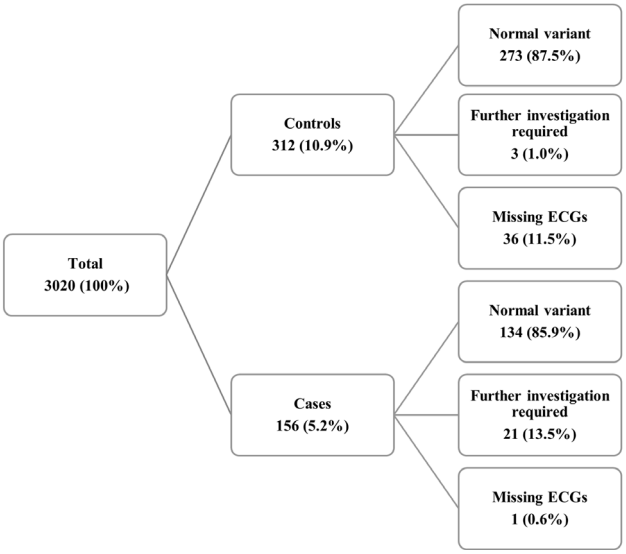


Table 2

Specific electrocardiogram (ECG) findings for the cases at first assessment; *left axis deviation: 30° to -90° in the frontal plane; #right axis deviation: 90° to 180° in the frontal plane; cRBBB – complete right bundle branch block (QRS duration greater than or equal to 120 ms with rsr', rsR' or rSR' pattern in leads V1 or V2); iLBBB – incomplete LBBB (QRS duration between 110 and 119 ms with broad notched or slurred R wave in leads I, aVL, V5, and V6, absent q waves in leads I, V5, and V6)

ECG findings classified as 'abnormal'	Cases *n* (% of total)
cRBBB	74 (55.2)
Left axis deviation*	19 (14.2)
iLBBB	14 (10.4)
Right axis deviation#	5 (3.7)
Other findings	22 (16.4)
Total	134

Table 3

Results of reassessed electrocardiograms for the cases and controls; *P = 0.342

Outcome	Cases: initial screening	Revision	Controls: initial screening	Revision	Total
Normal variant	21	113	48	225	407
Further investigation required	3	18	1	2	24
Total	24	131	49	227	431*

Table 4

Specific electrocardiogram (ECG) findings for the cases and controls; *left axis deviation: 30° to -90° in the frontal plane; #right axis deviation: 90° to 180° in the frontal plane; † ≥ 0.5 mm in depth in two or more contiguous leads; ‡ ≥ 470 ms (male), ≥ 480 ms (female) using Bazett's formula; cLBBB – complete left bundle branch block (QRS ≥ 120 ms with broad notched or slurred R wave in leads I, aVL, V5, and V6, absent q waves in leads I, V5, and V6; cRBBB – complete right bundle branch block (QRS duration greater than or equal to 120 ms with rsr', rsR' or rSR' pattern in leads V1 or V2); PVC – premature ventricular contractions (≥ 2 per 10 s)

ECG findings that required further investigation	Cases	Controls	Total
Profound nonspecific intra-ventricular conduction delay (QRS ≥ 140 ms)	9	1	10
cLBBB	4		4
cRBBB with right axis deviation#	2		2
cRBBB with left axis deviation*	2		2
PVC	2	1	3
ST-segment depression†	1		1
Epsilon wave	1		1
Prolonged QT interval‡		1	1

the risk of potential incidental findings of unknown clinical significance.

This reduced number of ECGs requiring further investigation is the result of major differences between ECG criteria derived from the 'AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram' and the ECG criteria derived from the 'International Recommendations for Electrocardiographic Interpretation in Athletes'. The main differences are the result of the, usually, lower weight and a more active lifestyle of young adults compared to older adults.

This results in ECG manifestations, that may be abnormal in the older population but normal in the young adult population. For instance, the usual voltage criteria for left and right ventricular hypertrophy do not apply to adults under the age of 35 years. High voltages are a normal finding in the young population. In the case of structural heart disease, for instance, a cardiomyopathy, there are usually additional ECG abnormalities like (deep) negative T waves. In addition, due to an increased vagal tone in young people, arrhythmias and conduction disturbances may occur like sinus bradycardia, nodal rhythms, premature atrial complexes (PACs), first degree atrio-ventricular (AV) block and second-degree Mobitz type 1 AV block (Wenckebach). Due to increased strain to the right ventricle caused by sports activities, the right ventricle may enlarge and an (incomplete) RBBB may appear. Also, repolarisation abnormalities like early repolarisation and non-specific ST and T wave abnormalities may appear in active young adults without clinical significance.

However, certain ECG abnormalities are also abnormal in young adults, like a complete LBBB, a QRS duration of > 140 ms, T wave inversion, ST segment depression ≥ 0.5 mm in depth in two or more contiguous leads, ≥ 2 PVCs on the ECG and atrial and ventricular arrhythmias. A left axis deviation, a left atrial enlargement, a right axis deviation, a right atrial enlargement and a complete RBBB are considered 'borderline', and further evaluation is not recommended if it occurs 'in isolation', but when two of more of these abnormalities are present, further evaluation is recommended. These recommendations are based on the finding that in athletes with these ECG abnormalities in isolation no abnormalities were found on echocardiography, especially those who engage in regular and long term participation in intensive exercise.[11] The most important changes in the assessment of ECGs comparing 'old' to 'new' criteria are the assessment of left axis deviation, right axis deviation and cRBBB with a duration < 140 ms in isolation as normal. These major changes together with a few of other findings resulted in a considerable reduction in abnormal ECGs.

To our knowledge, this is the first study to examine the application of these criteria in the context of fitness to dive assessments. A large study involving German military aircrew members analysed 6,284 ECGs and reported a prevalence of 0.21% of ECGs requiring further investigation.[14] This prevalence is approximately four times lower than observed in our study, which could be attributed to differences in physical fitness and exposure to environmental factors between divers and aircrew; for instance, left ventricular hypertrophy or an isolated left axis deviation are less likely to occur in non-athletes and are often a consequence of physiologic adaptation.[16] However, differences in national policy regarding screening and follow-up of findings may have also affected these differences. This could also explain the absence of profound nonspecific intra-ventricular conduction delay (QRS ≥ 140 ms) in the German study, which represented 42% of the abnormalities in the present study. Furthermore, we observed a 1.0% prevalence of ECGs requiring further investigation in the control group, prompting questions about the potential for missed abnormal findings across the entire dataset, even with our valid sample. It is reasonable to argue that ECG findings necessitating further investigation may exhibit low clinical relevance in practice since none of these divers has presented with diving related health issues or other medical problems leading to fatal diving incidents, which may justify the decision not to analyse all ECGs. Furthermore, even after reassessment, the risk of false-negative ECGs remains, as certain abnormalities such as intermittent Wolff-Parkinson-White, Brugada, atrial septal defect and long QT-syndrome, may not always be detectable on an ECG. Additionally, an ECG does not capture all forms of cardiac pathology, such as anomalies in the coronary arteries.

Several recent studies have evaluated the contribution of routinely performed tests, such as spirometry and audiometry, on medical assessment of fitness to dive.[17–19] Collectively, these studies advocate for a more selective approach to additional testing, emphasising the importance of subject history and physical examination in determining the necessity for further evaluation. One potential indicator for conducting a resting ECG is smoking status, as in our study there appeared to be an over-representation of smokers in the cases compared to all applicants. However, in young and fit candidates, smoking status is of little significance as coronary artery disease has not yet developed. Conversely, for divers older than 40 years old this could be very relevant, as smoking is a leading risk factor for cardiovascular diseases and mortality, particularly due to coronary artery disease and cardiac arrhythmias such as atrial fibrillation (AF).[20] Although the current study did not establish a statistically significant effect, a post-hoc power analysis revealed a study power of only 31.5%. Consequently, this study is underpowered to definitively relate smoking status to ECG findings using clinical criteria in healthy adults. Given that the application of sports medicine ECG criteria significantly reduced the number of ECGs requiring further investigation, this power is further diminished. Moreover, identifying cardiovascular risk factors in young athletes based on

history and physical examination may prove challenging, as cardiovascular diseases typically become clinically relevant starting in the fifth decade of life.

As a result, these findings cast further uncertainty on the role of resting ECGs in fitness to dive assessments, particularly with the goal of excluding ischaemia or coronary disease in military divers, though the implications may extend more broadly. While the resting ECG serves as a baseline for an exercise ECG, its added value remains to be determined. Different industries, such as recreational or commercial diving, assign varying levels of importance to resting ECGs. Aside from the prevalence of underlying disease, the decision whether or not to record an ECG at a dive medical assessment, or any other medical investigation for that matter, must also be based on the level of risk that an organisation is willing to accept. In the military diving domain, it may be deemed acceptable to forgo a resting ECG as a screening tool due to the generally fit and healthy population, where unnecessary referrals could leave a diver unfit for duty for extended periods. Conversely, cardiac complaints in operational settings could incapacitate divers, jeopardizing missions, although to our knowledge there have been no incapacitations of divers in the Royal Netherlands Armed Forces that were cardiac related.

For commercial or recreational diving, these considerations may be evaluated differently due to distinct population characteristics and varying costs associated with incidents. Therefore, a more realistic approach to risk assessment and the use of medical screening tools should be carefully considered. Specifically for our military population, we propose that a resting ECG at the start of a diving career may be warranted to exclude disease which can be potentially lethal for divers deployed to locations with limited access to regular medical care, such as underlying arrhythmogenic pathology such as genetic cardiomyopathies, long-QT, Brugada, or Wolff-Parkinson-White syndrome.

Of special importance to diving is the association of a right bundle branch block (RBBB) on the ECG with the presence of an atrial septal defect. In a retrospective study of 104,369 young (sportive) individuals who were analysed, and in whom also a 'selective' echocardiogram was performed, a group of 154 individuals with a complete RBBB was identified, of which seven had cardiac pathology. Four of these individuals had an ASD on echocardiogram (three others had a Brugada syndrome, progressive cardiac conduction disease and atrial fibrillation, respectively). All of the individuals could be identified using the international criteria for ECG interpretation in athletes (six had an axis deviation, one had a QRS duration of 141 ms). The authors suggest that complete RBBB should remain as a 'borderline' finding but only if the QRS duration is < 130 ms. Complete RBBB of ≥ 130 ms should be considered an 'abnormal' finding, warranting secondary evaluation for all with at least an echocardiogram.[21]

Conversely, after ascertaining the presence of an initial normal ECG, routinely performed ECGs, without a clinical indication, seems to be of very little value and could be omitted. Without clinical indications, it seems valid to exclude routine resting ECGs in healthy military divers. However, around the age of 45 years old the risk of cardiovascular disease and cardiac related death in diving increases, which may warrant continuation of ECG screening in older divers.[3,22] Whether this age is appropriate for military divers, who are generally in a better physical condition than the general population and at a lower risk for cardiovascular disease, to commence routine ECG screening remains to be determined.

STRENGTHS AND LIMITATIONS

To our knowledge, this study represents the first retrospective analysis of resting ECGs utilising criteria established in sports medicine during medical assessments of fitness to dive in healthy, relatively young and asymptomatic military divers. A notable strength of this study is the comprehensive analysis of a large sample of resting ECGs over an extended period of time. Furthermore, all examinations and assessments were performed under standardised conditions, employing consistent ECG criteria across multiple independent assessors.

However, this study has several limitations. First, the study population is highly selected, as healthy male and mainly young military divers probably are not representative of female divers and older, commercial and recreational divers. Nonetheless, it is relatively straightforward to ascertain whether divers in other sectors align with these findings or may be at greater risk for cardiac disease based on history and physical examination. We encourage colleagues to conduct and publish similar studies to determine if the results of this study can be applied across different sectors. Secondly, despite two matching attempts, the control group contained 36 missing ECGs, which slightly reduced its size compared to initial expectations. However, as the control group remained substantial and comparable to the overall group, we believe it is still representative and may not have affected the conclusions drawn from this paper. Lastly, and perhaps most importantly, not all cases in this study received a thorough evaluation by a cardiologist, even though referral to a cardiac specialist was deemed appropriate at the time. The reason for this varies from candidates' withdrawal from the process to omissions from the assessing physician. This is unfortunately the case in many retrospective analyses, and may obscure the 'true' presence of cardiac disease and could have affected the prevalence of 'true' cardiac pathology in our group. However, over the past three decades, there have only been two fatal diving accidents in the Royal Netherlands Armed Forces, neither of which was attributed to cardiac pathology.

Conclusions

This study demonstrates that the application of ECG interpretation criteria derived from sports medicine standards effectively reduces the number of ECGs requiring further investigation compared to traditional clinical criteria. This reduction is likely to decrease referrals to a cardiologist and minimise the necessity for additional investigations, along with their associated costs and potential incidental findings. As these findings are often of unknown clinical significance, a more nuanced approach to risk assessment and the utilisation of medical screening tools is warranted. We recommend that a resting ECG can be considered for military divers as part of an initial dive medical assessment, especially when deployed to locations with limited access to routine medical care, to screen for potentially lethal rhythm or conduction disorders and cardiomyopathies, but that subsequent routine ECGs without clinical indication can be omitted. Future research should investigate the generalisability of these findings to other diver populations, particularly females and older male divers. Additionally, studies should aim to determine which risk factors, including age, should prompt recording of an ECG as part of a fitness to dive assessment.

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 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.

3 Rienks R, Buwalda M, Bucx J, Dubois E, Wingelaar T, van Hulst R. Cardiovascular risk assessment in divers: toward safer diving. Undersea Hyperb Med. 2022;49:355–65. PMID: 36001568.

4 Marabotti C, Scalzini A, Menicucci D, Passera M, Bedini R, L'Abbate A. Cardiovascular changes during SCUBA diving: an underwater Doppler echocardiographic study. Acta Physiol (Oxf). 2013;209:62–8. doi: 10.1111/apha.12112. PMID: 23638629.

5 Weenink RP, Wingelaar TT. The circulatory effects of increased hydrostatic pressure due to immersion and submersion. Front Physiol. 2021;12:699493. doi: 10.3389/fphys.2021.699493. PMID: 34349668. PMCID: PMC8326965.

6 Wendling J, Vanden Eede R, Elliott D, Meintjes J, Meliet J-L, Nome T, editors. Medical assessment for work under pressure. Fitness to dive standards of European Diving Technology Committee. International Marine Contractors Association; 2024.

7 Maron BJ, Doerer JJ, Haas TS, Tierney DM, Mueller FO. Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980-2006. Circulation. 2009;119:1085–92. doi: 10.1161/CIRCULATIONAHA.108.804617. PMID: 19221222.

8 Harskamp RE. Electrocardiographic screening in primary care for cardiovascular disease risk and atrial fibrillation. Prim Health Care Res Dev. 2019;20:e101. doi: 10.1017/s1463423619000355. PMID: 32800007. PMCID: PMC8060828.

9 Chamley RR, Holdsworth DA, Rajappan K, Nicol ED. ECG interpretation. Eur Heart J. 2019;40(32):2663–6. doi: 10.1093/eurheartj/ehz559. PMID: 31433846.

10 Ashley EA, Raxwal V, Froelicher V. An evidence-based review of the resting electrocardiogram as a screening technique for heart disease. Prog Cardiovasc Dis. 2001;44:55–67. doi: 10.1053/pcad.2001.24683. PMID: 11533927.

11 Sharma S, Drezner JA, Baggish A, Papadakis M, Wilson MG, Prutkin JM, et al. International recommendations for electrocardiographic interpretation in athletes. Eur Heart J. 2018;39(16):1466–80. doi: 10.1093/eurheartj/ehw631. PMID: 28329355.

12 Health and Safety Executive. Medical examination and assessment of working divers (MA1); 2023. [cited 2024 Oct 9]. Available from: https://www.hse.gov.uk/pubns/ma1.htm2023.

13 Working Under Pressure Foundation. Guideline occupational health assessment: Working under positive pressure and diving [in Dutch: Keuringsrichtlijn. Arbeidsgezondheidskundig Onderzoek Werken onder Overdruk en Duikarbeid]. Pijnacker, the Netherlands: Working Under Pressure Foundation; 2021. [cited 2024 Oct 9]. Available from: https://www.arbocataloguswoo.nl/images/SWOD/pdf/NL/SWOD-ARBOCAT-WoO-Keuring-CAT003_1-2021-rev4-aug-2021.pdf.

14 Guettler N, Sammito S. Electrocardiographic abnormalities in medically screened German military aircrew. J Occup Med Toxicol. 2021;16(1):37. doi: 10.1186/s12995-021-00327-x. PMID: 34465360. PMCID: PMC8407018.

15 Surawicz B, Childers R, Deal BJ, Gettes LS, Bailey JJ, Gorgels A, et al. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part III: intraventricular conduction disturbances: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society: endorsed by the International Society for Computerized Electrocardiology. Circulation. 2009;119(10):e235–40. doi: 10.1161/CIRCULATIONAHA.108.191095. PMID: 19228822.

16 Petek BJ, Drezner JA, Churchill TW. The international criteria for electrocardiogram interpretation in athletes: common pitfalls and future directions. Cardiol Clin. 2023;41:35–49. doi: 10.1016/j.ccl.2022.08.003. PMID: 36368810. PMCID: PMC10292923.

17 Sames C, Gorman DF, Mitchell SJ, Zhou L. Long-term changes in spirometry in occupational divers: a 10-25 year audit. Diving Hyperb Med. 2018;48:10–6. doi: 10.28920/dhm48.1.10-16. PMID: 29557096. PMCID: PMC6467824.

18 Canarslan-Demir KC, Ozgok-Kangal K, Kilic S, Genc H. Does diving deteriorate hearing functions? Undersea Hyperb Med. 2023;50:313–8. PMID: 37708065.

19 Melessen A, Wingelaar TT, van Ooij PA. Clinical utility of dipstick urinalysis in assessing fitness to dive in military divers, submariners, and hyperbaric personnel. Diving Hyperb Med. 2024;54:105–9. doi: 10.28920/dhm54.2.105-109. PMID: 38870952. PMCID: PMC11444914.

20 Irfan A, Riggs DW, Koromia GA, Gao H, DeFilippis AP, Soliman EZ, et al. Smoking-associated electrocardiographic abnormalities predict cardiovascular mortality. Sci Rep.

2024;14(1):31189. doi: 10.1038/s41598-024-82503-7. PMID: 39730843. PMCID: PMC11680829.

21 MacLachlan H, Antonakaki A, Bhatia R, Fyazz S, Chatrath N, Androulakis E, et al. Prevalence and clinical significance of electrocardiographic complete right bundle branch block in young individuals. Eur J Prev Cardiol. 2025;32:1054–61. doi: 10.1093/eurjpc/zwae082. PMID: 38412448.

22 Jepson N, Rienks R, Smart D, Bennett MH, Mitchell SJ, Turner M. South Pacific Underwater Medicine Society guidelines for cardiovascular risk assessment of divers. Diving Hyperb Med. 2020;50:273–7. doi: 10.28920/dhm50.3.273-277. PMID: 32957130. PMCID: PMC7819720.

Conflicts of interest and funding: nil

Submitted: 13 January 2025
Accepted after revision: 8 June 2025

Review article

Assessing dive fitness in individuals with autism spectrum disorder

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

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

Corresponding author: *Dr Abraham L Querido, Praktijk Querido, Veerstraat 25, 1211 HJ Hilversum the Netherlands*
ORCiD: *0009-0001-1988-0832*
bram@praktijkquerido.nl

Keywords
Fitness-to-dive; Mental health; Psychiatry; Scuba

Abstract

(Querido AL, Wingelaar TT. Assessing dive fitness in individuals with autism spectrum disorder. Diving and Hyperbaric Medicine. 2025 30 September;55(3):254–261. doi: 10.28920/dhm55.3.254-261. PMID: 40986921.)
Scuba diving requires situational awareness, cognitive flexibility, and the ability to adapt to changing conditions. For individuals with autism spectrum disorder (ASD), these demands may pose unique challenges due to differences in executive functioning, sensory processing, and social cognition. This article explores the key considerations in assessing fitness to dive in individuals with ASD, including the impact of comorbidities, medication use, and cognitive abilities on diving safety. To provide a broader perspective, we examine research on ASD and high-risk activities such as driving, where similar cognitive and decision-making challenges exist. Additionally, we discuss the role of neuropsychological assessments in evaluating a diver's cognitive fitness and the limited but emerging evidence on scuba diving interventions for individuals with ASD. While ASD is not an absolute contraindication to diving, a careful, individualised assessment is essential to determine suitability. This review aims to provide guidance for diving professionals and medical examiners in making informed decisions regarding ASD and scuba diving.

Introduction

Evaluating fitness to dive in individuals with psychiatric conditions, such as autism spectrum disorder (ASD), is considered one of the more complex assessments in diving medicine. One Dutch study found that psychiatric conditions accounted for 9.6% of 291 cases presented to the board of experts over the decade-long analysis.[1] This highlights that while circulatory and respiratory diseases were the most prevalent, psychiatric issues also represented a significant portion of the cases encountered by medical examiners of diving (MEDs). While seemingly straightforward, this question quickly leads to further complexities. In the Netherlands, scuba diving is classified as an extreme sport by insurance providers; therefore, it is important to determine whether having ASD presents additional risks. ASD exists on a spectrum, meaning individual abilities and challenges vary widely, so if risks exist, do they apply universally or only to certain individuals?

ASD itself is not inherently a contraindication to diving, but certain risk factors, including impaired cognitive flexibility, comorbidities and medication use, play a significant role in determining fitness to dive. Individuals with impaired cognitive flexibility, for example, may struggle to adapt to unexpected situations or disengage from rigid thought patterns. This can be particularly problematic in diving, where rapid problem-solving and adaptability are crucial for safety. Given these considerations, an individualised evaluation is essential.

To provide colleagues who are less familiar with ASD with a clearer understanding of its complexities, we present a real-life case of a 13-year-old girl with ASD to illustrate key challenges, followed by a review of the literature on ASD and its implications for diving. The goal is to provide an evidence-based framework for evaluating whether individuals with ASD can safely participate in scuba diving.

Case example

The case is a 13-year-old girl diagnosed with ASD, attention deficit disorder (ADD), and Gilles de la Tourette syndrome, according to DSM-5 criteria. She is currently prescribed methylphenidate (a slow-release psychostimulant) 36 mg once daily for ADD and aripiprazole (antipsychotic) 12.5 mg once daily to manage tics. A comprehensive neuropsychological assessment provided critical insights into her cognitive functioning. While her overall intelligence falls within the average range, she presents with a disharmonic

intelligence profile, meaning significant discrepancies exist between her cognitive abilities. Specifically, she demonstrates notable impairments in attention, characterised by slow and inconsistent information processing speed; working memory, making it difficult for her to retain and manipulate information in real time; and executive functioning, leading to challenges in planning, impulse control, and cognitive flexibility.

These cognitive deficits make her highly susceptible to sensory overload, as she struggles to process and regulate external stimuli effectively. As a result, tension and anxiety escalate, causing her to miss critical information and struggle to respond appropriately. In overwhelming situations, she may become stuck in repetitive behaviours or experience a cognitive shutdown, further compounding her frustration. This, in turn, intensifies stress, anxiety, and feelings of insecurity. Given these cognitive and emotional vulnerabilities, the question arises: how do these factors influence her ability to engage in demanding activities such as scuba diving?

Methods

The protocol for the literature search strategy was prepared according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA).[2] A structured search of the literature was performed using PubMed up to 27 January 2025 to identify studies and case reports regarding diving and autism. A query involving diving and autism resulted in very few results, therefore, the keywords were expanded to include aviation and driving. Additionally, several handbooks on diving medicine that discussed psychiatry or psychology were screened for additional information. The full query was structured as: ((diving[Mesh] OR dive[tw] OR diving[tw] OR divers[tw] OR hyperbaric[tw] OR scuba[tw]) OR (aviation[mesh] OR flying[tw] OR altitude[tw]) OR (driving[mesh] OR driv*[tw] OR traffic[tw])) AND ((autism[Mesh] OR autism[tw])).

A systematic literature search conducted in January 2025 screened 2,417 studies. Of these, 59 were identified as potentially relevant, including two on water-based interventions, 23 on hyperbaric oxygen therapy (HBOT), and 34 on driving and ASD. After assessing titles and abstracts, 32 studies were deemed eligible for inclusion: two on water-based interventions, 10 on HBOT, and 20 on driving.

A total of 25 articles were excluded: 12 on driving and ASD, as well as 13 on HBOT for ASD, including two reviews due to quality concerns, and 11 clinical studies. A detailed discussion of clinical trials falls beyond the scope of this article. Additionally, many studies had methodological limitations that contributed to inconsistent findings.

The reference lists of the included papers were also used to identify additional studies. After carefully reading these articles, a total of nine of these papers were included in the present review. More details can be found in Figure 1.

Understanding ASD

ASD is a neurodevelopmental condition classified in the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5).[3] It is characterised by deficits in social communication and interaction, as well as restricted or repetitive behaviours or interests. The severity and presentation of these traits vary widely among individuals,

Figure 1
PRISMA diagram for results of systematic literature search

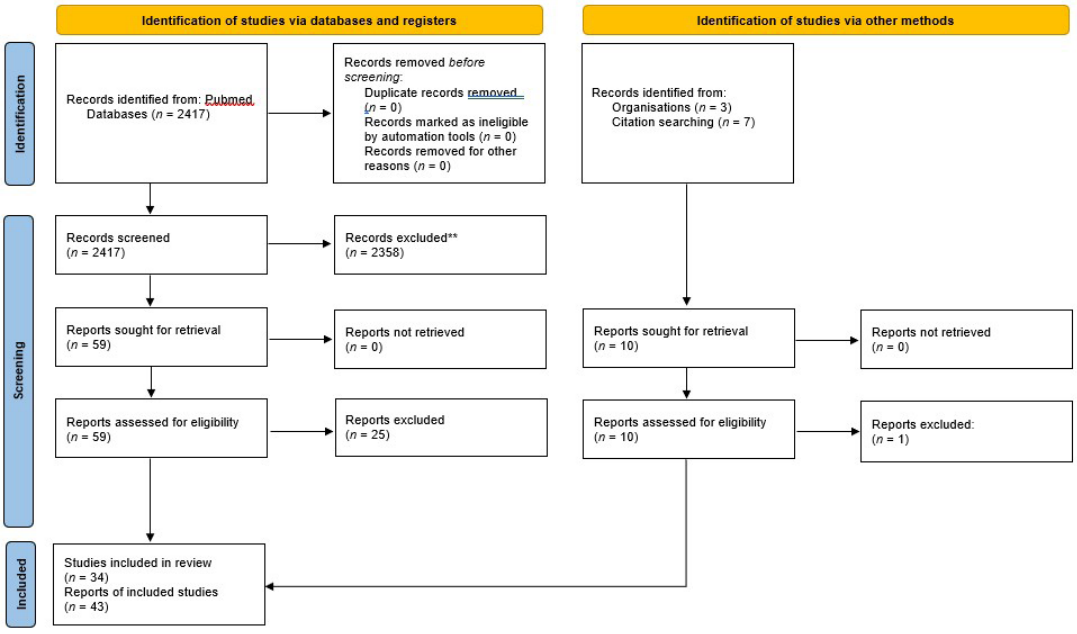


Table 1
Core features of autism spectrum disorder

Domain	Criteria
Social communication and interaction	1. Difficulties with social reciprocity 2. Difficulties in nonverbal communication used for social interaction 3. Deficits in developing and maintaining relationships with other people
Restricted and repetitive behaviour, interests, or activities	1. Stereotyped speech, repetitive motor movements 2. Rigid adherence to routines, ritualised patterns of verbal or nonverbal behaviours, and extreme resistance to change 3. Highly restricted interests with abnormal intensity or focus 4. Increased or decreased reactivity to sensory input or unusual interest in sensory aspects of the environment

the DSM-5 offers 3 levels severity (requiring support, substantial support or very substantial support). These core features of ASD are summarised in Table 1.

EXECUTIVE FUNCTIONS AND THEIR ROLE IN ASD

Executive functions are a set of cognitive processes essential for planning, organising, regulating, and adapting behaviour to achieve a specific goal. There are three core executive functions: working memory (ability to hold and manipulate information over short periods), inhibition (capacity to resist distractions, control impulses, regulate behaviour) and cognitive flexibility (ability switch between tasks, adapt to unexpected situations, and think creatively).[4,5] Executive functions continue to mature through childhood and into early adulthood. Cognitive flexibility begins to develop in early childhood, it continues to mature throughout adolescence and into early adulthood, influenced by brain development and various environmental factors.[6]

Executive function is commonly impaired in individuals with ASD.[7] Deficits in cognitive flexibility are particularly notable in ASD, making transitions, unpredictable events, and sudden environmental changes challenging. This difficulty in adjusting to new circumstances can be especially problematic in activities that require quick decision-making, such as driving, or diving.

Another key cognitive function affected in ASD is 'theory of mind', the ability to understand and interpret the thoughts, emotions, and intentions of others. Theory of mind is crucial for driving, as drivers must anticipate and respond to the actions of others to navigate safely. A study found that both theory of mind and executive function were strongly associated with driving performance in individuals with ASD, suggesting that better theory of mind and executive function skills correlate with safer driving behaviours.[8] Specifically, drivers with stronger executive function and theory of mind were better at recognising social hazards and making appropriate driving decisions. Given the cognitive demands of diving, assessing executive functioning, theory of mind and cognitive flexibility in individuals with ASD is crucial for determining fitness to dive. Standardised neuropsychological tests administered by a qualified mental health professional may be used to assess these cognitive domains, such as the Trail Making Test and the Wisconsin Card Sorting Test.

Comorbidities and their impact on diving

Comorbidity is highly prevalent among individuals with ASD. A large-scale study by Khachadourian et al., including over 42,000 individuals with ASD, found that 74% had at least one comorbid condition. Similarly, a systematic review by Bougeard et al., reported a comorbidity prevalence ranging from 54.8% to 94%. In a nationwide Swedish twin study, Lundström et al., found that 95% of individuals with ASD had at least one coexisting disorder, with more than half (50.3%) experiencing four or more comorbid conditions. These findings underscore the significant burden of comorbidities in ASD, emphasising the need for a comprehensive clinical approach.[9–11] Common comorbidities include psychiatric conditions, such as anxiety disorders, mood disorders, attention-deficit/hyperactivity disorder (ADHD) and neurological conditions like Gilles de la Tourette syndrome and epilepsy. Given the heterogeneity of language and cognitive abilities among individuals with ASD, it is important to tailor communication approaches during dive instruction and assessment. Where learning difficulties or language disorders are present, emphasising clear, positive instructions, focusing on what actions to take rather than what to avoid, may enhance understanding and safety.

Comorbidities can have important implications for diving safety. Epilepsy, characterised by unprovoked and recurrent seizures, is considered an absolute contraindication to diving due to the risk of seizures underwater, which could result in drowning.[12]

Gilles de la Tourette syndrome is a relative contraindication due to the potential risks associated with motor tics. Even

seemingly minor tics, such as coughing, throat clearing, or grunting, may obstruct airflow during ascent, increasing the risk of pulmonary barotrauma.[13]

Given the high prevalence of psychiatric and neurological comorbidities in ASD, a thorough medical evaluation is necessary to assess individual risks before determining diving fitness.

Medication use in ASD and considerations for diving

Certain medications are commonly used to manage behavioural symptoms in individuals with ASD, although no drug has been approved to target its core features. Aripiprazole and risperidone are the only medications approved by the U.S. Food and Drug Administration for treating irritability associated with ASD in children and adolescents.[14] However, no medications have been developed to target the core symptoms of ASD. In clinical practice, other medications are frequently prescribed off-label to manage specific symptoms.[15]

A study found that children with ASD were seven times more likely to use medications from major psychiatric drug classes (e.g., stimulants, antipsychotics, antidepressants, and mood stabilisers) and 21 times more likely to use multiple classes of these medications.[16] For example, stimulants are often used to enhance focus and reduce hyperactivity and antidepressants may help reduce irritability, aggression, and repetitive behaviours.[17]

DIVING CONSIDERATIONS AND PSYCHOTROPIC MEDICATIONS

Severe adverse effects of psychotropic medications are rare. Most individuals tolerate these drugs well and only experience mild side effects.[18] However, certain medications may increase sensitivity to nitrogen narcosis or oxygen toxicity, necessitating depth limitations.[19,20] Diving with psychotropic medication is often possible, but a careful risk assessment is required. Combining multiple medications may heighten the risk of side effects, making diving potentially unsafe.[21] Given these considerations, a thorough medical evaluation is essential for divers using psychotropic medication, particularly those on multiple drug regimens.

Scuba diving as an intervention for ASD

There has been increasing interest in water-based interventions, including scuba diving, for individuals with neurological disabilities, autism, and intellectual disabilities. However, evidence supporting the effectiveness of these interventions remains limited and of low quality overall.[22] A systematic review of water-based interventions found that only four studies met inclusion criteria, with just one study including individuals with ASD (three out of 23 participants).[23] These participants completed a specialised scuba training program provided by Disabled Divers International. While the review noted that participants generally enjoyed the experience and reported some improvements in self-concept, the quality of evidence was low, and no conclusive benefits were established. Further controlled studies are necessary to determine whether scuba diving has measurable therapeutic effects for individuals with ASD.

Although scuba diving may be a rewarding activity for some individuals with ASD, its safety and suitability should be assessed on a case-by-case basis, particularly in individuals with executive function challenges or sensory processing difficulties.

Hyperbaric oxygen therapy and ASD

Hyperbaric oxygen therapy (HBOT) has been proposed as a potential intervention for ASD, primarily based on neuroimaging findings indicating cerebral hypoperfusion in certain brain regions, particularly the temporal lobes.[24] This has led to the hypothesis that HBOT could increase oxygen delivery, potentially improving symptoms in individuals with ASD. Additionally, proposed pathophysiological mechanisms in ASD include inflammation, mitochondrial dysfunction, immune dysregulation, and oxidative stress factors that HBOT might theoretically modulate.[25]

However, scientific evidence does not support the routine use of HBOT for ASD. Multiple reviews have evaluated HBOT for ASD and consistently concluded that no solid evidence supports its effectiveness in improving core or associated symptoms.[26–32] The Undersea and Hyperbaric Medical Society (UHMS) does not recommend HBOT as a treatment for symptoms of ASD.[33]

Camouflaging in ASD

Camouflaging in individuals with ASD, particularly among women, though also observed in men, refers to the use of conscious coping strategies to conceal autism-related behaviours in social settings.[34] This includes efforts to appear socially adept and to mask or compensate for difficulties in communication and interaction. Camouflaging may also involve suppressing internal experiences such as anxiety and sensory overload, making it harder for others to detect distress.[35]

ASD and driving

Driving is a highly complex neuropsychological task that demands strong executive functioning, multitasking abilities, and a combination of psychomotor and cognitive skills, including attention, motor execution, memory, and navigation.[36,37]

Several studies indicate that drivers with ASD experience challenges in areas such as anxiety management, multitasking, decision-making in complex traffic situations, recognising

and responding to social hazards posed by other road users, environmental information processing, and coordination and timing.[37–43]

Individuals with ASD may experience attentional capture issues while driving, making it more difficult to focus on critical hazards.[44] They tend to notice hazards more slowly and may fixate longer on threats once detected, though not necessarily on the most relevant areas. Their attention distribution can be suboptimal, leading to delays in hazard recognition. Additionally, a high cognitive load or complex driving situations may further impair their ability to manage attention effectively, increasing safety concerns in rapidly changing traffic conditions.

Sheppard distinguished three specific areas of driving difficulty for autistic drivers regarding executive functioning (such as maintaining focus and handling multitasking while driving), cognition (interpreting traffic rules and understanding driving situations) and social awareness (interacting with other road users and understanding social cues while driving).[45]

Despite these challenges, research suggests that autistic drivers often adopt a cautious and structured driving style, demonstrating strengths in rule-following and adherence to traffic regulations. Compared to neurotypical drivers, they are less likely to engage in risky driving behaviours.[42,46–49]

A study on medical impairments in older drivers has suggested that ASD may contribute to crash risk, though its low prevalence in this population limits the reliability of these findings.[50] A recent review emphasises that while executive functioning and social awareness can present significant challenges, autistic drivers may compensate through careful planning and avoidance strategies, potentially contributing to safer driving outcomes compared to non-autistic drivers.[51]

Most studies that used driving simulators have reported higher accident rates and more driving difficulties among individuals with ASD compared to neurotypical individuals.[40,52–55] However, one study comparing newly licensed drivers with and without ASD, as well as experienced drivers, found that while ASD drivers performed worse in the simulator and were rated as less safe, fewer differences were observed during actual on-road driving.[56]

CASE EVALUATION: ASSESSING FITNESS TO DIVE IN A DIVER WITH ASD

We return to the 13-year-old girl diagnosed with ASD, attention deficit disorder, and Gilles de la Tourette syndrome described earlier.

Underwater situations involving unexpected challenges, such as equipment malfunction or abrupt environmental changes, require cognitive flexibility, and divers with impairments in this area may find it difficult to respond appropriately. Instead of adjusting their actions, they might become stuck in ineffective responses or fail to initiate an appropriate solution. This inability to shift strategies can result in sensory overload, escalating anxiety, and an increased risk of panic.

For this young diver, processing and regulating sensory stimuli is already challenging, particularly in complex, overstimulating environments. When overwhelmed, she may fail to extract relevant information, leading to rigid, repetitive behaviours or a complete cognitive shutdown. In such cases, frustration and distress may further impair her ability to respond appropriately, increasing the likelihood of maladaptive reactions.

A critical concern is the potential for panic-induced responses, such as bolting to the surface, a dangerous reaction that could lead to pulmonary barotrauma. Given these cognitive and emotional vulnerabilities, it is concluded that this young diver is not fit to dive. However, as executive functions continue to mature into early adulthood, her fitness to dive may warrant reassessment in the future. If her interest in diving persists, a reevaluation in a few years could be considered, taking into account potential improvements in cognitive flexibility, emotional regulation, and decision-making skills.

General considerations on ASD and diving (expert opinion)

ASD is not an absolute contraindication for diving, but individual risk factors must be carefully evaluated when assessing fitness to dive. A sufficient level of insight and awareness of one's condition is recommended for safe diving. Executive functioning, especially cognitive flexibility, is crucial for safe diving. Executive dysfunction is a common cognitive impairment in ASD. If concerns exist about executive function abilities, a neuropsychological assessment should be included as part of a comprehensive evaluation that considers cognitive, sensory, and physical abilities, as well as self-awareness and confidence in diving skills.[36]

Comorbidities are common in ASD; neurological conditions such as epilepsy constitute an absolute contraindication to diving, whereas Gilles de la Tourette syndrome is considered a relative contraindication. Additionally, comorbid anxiety disorders, mood disorders, and ADHD may present challenges and should be carefully evaluated. Ideally, this is done by a dive medicine professional with psychiatric expertise; however, when such professionals are not available, close collaboration between a psychiatrist and a dive medicine physician is encouraged.

When psychotropic medications are used for treatment, diving may be possible under certain conditions, such as the absence of side effects and adherence to a depth restriction. However, the use of multiple psychotropic medications is discouraged due to the increased risk of side effects.[21]

While there is no strict age limit for the diagnosis of ASD itself, in the context of assessing fitness for activities such as scuba diving, an age threshold of 18 years is often recommended due to the ongoing development of executive functions during adolescence.

Because camouflaging can obscure important signs of stress and difficulty, it may complicate the assessment of diving fitness. In high-stakes environments like diving where rapid decision-making, sensory processing, and clear communication are critical, these hidden challenges may go unnoticed by clinicians and instructors. A cautious, individualised approach is therefore recommended. Particular attention should be paid to sensory sensitivities, stress tolerance, and executive functioning, especially in women and others who may engage in camouflaging behaviours.

For individuals with ASD who have successfully obtained a driver's license and can navigate traffic safely, it is reasonable to expect that scuba diving may also be feasible. However, diving presents additional challenges due to the hyperbaric environment, where panic-induced reactions such as uncontrolled ascent pose significant risks. Additionally, coexisting conditions like epilepsy or severe anxiety may further complicate safety. As with driving, a careful, individualised assessment remains essential to determine whether diving is appropriate.

Conclusion

ASD is not an absolute contraindication to diving, but individualised assessment is crucial. Key factors to evaluate include executive functioning, cognitive flexibility, sensory processing, comorbidities, and medication use. Neuropsychological assessments may assist in determining cognitive suitability, particularly in cases where executive function deficits raise safety concerns. Further research is needed to establish evidence-based guidelines for diving with ASD and to explore potential adaptations or training strategies to enhance safety and accessibility.

References

1 Reus I, van de Sande E, Rienks R, Wingelaar T. Retrospective analysis of challenging cases for medical examiners of diving. Diving Hyperb Med. 2024;54:184–7. doi: 10.28920/dhm54.3.184-187. PMID: 39288922. PMCID: PMC11659069.

2 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.

3 The diagnostic and statistical manual of mental disorders, DSM–5. 5th ed. American Psychiatric Association; 2013.

4 Lehto JE, Juujärvi P, Kooistra L, Pulkkinen L. Dimensions of executive functioning: evidence from children. Br J Dev Psychol. 2003;21:59–80. doi: 10.1348/026151003321164627.

5 Miyake A, Friedman NP, Emerson MJ, Witzki AH, Howerter A, Wager TD. The unity and diversity of executive functions and their contributions to complex "Frontal Lobe" tasks: a latent variable analysis. Cogn Psychol. 2000;41:49–100. doi: 10.1006/cogp.1999.0734. PMID: 10945922.

6 Diamond A. Executive functions. Annu Rev Psychol. 2013;64:135–68. doi: 10.1146/annurev-psych-113011-143750. PMID: 23020641. PMCID: PMC4084861.

7 Robinson S, Goddard L, Dritschel B, Wisley M, Howlin P. Executive functions in children with autism spectrum disorders. Brain Cogn. 2009;71:362–8. doi: 10.1016/j.bandc.2009.06.007. PMID: 19628325.

8 Bednarz HM, Kana RK, Svancara AM, Sherrod GM, Stavrinos D. Neuropsychological predictors of driving hazard detection in autism spectrum disorder and ADHD. Child Neuropsychol. 2021;27:857–87. doi: 10.1080/09297049.2021.1908531. PMID: 33881380.

9 Bougeard C, Picarel-Blanchot F, Schmid R, Campbell R, Buitelaar J. Prevalence of autism spectrum disorder and co-morbidities in children and adolescents: a systematic literature review. Front Psychiatry. 2021;12:744709. doi: 10.3389/fpsyt.2021.744709. PMID: 34777048. PMCID: PMC8579007.

10 Khachadourian V, Mahjani B, Sandin S, Kolevzon A, Buxbaum JD, Reichenberg A, et al. Comorbidities in autism spectrum disorder and their etiologies. Transl Psychiatry. 2023;13(1):71. doi: 10.1038/s41398-023-02374-w. PMID: 36841830. PMCID: PMC9958310.

11 Lundström S, Reichenberg A, Melke J, Råstam M, Kerekes N, Lichtenstein P, et al. Autism spectrum disorders and coexisting disorders in a nationwide Swedish twin study. J Child Psychol Psychiatry. 2015;56:702–10. doi: 10.1111/jcpp.12329. PMID: 25279993.

12 South Pacific Underwater Medicine Society. [cited 2025 Feb 1]. Available from: https://www.uhms.org/resources/featured-resources/recreational-diving-medical-screening-system.html?view=article&id=1048&catid=2.

13 Divers Alert Network (DAN). Autism and diving. [cited 2025 Feb 1]. Available from: https://dan.org/alert-diver/article/autism-and-diving.

14 U.S. Food and Drug Administration (FDA). [cited 2025 Feb 1]. Available from: https://www.fda.gov/media/147966/download.

15 Levy SE, Mandell DS, Schultz, RT. Autism. Lancet. 2009;374:1627–38. doi: 10.1016/S0140-6736(09)61376-3. PMID: 19819542. PMCID: PMC2863325.

16 Madden JM, Lakoma MD, Lynch FL, Rusinak D, Owen-Smith AA, Coleman KJ, et al. Psychotropic medication use among insured children with Autism Spectrum Disorder. J Autism Dev Disord. 2017;47:144–54. doi: 10.1007/s10803-016-2946-7. PMID: 27817163. PMCID: PMC5328123.

17 Kan CC, Geurts HM, van den Bosch K, Forceville EJM, van Manen J, Schuurman CH, et al. Multidisciplinaire richtlijn diagnostiek en behandeling van autismespectrumstoornissen bij volwassenen. De Tijdstroom, Utrecht. [cited 2025 Feb 1]. Available from: https://richtlijnendatabase.nl/richtlijn/autismespectrumstoornissen_bij_volwassenen/autismespectrumstoornissen_bij_volwassenen_-start.html. Dutch.

18 Ćurković M, Dodig-Ćurković K, Erić AP, Kralik K, Pivac N. Psychotropic medications in older adults: a review. Psychiatr Danub. 2016;28:13–24. PMID: 26938816.

19 Manning EP. Central nervous system oxygen toxicity and

hyperbaric oxygen seizures. Aerosp Med Hum Perform. 2016;87:477–86. doi: 10.3357/AMHP.4463.2016. PMID: 27099087. PMCID: PMC7092644.

20 Wingelaar TT, van Ooij PAM, van Hulst RA. Oxygen toxicity and special operations forces diving: hidden and dangerous. Front Psychol. 2017;8:1263. doi: 10.3389/fpsyg.2017.01263. PMID: 28790955. PMCID: PMC5524741.

21 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.

22 Naumann K, Kernot J, Parfitt G, Gower B, Davison K. Water-based interventions for people with neurological disability, autism, and intellectual disability: a scoping review. Adapt Phys Activ Q. 2021;38:474–93. doi: 10.1123/apaq.2020-0036. PMID: 33873153.

23 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.

24 Rossignol DA, Rossignol LW. Hyperbaric oxygen therapy may improve symptoms in autistic children. Med Hypotheses. 2006;67:216–28. doi: 10.1016/j.mehy.2006.02.009. PMID: 16554123.

25 Rossignol DA. Hyperbaric oxygen therapy might improve certain pathophysiological findings in autism. Med Hypotheses. 2007;68:1208–27. doi: 10.1016/j.mehy.2006.09.064. PMID: 17141962.

26 Bennett M, Mitchell S, Neuman T, Piantadosi CA, Feldmeier J, Hampson NB, et al. Hyperbaric oxygen therapy and neurological disease. Undersea Hyperb Med. 2010;37:371–3. PMID: 20929186.

27 Rossignol DA, Bradstreet JJ, Van Dyke K, Schneider C, Freedenfeld SH, O'Hara N, et al. Hyperbaric oxygen treatment in autism spectrum disorders. Med Gas Res. 2012;2(1):16. doi: 10.1186/2045-9912-2-16. PMID: 22703610. PMCID: PMC3472266.

28 Ghanizadeh A. Hyperbaric oxygen therapy for treatment of children with autism: a systematic review of randomized trials. Med Gas Res. 2012;2:13. doi: 10.1186/2045-9912-2-13. PMID: 22577817. PMCID: PMC3369206.

29 Martin R, Srivastava T, Lee J, Raj N, Koth KA, Whelan HT. Using hyperbaric oxygen for autism treatment: A review and discussion of literature. Undersea Hyperb Med. 2015;42:353–9. PMID: 26403019.

30 Xiong T, Chen H, Luo R, Mu D. Hyperbaric oxygen therapy for people with autism spectrum disorder (ASD). Cochrane Database Syst Rev. 2016;10(10):CD010922. doi: 10.1002/14651858.CD010922.pub2. PMID: 27737490. PMCID: PMC6464144.

31 Sakulchit T, Ladish C, Goldman RD. Hyperbaric oxygen therapy for children with autism spectrum disorder. Can Fam Physician. 2017;63:446–8. PMID: 28615394. PMCID: PMC5471082.

32 Podgórska-Bednarz J, Perenc L. Hyperbaric oxygen therapy for children and youth with autism spectrum disorder: a review. Brain Sci. 2021;11(7):916. doi: 10.3390/brainsci11070916. PMID: 34356150. PMCID: PMC8303909.

33 Bennett M, Hart B. The treatment of Autism Spectrum Disorder with hyperbaric oxygen therapy. UHMS Position Paper (12/5/2009). [cited 2025 Feb 10]. Available from: https://www.uhms.org/images/Position-Statements/autism_position_paper.pdf.

34 Hull L, Petrides KV, Allison C, Smith P, Baron-Cohen S, Lai MC, Mandy W. "*Putting on my best normal*": social camouflaging in adults with autism spectrum conditions. J Autism Dev Disord. 2017;47:2519–34. doi: 10.1007/s10803-017-3166-5. PMID: 28527095. PMCID: PMC5509825.

35 Rynkiewicz A, Janas-Kozik M, Słopień A. Girls and women with autism. Psychiatr Pol. 2019 Aug 31;53:737–52. doi: 10.12740/PP/OnlineFirst/95098. PMID: 31760407.

36 Patrick KE, Schultheis MT, Agate FT, McCurdy MD, Daly BP, Tarazi RA, et al. Executive function "drives" differences in simulated driving performance between young adults with and without autism spectrum disorder. Child Neuropsychol. 2020;26:649–65. doi: 10.1080/09297049.2020.1713311. PMID: 31937180.

37 Chee DY, Lee HC, Falkmer M, Barnett T, Falkmer O, Siljehav J, et al. Viewpoints on driving of individuals with and without autism spectrum disorder. Dev Neurorehabil. 2015;18:26–36. doi: 10.3109/17518423.2014.964377. PMID: 25280078.

38 Almberg M, Selander H, Falkmer M, Vaz S, Ciccarelli M, Falkmer T. Experiences of facilitators or barriers in driving education from learner and novice drivers with ADHD or ASD and their driving instructors. Dev Neurorehabil. 2017;20:59–67. doi: 10.3109/17518423.2015.1058299. PMID: 26222490.

39 Daly BP, Nicholls EG, Patrick KE, Brinckman DD, Schultheis MT. Driving behaviors in adults with autism spectrum disorders. J Autism Dev Disord. 2014;44:3119–28. doi: 10.1007/s10803-014-2166-y. PMID: 24925544.

40 Classen S, Monahan M, Wang Y. Driving characteristics of teens with attention deficit hyperactivity and autism spectrum disorder. Am J Occup Ther. 2013;67:664–73. doi: 10.5014/ajot.2013.008821. PMID: 24195900.

41 Lindsay S. Systematic review of factors affecting driving and motor vehicle transportation among people with autism spectrum disorder. Disabil Rehabil. 2017;39:837–46. doi: 10.3109/09638288.2016.1161849. PMID: 27045872.

42 Wilson NJ, Lee HC, Vaz S, Vindin P, Cordier R. Scoping review of the driving behaviour of and driver training programs for people on the autism spectrum. Behav Neurol. 2018;2018:6842306. doi: 10.1155/2018/6842306. PMID: 30245750.

43 Myers RK, Carey ME, Bonsu JM, Yerys BE, Mollen CJ, Curry AE. Behind the wheel: specialized driving instructors' experiences and strategies for teaching autistic adolescents to drive. Am J Occup Ther. 2021;75(3):7503180110p1-7503180110p11. doi: 10.5014/ajot.2021.043406. PMID: 34781345.

44 Sheppard E, van Loon E, Underwood G, Ropar D. Attentional differences in a driving hazard perception task in adults with autism spectrum disorders. J Autism Dev Disord. 2017;47:405–14. doi: 10.1007/s10803-016-2965-4. PMID: 27866348.

45 Sheppard E, van Loon E, Ropar D. Dimensions of self-reported driving difficulty in autistic and non-autistic adults and their relationship with autistic traits. J Autism Dev Disord. 2023;53:285–95. doi: 10.1007/s10803-021-05420-y. PMID: 35022945. PMCID: PMC9889452.

46 Lovering N. 2023 – Are autistic people better at logical thinking? [cited 2025 Feb 4]. Available from: https://psychcentral.com/autism/why-people-with-autism-are-more-logical.

47 Jameel L, Vyas K, Bellesi G, Cassell D, Channon S. Great

expectations: the role of rules in guiding pro- social behaviour in groups with high versus low autistic traits. J Autism Dev Disord. 2015;45:2311–22. doi: 10.1007/s10803-015-2393-x. PMID: 25697738. PMCID: PMC4513197.

48 Chee DY, Lee HC, Patomella AH, Falkmer T. Driving behaviour profile of drivers with autism spectrum disorder (ASD). J Autism Dev Disord. 2017;47:2658–70. doi: 10.1007/s10803-017-3178-1. PMID: 28585117.

49 Huang P, Kao T, Curry AE, Durbin DR. Factors associated with driving in teens with autism spectrum disorders. J Dev Behav Pediatr. 2012;33:70–4. doi: 10.1097/DBP.0b013e31823a43b7. PMID: 22157351.

50 Skyving M, Forsman Å, Dukic Willstrand T, Laflamme L, Möller J. Medical impairment and road traffic crashes among older drivers in Sweden – a national, population-based, case-control study. Accid Anal Prev. 2021;163:106434. doi: 10.1016/j.aap.2021.106434. PMID: 34700248.

51 Mercier A, Sherrod G, Ennis R, Clay OJ, Richter CG, Stavrinos D. The driving profile of Autistic drivers and their driving experiences: a systematic review. J Autism Dev Disord. 2025;55:2141–56. doi: 10.1007/s10803-024-06586-x. PMID: 39395127.

52 Sheppard E, Ropar D, Underwood G, van Loon E. Brief report: driving hazard perception in autism. J Autism Dev Disord. 2010;40:504–8. doi: 10.1007/s10803-009-0890-5. PMID: 19890705.

53 Cox SM, Cox DJ, Kofler MJ, Moncrief MA, Johnson RJ, Lambert AE, et al. Driving simulator performance in novice drivers with autism spectrum disorder: the role of executive functions and basic motor skills. J Autism Dev Disord. 2016;46:1379–91. doi: 10.1007/s10803-015-26771. PMID: 26676628.

54 Reimer B, Fried R, Mehler B, Joshi G, Bolfek A, Godfrey KM, et al. Brief report: examining driving behavior in young adults with high functioning autism spectrum disorders: a pilot study using a driving simulation paradigm. J Autism Dev Disord. 2013;43:2211–7. doi: 10.1007/s10803-013-1764-4. PMID: 23338532.

55 Chee DYT, Lee HCY, Patomella AH, Falkmer T. Investigating the driving performance of drivers with and without autism spectrum disorders under complex driving conditions. Disabil Rehabil. 2019;41:1–8. doi: 10.1080/09638288.2017.1370498. PMID: 28845700.

56 Cox DJ, Owens JM, Barnes L, Moncrief M, Boukhechba M, Buckman S, et al. A pilot study comparing newly licensed drivers with and without autism and experienced drivers in simulated and on-road driving. J Autism Dev Disord. 2020;50:1258–68. doi: 10.1007/s10803-019-04341-1. PMID: 31901120.

Conflicts of interest and funding: nil

Submitted: 10 March 2025
Accepted after revision: 22 June 2025

Guideline

Revised guideline for central nervous system oxygen toxicity exposure limits when using an inspired PO_2 of 1.3 atmospheres

Joseph Hoyt[1], F Gregory Murphy[2], Neal W Pollock[3,4], Dawn Kernagis[5], Nicholas Bird[6], Michael Menduno[7], John Bright[8], Simon J Mitchell[9,10,11]

[1] *NOAA Diving Program, National Oceanographic and Atmospheric Administration, Seattle, WA, USA*
[2] *Navy Experimental Diving Unit, Panama City, FL, USA*
[3] *Department of Kinesiology, Faculty of Medicine, Université Laval, Québec, Canada*
[4] *Service de médecine hyperbare, Centre de médecine de plongée du Québec, Levis, Québec, Canada*
[5] *DEEP Research Inc., Miami, FL, USA*
[6] *Department of Hyperbaric Medicine, Virginia Mason Medical Center, Seattle, WA, USA*
[7] *InDEPTH Magazine, High Springs, FL, USA*
[8] *CPC Inc., San Diego, CA, USA*
[9] *Department of Anaesthesiology, University of Auckland, Auckland, New Zealand*
[10] *Department of Anaesthesia, Auckland City Hospital, Auckland, New Zealand*
[11] *Slark Hyperbaric Unit, North Shore Hospital, Auckland, New Zealand*

Corresponding author: *Professor Simon J Mitchell, Department of Anaesthesiology, University of Auckland, Private Bag 92019, Auckland, New Zealand*
sj.mitchell@auckland.ac.nz

Keywords
Oxygen clock; Rebreathers; Risk; Seizures; Technical diving

Abstract

(Hoyt J, Murphy G, Pollock NW, Kernagis D, Bird N, Menduno M, Bright J, Mitchell SJ. Revised guideline for central nervous system oxygen toxicity exposure limits when using an inspired PO_2 of 1.3 atmospheres. Diving and Hyperbaric Medicine. 2025 30 September;55(3):262–270. doi: 10.28920/dhm55.3.262-270. PMID: 40986922.)

Technical and scientific divers breathing gases delivering hyperbaric pressures of inspired oxygen may be at risk of developing cerebral oxygen toxicity which can manifest as a seizure with little or no warning. The principle preventative strategy is adherence to time limits based on inspired PO_2 levels promulgated in 1991. These limits had their origins in US Navy studies of exposures to higher inspired PO_2s than are typically utilised by modern divers. Indeed, the duration limits for inspired PO_2s in the range typically utilised by technical divers (≤ 1.3–1.4 atm) have relatively little experimental provenance. Contemporary technical dives often involve decompression durations that result in breaches of these limits, and anecdotally, this common occurrence seems associated with a low risk of cerebral oxygen toxicity. A committee of experts recently sought experimental evidence that might support an adjustment to the recommended duration limits for typical technical dives. Such evidence exists only for an inspired PO_2 of 1.3 atm, which is a common default in use of constant PO_2 rebreather devices. The (1991) limit for a single exposure to an inspire PO_2 of 1.3 atm is 180 min with a 24-hour maximum of 210 min. Recent studies provide reassurance that dives with an inspired PO_2 of 1.3 atm consisting of up to 240 min of working dive activity followed by up to 240 min of resting decompression are associated with an acceptably low risk of cerebral oxygen toxicity. This recommendation was promulgated and endorsed at a recent workshop convened by the National Oceanographic and Atmospheric Administration (NOAA) involving technical and scientific divers.

Introduction

Breathing oxygen at inspired partial pressures higher than incurred when breathing air (FiO_2 20.9%) at one atmosphere absolute (atm abs)* (101.3 kPa) may result in toxic effects. Three organ systems are recognised as potentially affected; the brain (central nervous system [CNS] oxygen toxicity); the lungs (pulmonary oxygen toxicity), and the eyes (hyperoxic myopia).[1] CNS toxicity may manifest as a tonic-clonic seizure with little or no warning and, if occurring underwater, this creates a significant risk of drowning. Pulmonary oxygen toxicity causes a progressive

***Footnote:** Although DHMJ policy is to use SI units for pressure, the use of atmospheres and atmospheres absolute is so inculcated into the language of oxygen utilisation in diving, that these latter units will be primarily used for the purposes of this paper.

inflammatory pneumonitis with early symptoms like dry cough, chest discomfort, and reduced lung volumes on spirometry. As its name implies, hyperoxic myopia causes a myopic shift in ocular refraction. The exact mechanisms of all three forms are incompletely characterised, but a central theme is increased oxidative stress and free radical production. Provided no secondary event supervenes (such as drowning after a CNS toxic seizure) the CNS and pulmonary forms seem fully reversible, with the possibility of some residual effects related to hyperoxic myopia.

All three forms of oxygen toxicity are dose-dependent, where dose is a function of inspired PO_2 and time, but the circumstances under which each form may occur differ somewhat. CNS oxygen toxicity only occurs with inspired PO_2s in the hyperbaric range, that is, at inspired PO_2s greater than 1.0 atm. It can occur in a single exposure, and risk may accumulate over multiple closely spaced exposures. Pulmonary oxygen toxicity can occur with very long sub-hyperbaric exposures, but develops more quickly at hyperbaric pressures. It can occur in a single substantial exposure, and risk may accumulate over multiple closely spaced exposures. Hyperoxic myopia only occurs with hyperbaric exposures. It is not known to occur with single exposures but rather develops gradually over multiple exposures. It is most commonly seen in patients having prolonged courses of daily hyperbaric oxygen treatment, but also occasionally in technical divers participating in long expeditions with multiple dives breathing oxygen at hyperbaric pressures.

Although pulmonary toxicity symptoms may be seen in long dives or dive series, and although troublesome symptoms need to be managed, there are no reports of this becoming incapacitating in the water or producing long term effects in divers. Divers have reported myopic change at the end of prolonged expeditions, with the effects being fully reversible in the vast majority of cases.

The management of oxygen exposure during diving has a primary focus on controlling the risk of CNS oxygen toxicity with its predilection for causing a dangerous oxygen toxicity seizure. This was the focus of the present workshop, and further perspectives on relevant diving activities and CNS toxicity itself are provided below.

Technical and scientific diving

The term 'technical diving' is applied broadly to a suite of techniques primarily aimed at allowing a diver to dive deeper or stay longer or both. These techniques are often employed by recreational, scientific, and exploration-oriented divers. Detailed review of 'technical diving' methods can be found elsewhere.[2]

Using gas mixes containing helium, diving beyond 50 m is now common, and even depths beyond 100 m are not uncommon. Extreme exponents have visited shipwrecks deeper than 200 m and caves deeper than 300 m. The decompressions prescribed for these increasingly deeper dives are frequently longer than four hours and may exceed 12 hours. A ubiquitous strategy to minimise the decompression obligation is to breathe as much oxygen as is considered safe because high fractions of inspired oxygen reduce inert gas absorption during time at depth and accelerate inert gas washout during decompression. This creates a tension between maximising the inspired PO_2 to reduce decompression duration and risk of decompression sickness (DCS) versus managing the risk of oxygen toxicity. Increasingly long decompressions often result in violation of long-standing guidelines for maximum safe oxygen dose prescribed to avoid CNS oxygen toxicity.

It is increasingly common for deep dives of long duration to be performed using underwater breathing apparatus referred to as 'rebreathers'. These recycle exhaled gas (thereby minimising gas consumption) through a soda-lime canister to remove exhaled carbon dioxide (CO_2) and replace metabolised oxygen to maintain a relatively constant inspired PO_2 chosen by the diver (often referred to as the PO_2 'setpoint'). There are varying beliefs and practices around the choice of setpoints. Broadly speaking, some divers choose to leave it constant throughout a dive whereas others strategically vary it based on perceived risk, for example, lowering it during the deep working phase of the dive when the risk of oxygen toxicity poses a greater life threat and increasing it during the resting decompression phase of the dive when the greatest decompression advantage can be gained.[3] A common (but not universal) default PO_2 setpoint chosen by rebreather manufacturers is 1.3 atm.

Central nervous system oxygen toxicity

As mentioned above, CNS oxygen toxicity can result in a seizure. The mechanism is uncertain, though in general terms it seems likely that through intermediary pathways, oxidative stress either suppresses GABAergic transmission or enhances excitatory (e.g., glutamatergic) transmission, or both. Although premonitory symptoms such as visual changes, nausea, tinnitus, confusion, and muscle twitching (especially the diaphragm and facial muscles) may precede a seizure, this is not always the case. A seizure may occur without warning.

A curious feature of CNS oxygen toxicity is that for a given inspired PO_2 the risk seems higher in immersed divers than for dry occupants in a hyperbaric chamber. This risk difference may be explained by the tendency of exercising divers exposed to an elevated work of breathing to experience a perturbation of normal respiratory control which reduces CO_2 responsiveness and causes CO_2 retention.[4] This is more likely if the respired gas density is inappropriately high ($> 6\ g{\cdot}L^{-1}$) which contributes to an increased work of breathing.[5] Failure of a rebreather's CO_2 scrubber and CO_2 rebreathing is another mechanism for CO_2 retention. Carbon dioxide retention promotes cerebral

vasodilation and increased blood flow which increases brain tissue oxygen tension in the absence of any change in the inspired PO_2.[6] It follows that hard work and elevated work of breathing are considered significant risk factors for CNS oxygen toxicity during diving. Other precipitating events can include diver error (such as mistakenly breathing a hyperoxic gas at an inappropriate depth) or equipment failures (such as an oxygen addition valve in a rebreather failing in an open position). Although some of these risk factors are understood, there is also a significant component of unexplained inter- and intra-individual variability in risk.[7]

RISK REDUCTION AND MITIGATION

The primary means of risk reduction for CNS oxygen toxicity has been adherence to published inspired PO_2-time exposure limits. The most widely promulgated compilation of such limits is published in the 1991 National Oceanographic and Atmospheric Administration (NOAA) diving manual[8] (Table 1) which are still the standard taught to modern divers. Concomitantly, NOAA also created the 'CNS clock', to track cumulative oxygen exposure for a dive or a day of diving. In particular, this approach allowed the diver to account for varying inspired PO_2s over the course of a dive by summing 'CNS percent' contributions. Each contribution is calculated as:

$$\text{CNS percent} = (\text{time at } PO_2/\text{time limit for that } PO_2) \times 100$$

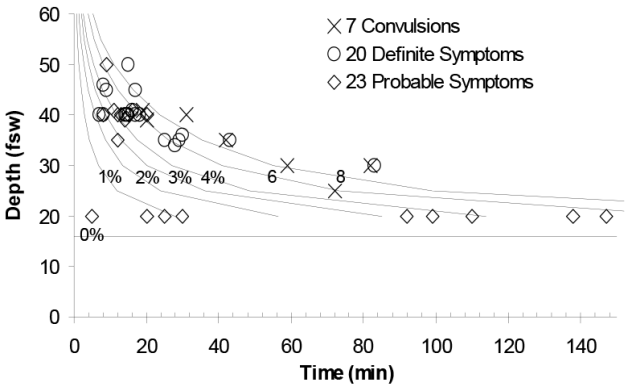
A CNS percent of 100 means that the diver has reached their maximum exposure limit for a dive. For example, based on the limits in Table 1, a CNS percent of 100 could be reached by a 180-min exposure to an inspired PO_2 of 1.3 atm or by an exposure of 90-min at 1.3 atm followed by 60-min at 1.5 atm (each of the latter two exposures being 50% of their respective limits, summing to 100%). A daily exposure percentage is calculated in the same way using the 24-hour day limits in Table 1. Most desktop decompression software programs used for generating decompression plans include CNS clock calculations for the dive.

There are a number of important points about the nature and provenance of the NOAA limits promulgated in Table 1. First, the table includes limits for lower inspired PO_2s that will not result in CNS oxygen toxicity. This reflects a goal for these limits to cover the risk of both pulmonary and CNS toxicity. Unfortunately, as the inspired PO_2 decreases, the transition from limits with primary relevance to CNS toxicity to those with a primary focus on pulmonary toxicity is undefined. Second, the experimental provenance of these limits is undescribed. However, if one searches the relevant literature for related information it becomes apparent (as described by Vann and Hamilton)[9] that "*these limits were based on best judgement from extensive experience, not on the statistical analysis of quantitative data*". This characterisation refers primarily to the limits around inspired PO_2s of most relevance to modern divers (less than 1.6 atm). There was, in fact, a moderate amount of testing at inspired PO_2s of 1.6 atm and above under the auspices of the US Navy.[10] For example, Vann[11] reported a large dataset of 773 working oxygen dives between depths of 20–50 ft (6–15 m, inspired PO_2 1.6–2.5 atm), and summarised outcomes for a

Table1
Table of oxygen exposure limits as published in the NOAA 1991 diving manual

Inspired PO_2 (atm)	Max duration (min) Single exposure	Max duration (min) 24 h day	Exceptional exposure (min)
2.0			30
1.9			45
1.8			60
1.7			75
1.6	45	150	120
1.5	120	180	150
1.4	150	180	180
1.3	180	210	240
1.2	210	240	
1.1	240	270	
1.0	300	300	
0.9	360	360	
0.8	450	450	
0.7	570	570	
0.6	720	720	

Figure 1
Statistical model of CNS oxygen toxicity risk (convulsions, definite and probable symptoms) reported by Vann and Hamilton[9] and based on US Navy testing data; fsw – feet of seawater



subset of that data including risk isopleths for a composite outcome of convulsions, definite or probable symptoms in the 2008 technical diving conference proceedings as shown in Figure 1.[9]

The 0% risk isopleth in Figure 1 is depicted as 16 ft or 1.5 atm oxygen. In fact, Vann and Hamiton reported that, informed by a model developed by the US Navy,[12] the 0% risk isopleth was, for practical purposes, considered to lie at 1.3 atm (132 kPa), and that this was the basis for the choice of 1.3 atm as the setpoint for US Navy mixed gas rebreather diving. Accordingly, one could then ask why the Navy (and subsequently NOAA) imposed a limit for 1.3 atm at all? In answering this question, Vann and Hamilton[9] cite a personal communication from Dr Ed Flynn stating that (despite the perception of little or no CNS risk at an inspired PO_2 of 1.3 atm) "*the Navy also imposed an arbitrary time limit of 240 min at 1.3 atm because of potential onset of pulmonary oxygen toxicity*". Despite this, in the same publication, Vann and Hamilton also caution that '0% risk' for CNS toxicity is an uncertain parameter, and describe several anecdotes that may represent seizure events among technical divers breathing an inspired PO_2 of 1.3 atm.[9]

There are other preventative strategies unrelated to inspired PO_2 and duration *per se*. Intermittency, referring to periodic switching to a lower inspired PO_2, typically an inspired PO_2 equivalent to breathing air at the same depth (and often referred to as 'air breaks'), is accepted to be effective in reducing the risk of CNS oxygen toxicity. However, there is no widely accepted or proven regimen in diving applications. A single five-minute air break in the middle of a 75-minute dry hyperbaric oxygen treatment at 2.5 atm was reported to more than halve the risk of oxygen seizures in human patients.[13] Another preventative approach embraces strategies to reduce the risk of CO_2 retention. These include: minimising work at depth, for example, through use of diver propulsion vehicles; maintaining respired gas density below 6.2 $g \cdot L^{-1}$;[5] and using underwater breathing apparatus with a low breathing resistance.[14] Strategies to mitigate the drowning risk should a seizure occur are also often employed by technical divers. There is some evidence to support the efficacy of mouthpiece retaining devices[15,16] and full-face masks[17] in protecting the airway in unconscious divers. Combined with use of a buddy system, these devices may make an underwater seizure more survivable. Conducting long decompressions in dry habitats[18] potentially lowers the risk of a seizure, and also improves survivability should one occur.

The modern context and workshop goals

To summarise the present situation, technical divers and other groups such as scientific divers conducting prolonged dives are working with restrictive oxygen exposure limits that are essentially untested for the outcome of greatest concern (CNS toxicity) in the range of inspired PO_2s commonly used (< 1.6 atm). As technical diving has progressed to deeper longer dives, divers are inevitably forced to ignore these limits.[19] Comments from rebreather divers who informally discussed the matter with author MM in related on-line communications reflect this situation (Table 2). As implied in comments in Table 2, there is abundant anecdote suggesting that when operating at the inspired PO_2s most commonly used by technical divers (~1.3–1.4 atm), CNS oxygen toxicity events are vanishingly rare, even when the current limits (Table 1) are substantially exceeded. Given this unsatisfactory situation of ignoring (or being unreasonably constrained by) limits that have little face validity and lack an evidential base, the context area experts among the author group for this report convened a workshop to evaluate whether it was possible to revise the current limits on an evidential basis. Importantly, this group contained strong representation from NOAA whose limits were under consideration.

The goals for the project were to evaluate relevant evidence that might allow an objective evidence-based revision of the present PO_2-time limits, and to run a workshop whose participants could have input into refining a set of statements/guidelines arising from the author committee's preliminary deliberations. This workshop took place on 29 March 2025.

Evidence supporting change

It is germane that author FGM has knowledge of and access to US Navy testing data that is relevant to this subject area. In preliminary discussions within the author group, it became clear that there are essentially no data that extend our ability to prescribe evidence-based duration limits for inspired PO_2 to avoid CNS toxicity other than at an inspired PO_2 of 1.3 atm. Since an agreed goal among authors was not to replace guesswork with more guesswork, consideration for revision was therefore limited to an inspired PO_2 of 1.3 atm. Fortunately, as previously mentioned, this corresponds with

Table 2

Representative commentary from current technical divers illustrating attitudes to current inspired PO_2 time limits; the last two boxed comments reflect the dilemma facing advanced technical diving instructors who find themselves teaching limits that they sometimes ignore, and may even exceed during training course dives

"To be honest we haven't worried about CNS toxicity for years. We do a 125 m dive on a 1.2 atm setpoint and end the dive with over 200% CNS. I've done this many, many times with little to no symptoms. The worst I ever felt was feeling a bit nauseous. We do conduct air breaks every 30 minutes."
"The [CNS limits] are something that run the risk of being ignored by deep tech divers, purely because it is unavoidable, regularly exceeded, and has little consequence or known issues. I am a big fan of using conservative limits, but they need to be based on some logic, studies, data or more modern evidence instead of what we currently have now; blindly sticking to old recommendations with no reason, is not a good strategy."
"We basically disregard the CNS clock. Pulmonary toxicity is really our only concern, and while uncomfortable it is not really a great danger, just something to be managed."
"I regularly exceed my CNS clock on our advanced 3–4-hour long CCR course dives, which can range from 130% plus on class dives. I've had my clock up to 680% on exploration dives, which have been up to 14 hours long. While I definitely teach the CNS clock, I am also very open about sharing our accumulated experience, which seems to prove that these numbers are overly conservative."
(In respect of teaching students) *"I present: here's the clock, and here's our experience. We want people to follow reasonable guidelines."*

a common default PO_2 setpoint adopted by many rebreather manufacturers, and is a pragmatic, useful setting for real-world rebreather dives.

The NOAA 1991 limit for an inspired PO_2 of 1.3 atm was 180 minutes for a single exposure and 210 minutes for any 24-hour day (Table 1). A PO_2 of 1.3 atm is currently the maximum setpoint allowed for NOAA rebreather operations, with the same exposure duration limits to those in the 1991 manual.[20] The question posed in this panel workshop was whether the published 1.3 atm duration limits remain appropriate. An abundance of community experience alluded to above and limited but relevant research data indicate that relaxation of the 1.3 limits is likely appropriate.

The US Navy single exposure duration limit for 1.3 atm inspired PO_2 is currently 240 minutes for up to four consecutive days.[21] Research trials conducted at the Navy Experimental Diving Unit (NEDU) in Panama City, FL have also included seizure-free studies out to an eight-hour (480 minute) duration. These data provide significant new information about the risk of CNS oxygen toxicity associated with diving at pressures of oxygen commonly seen in modern rebreather diving. A summary of these data is provided to help assess the relevance to the topic at hand.

Open-circuit gas supplies allow for precise control of the subject's breathing medium and allow for better control of a subject's oxygen exposure in a research setting. One hundred and twenty-six open-circuit exposures have been conducted by NEDU around the 1.3 PO_2 limit. Seventy-two six-hour (360-minute) and 54 eight-hour (480-minute) resting exposures were performed with PO_2s predominately at 1.36 atm but occasionally rising as high as 1.46 atm for brief periods due to changes in diver depth.[22] During these dives a 5-minute air break was permitted either every hour or every two hours of dive time.[23–25]

Exposures using closed-circuit rebreathers provide more comparable resistive effort and gas composition to civilian rebreather diving. The US Navy MK 16 MOD 1 rebreather controls the PO_2 in the breathing loop around a setpoint of 1.3 ± 0.15 atm from the time the diver descends deeper than 10 metres of seawater (msw) (33 feet of seawater [fsw]) until the diver ascends shallower than 3.7 msw (12 fsw).[26] Seventeen exposures lasting for eight hours with the diver at rest were conducted using the MK16 MOD 1 rebreather.[22] An additional 88 exposures were conducted using the MK 16 MOD 1 and MK 25 MOD 2 rebreathers in combination, with a duration of six–seven hours. The MK25 MOD 2 is a mechanical oxygen rebreather. However, due to the purging scheme employed during these dives and the off-gassing into the breathing loop by the divers the oxygen pressure in the breathing loop was typically 1.29 atm PO_2. These dives incorporated 43–60 minutes of exercise followed by decompression. Throughout the decompression 45 minutes of chamber air breathing occurred.[27]

More recently, 116 dives at a PO_2 setpoint of 1.3 atm were conducted using the MK 16 MOD 1 rebreather lasting between 5.4 and 8 hours.[28] These dives included moderate exercise during the first 90–150 minutes of bottom time. The decompression was completed warm and dry, there was a 20-minute air break upon reaching 30 msw (100 fsw) to facilitate transferring the divers out of the Ocean Simulation Facility (OSF) wet pot and into the OSF dry hyperbaric

chambers and then a five minute air break for every 60 minutes of decompression.

No oxygen toxicity seizures were recorded in any of the above dives, and none were terminated due to oxygen toxicity symptoms. While these outcomes are reassuring and provide an objective justification for change, a measured approach is still required since the data are limited, many of the exposures were conducted at rest, and some included five-minute air breaks every hour and portions of decompression in a dry state. All of these features may differ from typical diving practice. The relatively precise control of inspired PO_2 in research trials may represent another possible contrast with real world diving where variability in PO_2 due to differences in rebreather oxygen injection patterns or other influences may be greater. The US Navy limits the allowable setpoint variability to ± 0.15 atm, and it is unclear what variability would be seen with different rebreathers and controller configurations. Similar questions surround differences in physical activity between controlled and typical exposures, although it is generally true that in 'real-world' diving, PO_2 exposures most often approach limits during long decompressions when the divers are at rest, at shallower depths, and are breathing low-density gas. Under these circumstances CO_2 retention and its associated exacerbation of CNS oxygen toxicity risk is unlikely.[29]

It is acknowledged that this reported experience with 1.3 atm PO_2 exposures does not inform the risk assessment for either lower or higher setpoints. It can be expected that any duration limit deemed acceptable for a 1.3 atm PO_2 setpoint would result in a lower CNS toxicity risk at lower inspired PO_2 values, but there are insufficient data to generate limits that take the form of the progressively longer existing NOAA table limits (Table 1). Cases of CNS toxicity have been reported at ≥ 1.4 atm, but the true risk in the 1.4–1.6 atm exposure range remains very poorly characterised and once again, generating evidence-based limits that take the form of the existing NOAA table is not possible. In either direction, longer PO_2 exposures, particularly at the higher points in the range, will increase the likelihood of pulmonary toxicity becoming a controlling factor. While pulmonary oxygen toxicity managed in a timely manner is reversible with no known long-term consequences for divers, it may become a limiting factor that requires additional work to characterise appropriate operational restrictions.

Workshop discussion

The workshop was hosted by NOAA in Seattle in conjunction with the 2025 American Academy of Underwater Sciences Annual Meeting and it was attended by sixty participants. The participants were a mix of recreational technical divers and scientific divers. Many of the attendees were in leadership roles, and many used 'technical diving' methods in the course of their work. The consensus discussion was preceded by presentations by authors SJM (topics approximately confluent with the introduction, technical and scientific diving, and CNS oxygen toxicity sections of this paper), MM (content of Table 2), and FGM (experimental diving evidence supporting change). A series of draft consensus statements was then presented and discussed. A verbatim transcript of the discussion is not reported, but a summary of discussion themes is as follows.

Workshop participants convened for a discussion about the proposed changes to the current NOAA time limit at an inspired PO_2 of 1.3 atm. Although 1.4 atm inspired PO_2 is used in some rebreather systems as a manufacturer's setpoint, the data remain insufficient for formal recommendations related to an inspired PO_2 of 1.4 atm (or above). For this discussion, there was also an expressed need to avoid conflating pulmonary oxygen toxicity with CNS oxygen toxicity, to avoid the same ambiguity currently present in the NOAA limits.

Additional operational perspectives were provided from divers and clinicians participating in the workshop. Clinically, experts noted that hyperbaric oxygen treatments routinely involve higher inspired PO_2 exposure (e.g., over 2.0 atm) but these exposures are dry, resting, and do not involve significantly increased work of breathing or the risk of drowning from a CNS oxygen toxicity seizure. For recreational and scientific diving, an inspired PO_2 setpoint of 1.3 atm provides a balance between safety and decompression efficacy.

Participants asked about the physiological implications of multi-day and high-intensity diving when considering revised limits. While US Navy data pertaining to four consecutive days of diving reaching recommended limits at 1.46 atm inspired PO_2 suggested low CNS oxygen toxicity risk when incorporating mitigations such as rest during decompression and reduced gas density, pulmonary symptoms and hyperoxic myopia were reported following these exposures. Based on these data, the US Navy currently limits multi-day diving to four consecutive days with four hours at 1.3 atm inspired PO_2.

The US Navy data also underscore the need to distinguish between working and resting phases during dives, with resting decompression being more favorable for safely extending exposure durations, and 'resting' being formally defined as a respiratory minute volume (RMV) < 22.5 $L \cdot min^{-1}$. Practically, 'resting' is defined as not performing any activity beyond what a diver needs to do to decompress safely under optimal or near-optimal conditions. To expand operational data, dive profile data-sharing was suggested for capturing dive logs and outcomes. However, concerns were raised about the limitations of such data due to survivor bias and incomplete contextual information.

The consensus statement discussion concluded that traditional CNS limits are derived primarily from high-

PO_2 experimental conditions that are not reflective of PO_2 setpoints used in typical field diving, and available evidence supports the acceptability of extending exposure time for 1.3 atm inspired PO_2. Mitigation strategies, such as periodic breaks with lower PO_2 and reduced gas density, reduced activity during decompression, buddy systems, full-face masks, and decompression tethers, were encouraged for longer exposures. Importantly, this consensus was focused on adjusting time limits at 1.3 atm inspired PO_2, rather than treating 1.3 atm PO_2 as a rigid setpoint cap. For a potential limit extension beyond 240 minutes for a working dive at 1.3 atm inspired PO_2, there was concern about the limited robustness of both dry and wet dive data. However, 240 minutes at 1.3 atm inspired PO_2 in various operational and testing scenarios has resulted in safe CNS profiles with reasonably robust supporting data. Indeed, it was suggested that even a 240-minute exposure limit for 1.3 atm inspired PO_2 remains conservative.

Closing remarks addressed the practical application of these recommendations, especially within occupational diving programs, for increased diver productivity while still prioritising safety. The group favoured formalising a revised standard, allowing for up to four hours of working diving followed by up to four hours of resting decompression at an inspired PO_2 of 1.3 atm as being both conservative and evidence-based. Outreach to the wider diving community was highlighted as a necessary next step to explain the rationale behind the changes and ensure understanding. The vast majority of the workshop participants supported the consensus direction, with further contributions encouraged to refine these evolving standards.

The audience gave the author group permission to edit the draft statements to incorporate revisions or additions arising from the consensus discussion. Those revised statements are presented below. Of note, NOAA plans to update subsequent editions of the NOAA manual consistent with the information contained herein.

Workshop consensus statements for promulgation to the community

1. HISTORIC PERSPECTIVE

The pressure-time exposure limits promulgated by NOAA in 1991 allow 180 minutes (single exposure) and 210 minutes (cumulative exposure in 24 hours) for an inspired PO_2 of 1.3 atm, however, they are based almost exclusively on experimental exposure data for higher inspired PO_2s (≥ 1.6 atm) with an emphasis on open-circuit diving. Sustained moderate exposures (e.g., 1.3 atm) typically experienced in modern closed-circuit diving were not a consideration at the time. Further, although the limits are primarily employed to minimise the risk of central nervous system oxygen (CNS) toxicity, they were set in consideration of both CNS and pulmonary or 'whole-body' risk. While consideration of both these consequences of excessive oxygen exposures is important in dive planning, the severity of consequence is far greater for CNS. Therefore, the risk acceptance decision in setting limits to avoid CNS toxicity should be distinct from pulmonary toxicity.

2. ACCEPTABLE CENTRAL NERVOUS SYSTEM RISK OF 1.3 ATM INSPIRED PO_2

Available data suggest that a four-hour exposure to an inspired PO_2 of 1.3 atm* during the working phase of a dive is associated with a very low risk of CNS oxygen toxicity. Recent additional evidence suggests that a further four-hour exposure to an inspired PO_2 of 1.3 atm* under resting conditions[#] (for example, during decompression with minimal physical demands) is also associated with an acceptably low risk of CNS oxygen toxicity. There is anecdotal evidence describing even longer decompressions performed without incident by technical divers, but at present, extending dive time beyond eight-hours is poorly informed by data.

** Most relevant data come from studies where the inspired PO_2 was precisely controlled. There may be variability in inspired PO_2 around a selected setpoint when using a rebreather, or arising from changes in depth when using open-circuit gas. Divers should be cognisant of this issue, and attempt to minimise upward drift in the inspired PO_2 when applying this guideline.*

[#] Resting involves no greater physical activity than that required to maintain depth position and physical comfort under controlled conditions. When decompressing, use of a decompression stage or surface marker buoy to minimise required effort would aid in reducing physical activity.

3. PULMONARY OXYGEN TOXICITY RISK

Longer exposures to elevated inspired PO_2s will result in an inevitable tendency to greater development of pulmonary oxygen toxicity symptoms which may become a limiting factor in dive duration. Pulmonary symptoms do not carry the same absolute life threat as CNS manifestations, and seem invariably reversible if allowed to fully recover after the dive in which they first appear. It is strongly recommended that if pulmonary oxygen toxicity symptoms develop, no further diving is undertaken until they are fully resolved.

4. DAILY EXPOSURE GUIDANCE

When using this consensus to guide cumulative exposure to an inspired PO_2 of 1.3 atm for more than one dive in a day it is recommended that the cumulative total does not exceed the combination of four hours of working dive and four hours of underwater rest (e.g., during decompression

with minimal physical demands) per 24-hour period (starting from the beginning of the first dive).

5. RISK MITIGATION

There are several strategies divers may consider to mitigate the risk of CNS oxygen toxicity or its consequences should it occur. Risk of occurrence can be reduced by periodic five-minute 'breaks' breathing a lower inspired PO_2 (for example, an inspired PO_2 equivalent to breathing air at the same depth). This would typically be undertaken during decompression. Risk of occurrence can also be reduced by lowering the likelihood of CO_2 retention through strategies such as minimising underwater work where practicable, avoiding gas densities greater than ~6 $g{\cdot}L^{-1}$, appropriate management of CO_2 scrubbers in rebreathers, and use of low work-of-breathing underwater breathing apparatus. The risk of adverse outcomes associated with an underwater oxygen seizure can be reduced by the use of a buddy system and a mouthpiece retaining device or full-face mask, by tethering to decompression stages and use of habitats during decompression.

6. LIMITATIONS ON SCOPE OF GUIDANCE

In the absence of relevant data, no calculated increase in exposure duration guidelines for inspired $PO_2 < 1.3$ atm is proposed. It can be confidently assumed the revised 1.3 atm guidelines promulgated here could be applied at an inspired PO_2 of < 1.3 atm with less risk of CNS oxygen toxicity. No changes to the exposure duration guidelines for $PO_2 > 1.3$ atm are proposed given the lack of high-quality safety data. Although this revised guideline is based on superior evidence to the one it replaces, it should nevertheless be understood that adherence to these limits does not guarantee that CNS oxygen toxicity will not occur.

References

1 Clark JM, Thom SR. Oxygen under pressure. In: Brubakk AO, Neuman TS, editors. Bennett and Elliotts physiology and medicine of diving. 5th ed. Edinburgh: Saunders; p. 358–419.

2 Mitchell SJ, Doolette DJ. Recreational technical diving part 1: an introduction to technical diving methods and activities. Diving Hyperb Med. 2013;43:86–93. PMID: 23813462. [cited 2025 Jun 22]. Available from: https://dhmjournal.com/images/IndividArticles/43June/Mitchell_dhm.43.3.86-93.pdf.

3 Gouin E, Monnot DPM, Michot T, Guerrero F, Blatteau J-É. Diving practices in technical divers' community and behaviour towards self-reported unusual symptoms. Diving Hyperb Med. 2025;55:114–25. doi: 10.28920/dhm55.2.114-125. PMID: 40544139.

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

5 Anthony TG, Mitchell SJ. Respiratory physiology of rebreather diving. In: Pollock NW, Sellers SH, Godfrey JM, editors. Rebreathers and scientific diving. Proceedings of NPS/NOAA/DAN/AAUS June 16-19, 2015 workshop. Wrigley Marine Science Center, Catalina Island, CA; 2016. p. 66–79.

6 Lambertsen CJ, Ewing JH, Krough RH, Gould R, Stroud MW 3rd. Oxygen toxicity; arterial and internal jugular blood gas composition in man during inhalation of air, 100% O_2 and 2% CO_2 in O2 at 3.5 atmospheres ambient pressure. J Appl Physiol. 1955;8:255–63. doi: 10.1152/jappl.1955.8.3.255. PMID: 13271251.

7 Donald KW. Oxygen poisoning in man. Br Med J. 1947;17;1(4506):667–72. PMID: 20248086. PMCID: PMC2053251.

8 NOAA Diving Manual. United States National Oceanic and Atmospheric Administration: Office of Undersea Research. October 1991. [cited 2025 Jun 22]. Available from: https://ia800204.us.archive.org/3/items/noaadivingmanual00unit/noaadivingmanual00unit.pdf.

9 Vann RD, Hamilton RW. Central nervous system oxygen toxicity. In: Vann RD, Mitchell SJ, Denoble PJ, Anthony TG, editors. Technical diving. Proceedings of the Divers Alert Network 2008 January 18-19 conference. Durham NC: Divers Alert Network; 2009. p. 38–66. [cited 2025 Jun 22]. Available from: https://apps.dan.org/publication-library/.

10 Butler FK Jr, Thalmann ED. Central nervous system oxygen toxicity in closed circuit scuba divers II. Undersea Biomed Res. 1986;13:193–223. PMID: 3727183.

11 Vann R. Oxygen toxicity risk assessment. Final report on ONR Contract N00014-87-C-0283. Durham NC: F.G Hall Environmental Research Laboratory; 1988. [cited 2025 Jun 22]. Available from: https://apps.dtic.mil/sti/tr/pdf/ADA299552.pdf.

12 Harabin AL, Survanshi SS, Homer LD. A model for predicting central nervous system oxygen toxicity from hyperbaric oxygen exposures in humans. Toxicol Appl Pharmacol. 1995;132:19–26. doi: 10.1006/taap.1995.1082. PMID: 7747281.

13 Costa DA, Ganilha JS, Barata PC, Guerreiro FG. Seizure frequency in more than 180,000 treatment sessions with hyperbaric oxygen therapy – a single centre 20-year analysis. Diving Hyperb Med. 2019;49:167–74. doi: 10.28920/dhm49.3.167-174. PMID: 31523791. PMCID: PMC6884101.

14 Warkander DE, Norfleet WT, Nagasawa GK, Lundgren CEG. Physiologically and subjectively acceptable breathing resistance in divers' breathing gear. Undersea Biomed Res. 1992;19:427–45. PMID: 1304670.

15 Haynes P. Increasing the probability of surviving loss of consciousness underwater when using a rebreather. Diving Hyperb Med. 2016;46:253–9. PMID: 27966205. [cited 2025 Jun 22]. Available from: https://dhmjournal.com/images/IndividArticles/46Dec/Haynes_dhm.46.4.253-259.pdf.

16 Gempp E, Louge P, Blatteau J-E, Hugon M. Descriptive epidemiology 153 diving injuries with rebreathers among French military divers from 1979–2009. Mil Med. 2011;176;446–50. doi: 10.7205/milmed-d-10-00420. PMID: 21539168.

17 van Waart H, Harris RJ, Gant N, Vrijdag XCE, Challen CJ, Lawthaweesawat C, Mitchell SJ. Deep anaesthesia: The Thailand cave rescue and its implications for management of the unconscious diver underwater. Diving Hyperb Med. 2020;50:121–9. doi: 10.28920/dhm50.2.121-129. PMID: 32557413. PMCID: PMC7481118.

18 Harris RJ, Challen CJ, Mitchell SJ. The first deep rebreather dive using hydrogen: case report. Diving Hyperb Med.

2024;54:69–72. doi: 10.28920/dhm54.1.65-68. PMID: 38507913. PMCID: PMC11065502.
19 Gouin E, Dugrenot E, Gardette B. Extremely deep bounce dives: planning and physiological challenges based on the experiences of a sample of French-speaking technical divers. Diving Hyperb Med. 2025;55:203–10. doi: 10.28920/dhm55.2.203-210. PMID: 40544151.
20 NOAA Diving Standards and Safety Manual. Seattle (WA): National Oceanographic and Atmospheric Administration; 2023. [cited 2025 Jun 22]. Available from: https://omao.noaa.gov/sites/default/files/2023-05/NDSSM%20MAY%202023%20FINAL.pdf.
21 US Navy Diving Manual Revision 7, Change A. Washington: US Department of the Navy; 2016. [cited 2025 Jun 22]. Available from: https://www.navsea.navy.mil/Portals/103/Documents/SUPSALV/Diving/Dive%20Manual%20Rev%207%20Change%20A.pdf.
22 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.
23 Shykoff BE. Pulmonary effects of submerged breathing of air or oxygen. U.S. Navy Experimental Diving Unit Technical Report; 02-04. 2002.
24 Shykoff BE. Pulmonary effects of submerged oxygen breathing: 4-, 6-, and 8-hour dives at 140 kPa. Undersea Hyperb Med. 2005;32:351–61. PMID: 16457084.
25 Shykoff BE, Pulmonary oxygen toxicity with exercise: single MK 25 rebreather dives or split 6-hour exposures. U.S. Navy Experimental Diving Unit Technical Report; 07-14. 2007.
26 Gerth WA, Johnson TM. Development and validation of 1.3 ATA PO2-in-He decompression tables for the MK 16 MOD 1 UBA. U.S. Navy Experimental Diving Unit Technical Report; 02-10. 2002.
27 Shykoff BE. Pulmonary effects of multilevel HeO_2 dives using the MK 16 MOD 1 UBA. U.S. Navy Experimental Diving Unit Technical Report; 11-01. 2011.
28 Doolette DJ, Murphy FG, Gerth WA. Validation of XVAL-He-8_040 and XVAL-He-9_040 Thalmann algorithm parameter sets for computing decompression schedules for extended duration 1.3 atm PO_2 He-O_2 diving with N_2-O_2 decompression. U.S. Navy Experimental Diving Unit Technical Report; 19-05. 2019.
29 Mitchell SJ, Mesley P, Hannam JA. End tidal CO_2 in recreational rebreather divers on surfacing after decompression dives. Aerosp Med Hum Perform. 2015;86:41–5. doi: 10.3357/AMHP.4113.2015. PMID: 25964034.

Conflicts of interest and funding

Professors Simon Mitchell and Neal Pollock are on the editorial board of *Diving and Hyperbaric Medicine*. This guideline is derived from a consensus workshop and deliberations of a panel of experts. It was therefore not subject to further review and there was no requirement for an editorial decision about suitability for publication. No sources of funding were declared.

Submitted: 30 June 2025
Accepted: 7 July 2025

Case reports

Serial chest computed tomography imaging in a freediver with a case of pulmonary barotrauma of descent (lung squeeze) showing the time course of resolution

Madeleine E Wagner[1], Elaine Yu[2], Anna Lussier[1], Nicole Lin[3], Henry Guo[4], Peter Lindholm[1,2]

[1] *University of California San Diego School of Medicine, San Diego, USA*
[2] *Department of Emergency Medicine, University of California at San Diego, San Diego, USA*
[3] *Division of Thoracic Surgery, Department of Cardiothoracic Surgery, Stanford University, School of Medicine, Stanford, USA*
[4] *Department of Radiology, Stanford University, Stanford, USA*

Corresponding author: *Dr Madeleine Wagner, University of California San Diego School of Medicine, San Diego, USA*
ORCiD: *0009-0005-5424-4267*
madeleineewagner@gmail.com

Keywords
Breath-hold diving; Diving medicine; Hemoptysis; Pulmonary oedema; Radiological imaging

Abstract

(Wagner ME, Yu E, Lussier A, Lin N, Guo H, Lindholm P. Serial chest computed tomography imaging in a freediver with a case of pulmonary barotrauma of descent (lung squeeze) showing the time course of resolution. Diving and Hyperbaric Medicine. 2025 30 September;55(3):271–274. doi: 10.28920/dhm55.3.271-274. PMID: 40986923.)
Freedivers can suffer respiratory symptoms indicative of freediving induced pulmonary syndrome (FIPS). Aetiology includes immersion pulmonary oedema and barotrauma of descent in the tracheobronchial or pulmonary parenchyma, also colloquially called 'squeeze'. The pathophysiology and natural history are still largely unknown. This case report describes a freediver who developed haemoptysis following a 49 m personal best constant weight bi-fin dive, presenting with two episodes of haemoptysis within 24 hours post-dive. This style of diving entails finning down to the desired depth, turning with a single pull on the rope, and then finning up to the surface without use of the arms. The diver exhibited no other symptoms and remained haemodynamically stable. Computed tomography (CT) imaging performed two days post-dive showed ground-glass opacities in the right upper and middle lobes. Treatment involved hospitalisation, high-dose corticosteroids, and antibiotics. Follow-up CT scans post-dive revealed almost complete resolution (six days) followed by complete resolution of pulmonary abnormalities (21 days). This case is unique for its documentation of changes in lung findings over three sequential CT scans, providing a timeline of anatomical recovery. Serial CT scanning would not be routinely recommended from a radiation safety perspective but yielded interesting data into the time course of this trauma. The findings raise questions about the underdiagnosis of squeeze injuries, as this diver displayed minimal symptoms despite radiographic evidence of ground-glass opacities. This case highlights the need for standardised imaging and management protocols, as well as further research into the natural history and clinical significance of FIPS.

Introduction

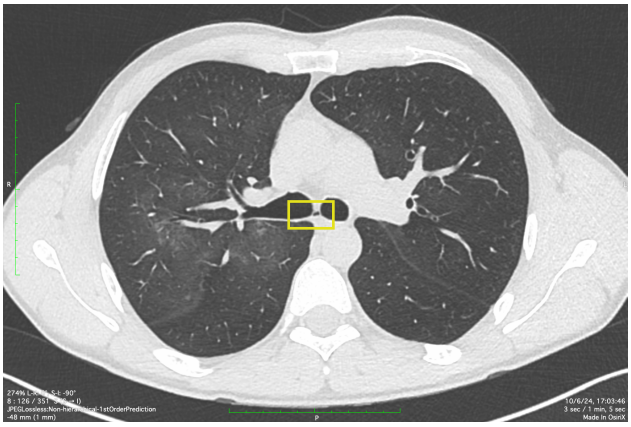
Freediving induced pulmonary syndrome (FIPS) encompasses signs and symptoms freedivers can experience after diving from immersion pulmonary oedema, barotrauma of descent, or airway barotrauma.[1,2] Squeeze is the colloquial term often used to describe FIPS, with a poorly understood time course of resolution. Squeeze is caused by a changing pressure differential between a gas containing space in the body and the surrounding ambient pressure, causing a relative negative pressure inside the lungs. It can manifest as regional lung collapse/atelectasis, fluid shift from the alveolar capillary beds causing pulmonary oedema, or rupture of the bronchial and/or alveolocapillary beds producing alveolar haemorrhage leading to haemoptysis.[3] There are few cases with computed tomography imaging (CT) after a squeeze, and none with serial follow-up imaging to track radiographic resolution of the injury.

Case report

The diver described here provided written consent for publication of his case history and images.

We report a freediver who experienced haemoptysis manifesting as two isolated events of blood clots cleared from the throat at two and 24 hours post-dive respectively. He was hospitalised for further workup and found to have ground-glass opacities in the right upper and middle lobes on a non-contrast CT scan at two days post-dive, along

Figure 1
Initial CT chest on day two post-dive showing the 2 mm bronchial diverticulum as indicated by the box

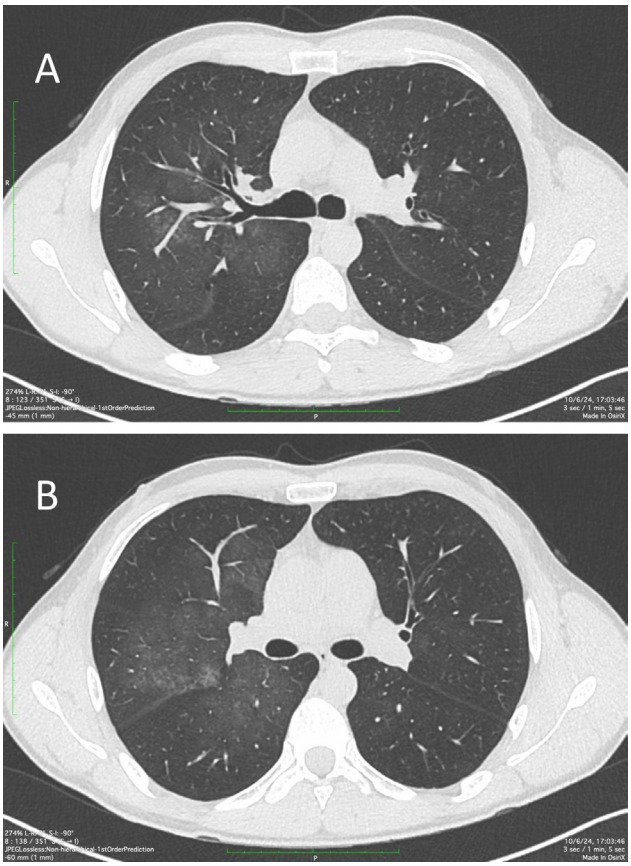


with an incidental finding of bronchial diverticula, and was discharged six days post-dive after ground-glass opacities were found to be resolving upon repeat chest CT. This case is unique as this diver underwent three CTs following the resolution of lung squeeze.

On the morning of the incident, the diver reported a training dive session in which he performed three constant weight bi-fin dives to 20 m, 40 m and 49 m (a personal best). He was practicing ear equalising with mouth-fill only when feeling slight ear pressure rather than in anticipation of ear pressure, which resulted in fewer equalisations. He denied any exertion or physical discomfort during any of the dives. On the last dive to 49 m he reported extending his neck to look up at the surface on the initial ascent. He also tugged on the rope three times rather than one time. He surfaced from all dives feeling well. In the evening, he cleared his throat as he would normally clear mucus and expelled an approximately 2 x 2 cm thick, dark red blood clot. He denied coughing, shortness of breath, congestion, throat soreness, or any discomfort anywhere in his body prior to or after expelling the blood. After 10 hours of sleep, he tried to clear his throat gently and a similar amount and quality of blood clot was expelled again.

On day two post-dive he was seen by a pulmonologist group who performed a bilateral ultrasound of his anterior lung fields and reported right-sided pulmonary oedema. A nasopharyngolaryngoscopy was clear, and a CT chest showed "*mild ill-defined central peri-bronchovascular ground glass opacities in the right upper and middle lobe. A tiny diverticulum measuring up to 2 mm diameter projects from the medial inferior aspect of the left main bronchus and appears to make a connection with a similar sized outpouching projecting from the inferior medial aspect of the right main bronchus, reflecting tiny diverticula/trace fissures connection between the 2 bronchi*" (Figure 1 and Figures 2A–2B). The patient was offered a bronchoscopy

Figure 2
A – CT chest at level of the carina on day two post-dive showing central peri-bronchovascular ground glass opacities in the right upper and middle lobe; B – CT chest at level below the carina on day two post-dive showing central peri-bronchovascular ground glass opacities in the right upper and middle lobe

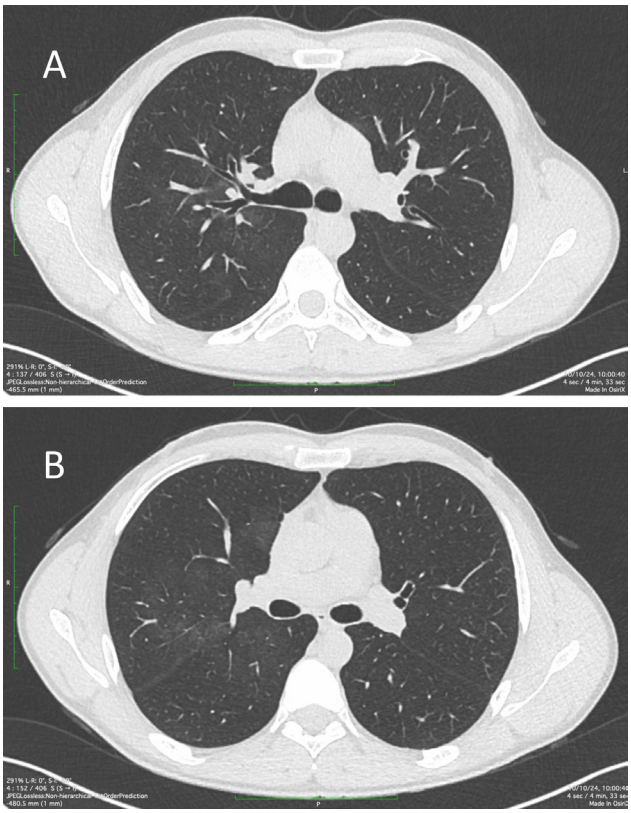


and declined because he did not want to be intubated. He was hospitalised and started on amoxicillin-clavulanic acid 875-125 mg twice a day (BID) for 10 days, methylprednisolone 16 mg BID for 14 days and then a 14-day taper, and omeprazole 20 mg daily. Hospital admission vital signs were blood pressure 126/70, heart rate 80, respiratory rate 15 per minute, peripheral oxygen saturation 97%, temperature 36.4°C. The diver remained asymptomatic.

On day six post-dive, a repeat CT scan showed "*interval decreased extent and density of previous mild ground glass opacities*." (Figure 3). He was discharged with prescriptions to finish his course of antibiotics, steroids, and a proton pump inhibitor, and given instructions to return for a repeat CT 21 days post-dive.

CT day 21 post-dive showed "*interval complete resolution of previous mild ground glass opacities. The tiny diverticula versus minimal fistulous connection between the main bronchi are unchanged*." The diver traveled back home the following week.

Figure 3
A – CT chest at level of the carina on day six post-dive showing decreased extent of ground glass opacities and unchanged bronchial diverticulum; B – CT chest at level below the carina on day six post-dive showing decreased extent of ground glass opacities



Discussion

Lung squeeze, or barotrauma of descent, is a sequela of a failed attempt to maintain pressure equilibrium in the chest cavity during a breath hold dive as the ambient pressure becomes too great. As a result, atelectasis, pulmonary edema, and bleeding from alveolocapillary rupture can occur. Symptoms typically present as dyspnoea, hypoxia, cough, throat soreness, hoarseness, chest pain.[4] CT is of limited value in distinguishing the exact aetiology of alveolar consolidations presenting as ground-glass opacities and requires correlation with clinical context to separate e.g., haemorrhage from oedema.[5] The initial CT of this diver showed ground glass opacities in the right middle and upper lobes, which could be signs of inflammation, pulmonary oedema, or alveolar haemorrhage.[6] Given the diver's only symptom was haemoptysis after a dive, the ground glass opacities seen on his initial CT were likely alveolar haemorrhage. Without bronchoscopy or bronchoalveolar lavage to confirm the bleeding source the haemoptysis could have originated from the alveoli, bronchi, or trachea. The haemoptysis could also possibly be due to tracheobronchial barotrauma and the ground glass opacities may have arisen from upper airway bleeding down the right mainstem bronchus into the right and middle lobe alveoli. We believe this to be unlikely as gravity dependent bleeding would also be expected in the left lung. For this reason it is also unlikely that the small 2 mm bronchial diverticulum is the source. Additionally, at 2 mm it is too small to be a pooling site for bleeding below or above the diverticulum. It is also noteworthy that the diver had findings in the right upper lobe, the same location reported with bleeding after squeeze studied with bronchoscopy.[7]

The freediver reported pulling on the rope three times with his right arm to begin ascent, likely increasing tensile stress on the right lung and causing alveolar haemorrhage. This stress may result from a compressed lung at depth being stretched during unilateral reaching that preferentially expands the right lung, leading to localised barotrauma, while the left lung remains atelectatic. Alveolar haemorrhage often presents as ground-glass or consolidative opacities, and depending on the mechanism of injury, will resolve radiographically and symptomatically within three to 21 days.[6,8,9] The literature yields a few case reports of divers with haemoptysis, some also with dyspnoea, that show radiographic resolution of lung squeeze by 14 to 21 days post-dive. Kiyan et al., and Boussuges et al., report on healthy breath hold divers who experienced haemoptysis immediately post dive. One was found to have bilateral alveolar infiltration suggestive of alveolar haemorrhage on chest CT two hours later with a follow-up CT three weeks post dive showing resolution of alveolar injury. Another was found to have disseminated alveolar opacification on chest CT one day post-dive and confirmed alveolar haemorrhage on broncho-alveolar lavage 36 hours later. Three others showed no lung injury on chest CT two weeks post-dive.[8,9] The present case is unique since the diver received three CTs, shedding light on the time course of resolution.

In this case, the initial CT scan showed ground glass opacities suggesting pulmonary haemorrhage from his dive two days prior. The second CT scan taken six days post-dive showed resolving ground glass opacities indicating the haemorrhage was self-limited and resolving. Clinical history of haemoptysis along with presence of alveolar consolidations six days post dive further suggest an exudative source (such as blood from barotrauma) versus a transudative source (such as immersion pulmonary oedema). Although subclinical fluid extravasation cannot be ruled out, overt immersion pulmonary oedema from extravasating fluid into the pulmonary tissue is unlikely as it typically resolves symptomatically and radiographically in 48 hours.[7,10–13] The final CT scan showed complete resolution of the alveolar haemorrhage. These findings, consistent with haemorrhage on CT but with no hypoxia, dyspnoea, cough, throat or chest discomfort, could indicate that barotrauma of descent in freedivers may be more common than presently thought, and may last much longer.

Corticosteroids have been used previously to treat diving injuries such as decompression illness, barotrauma of the inner ear and sinuses, and swimming induced pulmonary oedema.[14,15] However, new guidelines do not recommend

use of corticosteroids for treatment of these diving related injuries or lung squeeze.[16,17] Treatment of lung squeeze, as reported in the literature, is often conservative management and 100% oxygen.[16] To our knowledge, there are no other reports of high dose corticosteroids and antibiotics used in this context.[18] Corticosteroids are often used clinically for inflammatory pulmonary conditions with ground glass opacities on CT. Poole and Erickson show that in horses, repeated bleeding has been shown to increase airway inflammation.[19]

Finally, the long-term impacts of alveolar haemorrhages among freedivers are not established. Post-dive haemoptysis is relatively common among freedivers,[2] and is not always reported or medically investigated. A recent CT study of 14 free divers with reports of prior squeeze events showed clinically 'normal' scans suggesting that there is no overt chronic pulmonary damage.[20] Though the high frequency of FIPS and haemoptysis among breath-hold divers, along with the increasing popularity of the sport, support the need for further research, both to document the incidence of FIPS and to track pulmonary health in divers throughout the lifespan.

References

1 Lindholm P, Lang MA, Tillmans F, editors. Barotrauma and SIPE in freediving. Proceedings of the San Diego Center of Excellence in Diving/Divers Alert Network 2023 Oct 27-28 workshop. San Diego, CA: Divers Alert Network; 2024. p. 108.

2 Yu E, Valdivia-Valdivia J, Silva F, Lindholm P. Breath hold diving injuries–A primer for medical providers. Curr Sports Med Rep. 2024;23:199–206. doi: 10.1249/JSR.0000000000000583. PMID: 38709946.

3 Yu E, Silva F, Lussier A, Lindholm P. Lung ultrasound as an adjunct to pulse oximetry and respiratory symptoms in the diagnosis of freediving-induced pulmonary syndrome. Wilderness Environ Med. 2024;35:409–16. doi: 10.1177/10806032241281463. PMID: 39279453.

4 Lindholm P, Lundgren CE. The physiology and pathophysiology of human breath-hold diving. J Appl Physiol. 2009;106:284–92. doi: 10.1152/japplphysiol.90991.2008. PMID: 18974367.

5 Reisman S, Chung, Bernheim A. A review of clinical and imaging features of diffuse pulmonary hemorrhage. AJR Am J Roentgenol. 2021;216:1500–9. doi: 10.2214/AJR.20.23399. PMID: 33826359.

6 Marquis KM, Raptis CA, Zak Rajput M, Steinbrecher KL, Henry TS, Rossi SE, et al. CT for evaluation of hemoptysis. Radiographics. 2021;41:742–61. doi: 10.1148/rg.2021200150. PMID: 33939537.

7 Barković I, Maričić V, Reinić B, Marinelli F, Wensveen TT. Haemoptysis in breath-hold divers; where does it come from? Diving Hyperb Med. 2021;51:299–302. doi: 10.28920/dhm51.3.299-302. PMID: 34547782. PMCID: PMC8608440.

8 Kiyan E, Aktas S, Toklu AS. Hemoptysis provoked by voluntary diaphragmatic contractions in breath-hold divers. Chest. 2001;120:2098–100. doi: 10.1378/chest.120.6.2098. PMID: 11742946.

9 Boussuges A, Pinet C, Thomas P, Bergmann E, Sainty JM, Vervloet D. Hemoptysis after breath-hold diving. Eur Respir J. 1999;13:697–9. doi: 10.1183/09031936.99.13369799. PMID: 10232449.

10 Cialoni D, Sponsiello N, Marabotti C, Marroni A, Pieri M, Maggiorelli F, et al. Prevalence of acute respiratory symptoms in breath-hold divers. Undersea Hyperb Med. 2012;39:837–44. PMID: 22908840.

11 Lindholm P, Ekborn A, Oberg D, Gennser M. Pulmonary edema and hemoptysis after breath-hold diving at residual volume. J Appl Physiol (1985). 2008;104:912–7. doi: 10.1152/japplphysiol.01127.2007. PMID: 18202166.

12 Linér MH, Andersson JP. Pulmonary edema after competitive breath-hold diving. J Appl Physiol (1985). 2008;104:986–90. doi: 10.1152/japplphysiol.00641.2007. PMID: 18218906.

13 Moon RE. Treatment of diving emergencies. Crit Care Clin. 1999;15:429–56. doi: 10.1016/S0749-0704(05)70062-2. PMID: 10331136.

14 Moon RE. Immersion pulmonary edema: drowning from the inside. Undersea Hyperb Med. 2019;46:577–9. PMID: 31683355.

15 Yamanashi H, Koyamatsu J, Nobuyoshi M, Murase K, Maeda T. Exercise-induced pulmonary edema in a triathlon. Case Rep Med. 2015;2015:968152. doi: 10.1155/2015/968152. PMID: 26229538. PMCID: PMC4503573.

16 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.

17 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.

18 Anderson W, Murray P, Hertweck K. Dive medicine: current perspectives and future directions. Curr Sports Med Rep. 2019;18:129–35. doi: 10.1249/JSR.0000000000000583. PMID: 30969238.

19 Poole DC, Erickson HH. Exercise-induced pulmonary hemorrhage: where are we now? Vet Med (Auckl). 2016;7:133–48. doi: 10.2147/VMRR.S120421. PMID: 30050846. PMCID: PMC6044800.

20 Grey T, Dujić G, Lindholm P, Drviš I, Barak O, Dujić Z, et al. No evidence of lung pathology in freedivers with history of pulmonary barotrauma. Chest. 2025;167:1458–61. doi: 10.1016/j.chest.2024.08.028. PMID: 39214415.

Conflicts of interest and funding

This work was funded by the Divers Alert Network. The funder had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript. No conflicts of interest were declared.

Submitted: 21 April 2025
Accepted after revision: 22 June 2025

Hyperbaric oxygen therapy for late onset dropped head syndrome following mantle field radiation therapy for Hodgkin lymphoma: a case report and literature review

Shane A Rosenzweig[1,2], Stephen R Thompson[3,4], Michael H Bennett[4,5]

[1] *Department of Radiation Oncology, Crown Princess Mary Cancer Centre, Westmead Hospital, Sydney, Australia*
[2] *Central Clinical School, University of Sydney, Sydney, Australia*
[3] *Department of Radiation Oncology, Prince of Wales Hospital, Sydney, Australia*
[4] *Prince of Wales Clinical School, Faculty of Medicine, University of New South Wales, Sydney, Australia*
[5] *Department of Diving and Hyperbaric Medicine, Prince of Wales Hospital, Sydney, Australia*

Corresponding author: *Dr Shane A Rosenzweig, Crown Princess Mary Cancer Centre, 166-174 Hawkesbury Rd, Westmead, NSW 2145, Australia*
ORCiD: *0009-0007-1891-5339*
shane.rosenzweig@health.nsw.gov.au

Keywords
Case reports; Malignancy; Musculo-skeletal; Radiotherapy; Treatment sequelae

Abstract

(Rozenzweig SA, Thompson SR, Bennett MH. Hyperbaric oxygen therapy for late onset dropped head syndrome following mantle field radiation therapy for Hodgkin lymphoma: a case report and literature review. Diving and Hyperbaric Medicine. 2025 30 September;55(3):275–279. doi: 10.28920/dhm55.3.275-279. PMID: 40986924.)
The authors present the first documented case of the apparent effective use of hyperbaric oxygen therapy (HBOT) for the treatment of radiation-induced dropped head syndrome (DHS). DHS is a condition associated with progressive and often severe weakness of cervical paraspinal muscles, especially the neck extensors. This results in loss of horizontal gaze and in advanced cases causes a chin-on-chest deformity. Radiation-induced DHS is a rare, primarily late-term complication first described in patients treated with mantle field radiotherapy for Hodgkin lymphoma, though there is an increasing body of literature demonstrating a wide range of presentations. A 59-year-old man with a history of stage 2A Hodgkin lymphoma 34 years prior had been treated with extended field radiotherapy, including mantle radiotherapy, totalling 40 Gy in 19 fractions. He presented with three years of progressive neck extension weakness with associated stiffness and intermittent dysphagia. The patient underwent 60 sessions of HBOT, in conjunction with physiotherapy and thiamine replacement and demonstrated improvement of his postural maintenance and dysphagia. His improved function was maintained at three years follow-up. We discuss the literature on the management of this rare condition, including the rationale for using HBOT which is well documented for the treatment of other late-term radiotherapy side effects. This case adds to the increasing literature on the management of DHS and describes a novel approach to the management of this often-debilitating condition.

Introduction

Dropped head syndrome (DHS) is a condition associated with progressive and often severe weakness of cervical paraspinal muscles, especially the neck extensors. This results in the mechanical inability to maintain horizontal gaze and in advanced cases causes a chin-on-chest deformity.[1] DHS is a known complication of a variety of conditions including; inflammatory myopathy, amyotrophic lateral sclerosis, Cushing's syndrome, myasthenia gravis and ankylosing spondylitis.[2] Its relation to radiotherapy was first described by Johansson et al.,[3] in the setting of mantle field radiation therapy (RT) for Hodgkin lymphoma (HL).

Previously, the standard of care for HL involving cervical or mediastinal lymph nodes was extended field RT (including mantle field RT) or, more recently, chemotherapy and mantle field RT. Mantle field RT consists of fields encompassing the submandibular, cervical, supraclavicular, infraclavicular, axillary, mediastinal, subcarinal and hilar lymph nodes.[4] Although the use of mantle field RT has been radically reduced with the introduction of more modern techniques and combination chemotherapy protocols, there are many surviving patients who underwent this treatment.[5] As of 2009, 1,415 HL survivors were treated with mantle field RT between 1965 and 1995 in the Netherlands alone.[6] In the first of its kind, a recent study of childhood survivors who received radiotherapy to the neck for HL and other malignancies demonstrated five out of 41 (12%) patients had DHS.[7]

Whilst DHS was first reported in HL survivors, likely due to their good oncological outcomes, radiation-induced DHS is primarily associated with high-dose RT to the head, neck

and upper chest.[5,7,8] This is demonstrated with increasing recognition of early and late onset DHS in head and neck cancers,[9–14] indicating that it is likely a radiation effect rather than a tumour-specific pathogenesis.

Despite this increasing body of literature, DHS remains a rare condition and there is no consensus on the appropriate management.[15] Here we describe a case of DHS with a sustained positive outcome following treatment with hyperbaric oxygen therapy (HBOT) in combination with physiotherapy and thiamine replacement. To the best of our knowledge this is the only documented case of the use of HBOT.

Case report

The patient provided written consent for reporting of his case details.

A 59-year-old male presented with a three-year history of neck extension weakness with associated stiffness and intermittent dysphagia described as choking on foods once per month. He reported no lower limb difficulties. Examination revealed a hoarse voice, and his head was dropped in flexion to 45 degrees. There was wasting of the neck and proximal shoulder muscles bilaterally, with reduced proximal power. All reflexes were symmetrical, and coordination was normal. There was preserved proprioception and a mild cape-like sensory loss over the chest and upper back – though this was only elicited by one of the two neurologists that were consulted. Examination of the lower limbs was normal.

He had a history of stage 2A HL 34 years prior, and had been treated with extended field RT, including mantle field RT, without chemotherapy, totalling 40 Gy in 19 fractions (including a single sequential boost to the mediastinum), by parallel opposed 6 MV anterior-posterior (AP)/posterior-anterior (PA) photon fields (with AP and PA fields given on alternate days). Cervical spinal cord dose exposure was retrospectively estimated at 42 Gy ($\alpha/\beta = 2$). Following treatment, the patient experienced self-limiting subacute L'Hermitte's syndrome, but otherwise recovered well.

Magnetic resonance imaging (MRI) of the spine showed mild disc degenerative change at C4/5 and C5/6, with no other abnormalities. A dedicated MRI of the neck and shoulder girdle was not conducted. Nerve conduction studies demonstrated a generalised sensory neuropathy of axonal type, with motor studies within normal limits. Limited electromyography (EMG) was undertaken given concurrent warfarin prescription, but did not demonstrate evidence of neurogenic change within the muscles. In light of the presentation and findings, a diagnosis of DHS, due to radiation myelopathy, was made.

An initial trial of prednisolone was commenced six weeks after presentation, starting at 25 mg twice daily for three days and then weaned over 12 days. This resulted in an improvement in subjective well-being but without improvement in strength. The patient had not previously undergone physiotherapy and was noted to consume 40 g of alcohol per day. He was commenced on a regimen of physiotherapy and thiamine replacement. Given the evidence of HBOT in the treatment of other late radiation sequelae and incomplete improvement of symptoms with prednisolone, he was also referred to the Diving and Hyperbaric Medicine Department and completed a course of 40 HBOT treatments. A month later this had led to a significant improvement in postural maintenance, thereafter, reducing over the following three months, although not to pre-treatment levels. He completed a further 20 HBOT treatments seven months later, to further benefit. Six months following completion of HBOT, improvement was maintained, with the patient being able to maintain neck extension for prolonged periods. Examination revealed strength 4+/5. He described improved function, and resolution of dysphagia. He had remained on thiamine replacement and continued physiotherapy. Nearly three years later he had maintained improvement in neck strength and resultant function.

Discussion

Radiation-induced DHS remains a very rare complication of not only HL survivors treated with mantle field RT but also patients who received radiotherapy for lung and head and neck cancers. Radiation-induced DHS has now been described as having a bimodal presentation of early and late onset – with likely differing pathophysiology.

A small series[12] presented four patients with early-onset DHS who had received concurrent radiotherapy and chemotherapy for head and neck squamous cell carcinoma and developed DHS within six months. The authors proposed that the mechanism of this neurotoxicity is secondary to a combination of inflammation, reactive oxygen damage, and mitochondrial dysfunction resulting in apoptotic cell death at a neuronal level.[12] Others have proposed that there is an autoimmune response to the resultant inflammation and that early-onset is usually self-limiting and responsive to corticosteroid treatment.[9] The role of chemotherapy in DHS is difficult to elucidate, to date, DHS has not been linked to chemotherapy alone, though there may be a synergistic effect with radiation.[4,13]

Late-onset DHS can have a latency of over 30 years, as discussed in the case presented. Despite the increasing body of literature there remains no consensus on the appropriate management of late-onset DHS.[15] Likewise, the mechanism of late-onset radiation-induced DHS is unclear, some findings have been consistent with primary damage to neurons and others have shown primary or secondary damage to muscles.[5,9] Experimental results have showed that the majority of muscles within the previous radiation field have demonstrated mostly myogenic damage whereas muscles outside of the field showed mostly neurogenic

damage. It has been proposed that muscle damage beyond the RT fields was secondary to neurogenic damage of upstream nerve roots and/or of the brachial plexus which were situated within the radiation field.[5,9,16]

It is well known, and not exclusively linked to DHS, that endothelial cells and vascular smooth muscles are susceptible to radiation damage and thus, it is hypothesised that myogenic damage may be due to extrinsic factors such as progressive microvascular fibrosis.[5,13]

Given the complexity, prolonged latency, and rarity, it can be difficult to diagnose DHS and provide a comprehensive evidence-based management plan. In the first instance, assessment and exclusion of any other underlying conditions that can cause DHS should be thoroughly undertaken. We briefly review the literature to discuss conservative and surgical management, prevention and screening, and discuss the rationale behind hyperbaric oxygen therapy.

CURRENT MANAGEMENT

Conservative management has remained the mainstay of treatment for DHS, though this demonstrates mixed efficacy in case reports and there is no consensus on optimal management. In case reports of late-onset DHS, intensive rehabilitation has been shown to stabilise,[17] or gradually improve symptoms, though not back to baseline.[8]

Surgery has been proposed as a second-line treatment in patients who have failed conservative treatment or who have refractory symptoms.[18] Surgical options include pre-operative spinal traction, posterior fusions, anterior/posterior fusion or multi-level osteotomies. In a small case series[14] of seven patients with radiation induced DHS, surgery demonstrated significant improvement in both clinical and radiological outcomes. However, it was associated with high peri-operative complications, such as pneumonia and respiratory distress requiring temporary reintubation. These were short-lived and overall, the patient reported long-term outcomes remained satisfactory.[14]

A systematic review[15] including 129 patients assessing all causes of DHS demonstrated that for the 14 patients that underwent surgery alone, 93% had positive outcomes. The rate of positive outcomes reduced to 73–88% with combinations of medical and immunosuppressive treatments and significantly reduced to 18% with bracing or physiotherapy alone.[15] The importance of a multi-modal approach is reflected in our case report, where a combination of physiotherapy, thiamine replacement and HBOT provided a meaningful improvement in the patient's symptoms.

PREVENTION AND SCREENING

There appears to be a clear association between dose of radiation the extensor muscles receive and incidence of DHS. In their review[10] of early-onset DHS in head and neck cancer, Inaba et al., discussed three patients with DHS who had mean neck extensor muscle dose of 42.3 Gy, 58.5 Gy and 60.9 Gy compared with nine control patients who received less than 50 Gy. This study also compared the dose-volume histogram which revealed that V_{60Gy} (the volume receiving 60 Gy) and V_{70Gy} (the volume receiving 70 Gy) were significantly greater in the patients with DHS compared to those without DHS. The mean value for V_{60Gy} and V_{70Gy} in patients with DHS was 32.7% and 7.0% respectively and 5.7% and 0.5% in patients without DHS. Thus a dose constraint of less than 46 Gy–50 Gy was proposed.[10] However, given the rare nature of DHS and insufficient evidence, this dose constraint should not impede the adequate dosage and local control of the primary malignancy.

Screening and early intervention can reduce the morbidity of DHS. Rieken et al.,[7] in their recent systematic review, proposed a diagnostic algorithm for radiation-induced DHS which focused on a detailed history and clinical examination including; measurements of neck circumference, detailed neurological examination and patient reported questionnaires to assess for cervical muscle atrophy. In their algorithm EMG and muscle biopsy were not routinely included given the often-conflicting results. A biopsy was undertaken as 'ultima ratio' for unresolved cases. They also indicated that MRI scans did not reveal additional information in their study however recommended further evaluation in larger cohorts. It was advised that a focused history and clinical examination were sufficient to diagnose radiation-induced DHS. They went on to recommend the integration of diagnostic algorithms to facilitate prompt diagnosis and intervention as part of comprehensive long-term follow-up care of childhood cancer survivors.[7] This algorithm lays the foundation for recognition of DHS, but work must be done in devising consensus guidelines for the management of DHS.

HYPERBARIC OXYGEN THERAPY

A small case series by van Leeuwen-Segarceanu et al.,[5] presented 12 HL survivors who underwent mantle field RT. Examination revealed ten patients with neck weakness, of which two patients had severe neck weakness resulting in DHS. The remaining two patients did not have neck weakness and it was noted that they performed sports with high neck activation. The authors proposed that neck muscle weakness was secondary to primary vascular injury which led to myogenic damage of the muscles in-field. Thus, treating DHS with intensive rehabilitation may promote increased vascularisation by formation of collateral vessels which may contribute to maintaining muscle strength.[5] This hypothesis can be extended to the use of HBOT.

HBOT is often given at pressures of 2.0–2.5 absolute atmospheres for periods up to 120 minutes daily for a total of 30–60 sessions. HBOT causes a series of physiological effects through increasing plasma oxygen transport and increasing tissue oxygen availability. Subsequently, a high oxygen gradient influences angiogenesis and stimulates

microvascularisation and neocollagenisation leading to the induction of tissue repair. The oxygen tension of irradiated tissues recovers over the course of treatment and is maintained for a minimum of three years without the continued need for HBOT.[19]

Serious radiation complications have been documented in 5% of all patients receiving RT and HBOT has been used as a safe and effective treatment for some radiation-induced tissue injuries.[19] Whilst there are few randomised controlled trials, retrospective data shows that there is statistically significant improvement seen in xerostomia, osteoradionecrosis of the mandible, osteoradionecrosis prophylaxis, soft tissue necrosis and radiation proctitis or cystitis.[20] It has also been showed that the majority of these cases had sustained improvement through a medium follow-up of 3.8 years.[21]

A prolonged beneficial effect was associated with HBOT in the case presented, whereby he had improved neck strength and function at nearly three years follow up.

Conclusions

Prognosis of DHS remains poor and there are no guidelines on the management of this often-debilitating condition. It is crucial that clinicians become aware of DHS as both a rare early and late-term sequalae of RT. Diagnostic algorithms, dose constraints, conservative and operative management have been discussed in the literature. HBOT has proven beneficial in other late adverse effects of RT[22] and is well established for inducing angiogenesis and neocollagenisation which are essential for myogenic recovery. Exclusion of other causes, conservative management and optimisation of medical co-morbidities play important roles in the management of DHS. Though we cannot fully evaluate the efficacy of HBOT in a single case, HBOT in combination with physiotherapy and thiamine replacement, when necessary, may offer an alternative in refractory cases and may be more efficacious than conservative and medical management. More work is required in this field to fully ascertain the effects of HBOT in DHS and efforts are required to propose and evaluate guidelines on the management of radiation-induced DHS.

References

1 Robert M, Lessard LER, Bouhour F, Petiot P, Fenouil T, Svahn J, et al. Inaugural dropped head syndrome and camptocormia in inflammatory myopathies: a retrospective study. Rheumatology (Oxford). 2024;63:506–15. doi: 10.1093/rheumatology/kead347. PMID: 37462538. PMCID: PMC10837000.

2 Luo JJ. Isolated head drop triggered by neck surgery following concomitant chemoradiotherapy. J Clin Neuromuscul Dis. 2008;9:348–51. doi: 10.1097/CND.0b013e3181629396. PMID: 18344717.

3 Johansson AS, Erlanson M, Lenner P, Lindh J, Osterman B. Late side-effects are common after treatment of Hodgkin's disease. Muscular atrophy following radiotherapy is a neglected risk. Lakartidningen. 1998;95:44–7. PMID: 9458645.

4 Grimm S, Chamberlain M. Hodgkin's lymphoma: a review of neurologic complications. Adv Hematol. 2011;2011:624578. doi: 10.1155/2011/624578. PMID: 20975772. PMCID: PMC2957132.

5 van Leeuwen-Segarceanu E, Dorresteijn L, Pillen S, Biesma D, Vogels O, van Alfen N. Progressive muscle atrophy and weakness after treatment by mantle field radiotherapy in Hodgkin lymphoma survivors. Int J Radiat Oncol Biol Phys. 2012;82:612–8. doi: 10.1016/j.ijrobp.2010.11.064. PMID: 21288655.

6 De Bruin ML, Dorresteijn LD, van't Veer MB, Krol AD, van der Pal HJ, Kappelle AC, et al. Increased risk of stroke and transient ischemic attack in 5-year survivors of Hodgkin lymphoma. J Natl Cancer Inst. 2009;101:928–37. doi: 10.1093/jnci/djp147. PMID: 19535773.

7 Rieken S, Kronziel LL, Langer T, Rades D, Boppel T, Trillenberg P, et al. Proposal of a diagnostic algorithm for radiation-induced dropped head syndrome in long-term childhood cancer survivors based on a prospective study in a specialized clinical setting and a review of the literature. J Cancer Res Clin Oncol. 2023;149:17865–79. doi: 10.1007/s00432-023-05480-w. PMID: 37947868. PMCID: PMC10725355.

8 Ochi N, Yamane H, Honda Y, Takigawa N. Dropped head syndrome following mantle radiation therapy. Intern Med. 2016;55:421–2. doi: 10.2169/internalmedicine.55.5579. PMID: 26875972.

9 Shields LB, Iyer VG, Liang J, Zhang YP, Shields CB. Early-onset dropped head syndrome and person-in-the-barrel syndrome after radiation therapy: Clinical, electromyographic, and MRI findings. Cureus. 2022;14(8):e28279. doi: 10.7759/cureus.28279. PMID: 36158382. PMCID: PMC9492447.

10 Inaba K, Nakamura S, Okamoto H, Kashihara T, Kobayashi K, Harada K, et al. Early-onset dropped head syndrome after radiotherapy for head and neck cancer: dose constraints for neck extensor muscles. J Radiat Res. 2015;57:169–73. doi: 10.1093/jrr/rrv055. PMID: 26684338. PMCID: PMC4795943.

11 Inoue R, Sawamura M, Yoshimura M, Takahashi R. Dropped head syndrome due to chemoradiotherapy for nasal lymphoma, characterised by short tau inversion recovery high signal intensity in the cervical paraspinal muscles. BMJ Case Rep. 2022;15(11):e252446. doi: 10.1136/bcr-2022-252446. PMID: 36368727. PMCID: PMC9660595.

12 Smillie I, Ellul D, Townsley R, James A, Clark B, Petty RK, et al. Head drop syndrome secondary to multimodality treatments for head and neck cancer. Laryngoscope. 2013;123:938–41. doi: 10.1002/lary.23857. PMID: 23404786.

13 Hashimoto Y, Maebayashi K, Izumi S, Motegi A, Mitsuhashi N. Dropped head syndrome induced by chemoradiotherapy for nasopharyngeal carcinoma: A case report. Jpn J Clin Oncol. 2012;42:1091–3. doi: 10.1093/jjco/hys135. PMID: 22914321.

14 Verla T, Vedantam A, North RY, Xu DS, Raber MR, Fuentes A, et al. Surgical management of postradiation, dropped head spinal deformity in patients with head and neck cancer. World Neurosurg. 2021;156:e1–e8. doi: 10.1016/j.wneu.2021.07.001. PMID: 34245881.

15 Drain JP, Virk SS, Jain N, Yu E. Dropped head syndrome: A systematic review. Clin Spine Surg. 2019;32:423–9. doi: 10.1097/bsd.0000000000000811. PMID: 30844858.

16 Ando J, Ueno Y, Yasuda H, Ando M, Edahiro Y, Honda T, et al. Radiation-induced myopathy developing in a

Hodgkin Lymphoma patient: an autopsy case with systemic muscle sampling. Case Rep Oncol. 2021;14:338–42. doi: 10.1159/000512070. PMID: 33776726. PMCID: PMC7983573.

17 Di Stefano V, Attanasi C, Ferrante C, Di Muzio A. Late onset of dropped head syndrome following mantle radiation therapy for Hodgkin lymphoma. BMJ Case Rep. 2018;11(1):e226822. doi: 10.1136/bcr-2018-226822. PMID: 30567263. PMCID: PMC6301549.

18 Brodell JD, Sulovari A, Bernstein DN, Mongiovi PC, Ciafaloni E, Rubery PT, et al. Dropped head syndrome: an update on etiology and surgical management. JBJS Rev. 2020;8:e0068–e. doi: 10.2106/JBJS.RVW.19.00068. PMID: 32105239.

19 Fernández E, Morillo V, Salvador M, Santafé A, Beato I, Rodríguez M, et al. Hyperbaric oxygen and radiation therapy: a review. Clin Transl Oncol. 2021;23:1047–53. doi: 10.1007/s12094-020-02513-5. PMID: 33206332.

20 Lin ZC, Bennett MH, Hawkins GC, Azzopardi CP, Feldmeier J, Smee R, et al. Hyperbaric oxygen therapy for late radiation tissue injury. Cochrane Database of Syst Rev. 2023;8(8):CD005005. doi: 10.1002/14651858.CD005005.pub5. PMID: 37585677.

21 Tahir ARM, Westhuyzen J, Dass J, Collins MK, Webb R, Hewitt S, et al. Hyperbaric oxygen therapy for chronic radiation-induced tissue injuries: Australasia's largest study. Asia Pac J Clin Oncol. 2015;11:68–77. doi: 10.1111/ajco.12289. PMID: 25382755.

22 Oscarsson N, Rosén A, Müller B, Koskela LR, Giglio D, Kjellberg A, et al. Radiation-induced cystitis treated with hyperbaric oxygen therapy (RICH-ART): long-term follow-up of a randomised controlled, phase 2-3 trial. EClinicalMedicine. 2025;83:103214. doi: 10.1016/j.eclinm.2025.103214. PMID: 40291346. PMCID: PMC12033922.

Acknowledgements

The late Professor Michael H Bennett AM is included in the authorship for his significant contributions to the clinical care and management of this case prior to his passing. His dedication to patient care and clinical excellence is remembered with deep respect.

Conflicts of interest and funding: nil

Submitted: 6 March 2025
Accepted after revision: 5 July 2025

Periorbital emphysema after a dry hyperbaric chamber exposure

Ping Wu[1], Kin Bong Tang[2], Wing Wa Yan[2]

[1] *Tuen Mun Hospital, Hong Kong*
[2] *Pamela Youde Nethersole Eastern Hospital, Hong Kong*

Corresponding author: *Dr Ping Wu, Tuen Mun Hospital, Tsing Chung Koon Road, Hong Kong*
ORCiD: *0000-0001-6215-4847*
wp013@ha.org.hk

Keywords
Case report; Complications; Hyperbaric oxygen; Hyperbaric medicine; Ophthalmology; Side effects

Abstract

(Wu P, Tang KB, Yan WW. Periorbital emphysema after a dry hyperbaric chamber exposure. Diving and Hyperbaric Medicine. 2025 30 September;55(3):280–283. doi: 10.28920/dhm55.3.280-283. PMID: 40986925.)
We report a case of a patient developing extensive periorbital, facial, and neck emphysema during hyperbaric oxygen treatment for his facial osteoradionecrosis after sequestrectomy. Hyperbaric physicians should be alert for the potential development of this complication during the treatment and have a contingency plan.

Introduction

Middle ear barotrauma is the most common complication of hyperbaric oxygen therapy (HBOT), with reported incidence rates up to 43.2%.[1] While Valsalva manoeuvre is the most effective pressure-equalising techniques, it may rarely cause cervical surgical emphysema, particularly in patients with pre-existing anatomical defects.[2,3] Here we describe a case of a patient who developed extensive periorbital, facial, and neck emphysema during HBOT for osteoradionecrosis.

Case report

The patient reported here provided written consent for publication of his case history and images.

Our patient was a 70-year-old man with a history of nasopharyngeal carcinoma treated with radiotherapy in 1996. This was later complicated with bilateral sensorineural hearing loss, empty sella syndrome, and bilateral temporal bone osteoradionecrosis. His medical history also included isolated idiopathic right 6th nerve palsy, tuberculosis of the kidney for which a left nephrectomy was performed, and hyperlipidaemia. Around half a year ago, he had dental extractions (sites 41 to 47). In this index admission, he developed fever, right buccal swelling, jaw pain, and headache. Physical examination showed pus discharge on expression at the 46–47 dental extraction site. Computed tomography with contrast was performed on day two of admission, and it showed osteolytic lesions at the right mandible retromolar area and the right posterior maxilla. Right mandibular and maxillary osteoradionecrosis was diagnosed. Debridement and sequestrectomy of the right facial osteoradionecrosis were performed on day four of admission in view of active infection. Necrotic bone was removed with placement of a buccal fat graft during the surgery. A total of 30 sessions of hyperbaric oxygen therapy was planned to promote wound healing postoperatively.

On Day five of his admission, we arranged his first session of hyperbaric oxygen therapy (U.S. Navy Treatment Table 9, maximum pressure at 243 kPa (2.4 atmospheres absolute [atm abs]). During the compression, the patient reported no difficulty in equalising the pressure in both ears with Valsalva manoeuvres. However, at 50 kPa, we noticed he developed progressive, painless right periorbital and facial swelling. He continued the compression and reached the treatment pressure. He reported difficulty in opening his right eye, but he experienced no pain or discomfort. Physical examination showed right periorbital and facial crepitus, with normal bilateral pupillary light reaction, and his ocular movement was the same as before the session. Chest examination was also performed to ensure he had no pneumothorax. There was no chest wall emphysema and the air entry was equal for both sides. Emergency equipment for needle thoracocentesis, chest drain insertion, lateral canthotomy, and inferior cantholysis was prepared outside the chamber for use in case of pneumothorax or orbital compartment syndrome development before the decompression. During the decompression, the swelling in his right periorbital and facial areas continued to increase. We closely monitored the patient's symptoms and, in particular, any development of pain in the eyes or chest. We performed the decompression at 9.7 kPa per minute. Although there was a visible increase in the right periorbital and facial swelling during the decompression, he reported no pain or shortness of breath.

Figure 1
Extensive non-tender swelling over the right periorbital and facial region

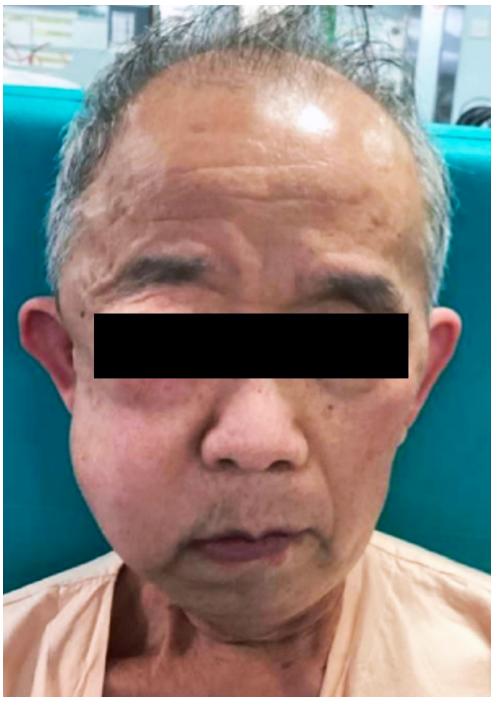
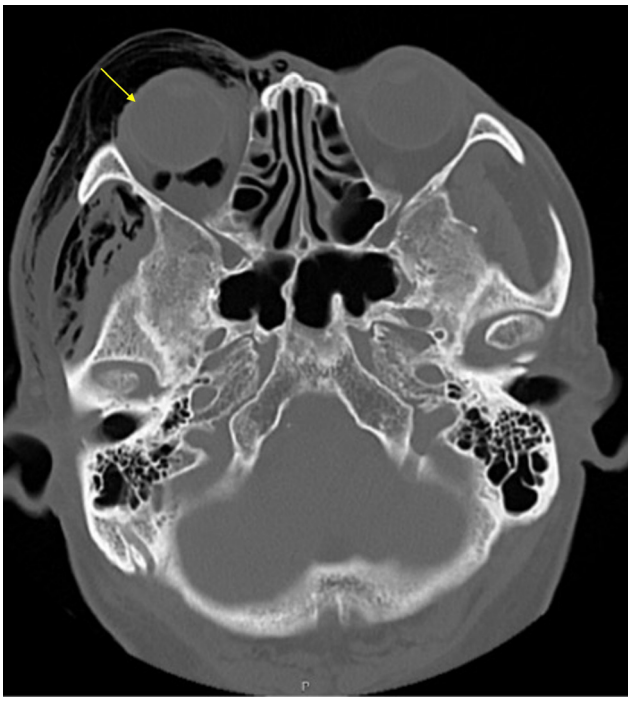


Figure 2
Axial plain computed tomography of the head showed extensive right orbital emphysema with the involvement of the retro-orbital area (with arrow pointing to affected region)



The HBOT session lasted 125 minutes. After decompression, a repeated physical examination was performed and showed extensive non-tender swelling over the right periorbital and facial region (Figure 1) with crepitus on palpation. Plain computed tomography imaging of his face and orbit was also performed. It showed extensive subcutaneous emphysema at the right masticator space, left parapharyngeal and left masticator space, left posterior cervical space, and left carotid space, as well as extensive right orbital emphysema with the involvement of the retro-orbital area (Figures 2 and 3). There was no tenting of the posterior sclera of the right globe or right eye proptosis. A lateral neck X-ray showed prevertebral gas (Figure 4), and a chest X-ray demonstrated left neck surgical emphysema. There was no pneumothorax or pneumomediastinum. The patient was then assessed by an otorhinolaryngologist; tracking of gas from the Valsalva manoeuvre via the right pterygopalatine fossa defect created during surgery was suspected. He was also assessed by an ophthalmologist. The slit lamp examination was normal. We carefully reviewed the benefits and risks of HBOT and decided to continue with additional HBOT sessions. Bilateral myringotomy with gold plate insertion was performed to prevent any further need of the Valsalva manoeuvre during the therapy. The patient continued to receive the remaining planned daily session of HBOT uneventfully. Three days afterwards, his right periorbital and facial swelling completely subsided.

Discussion

The current treatment strategy for osteonecrosis of the jaw is aimed at a multimodal approach, which combines surgery and HBOT as adjuvant therapy.[4] The famously quoted protocol used in the treatment of osteonecrosis is the Marx protocol, which had 30 sessions arranged before surgery, such as dental extraction, and 10 more sessions scheduled after the surgery.[5] In this case, we were unable to provide pre-surgical hyperbaric oxygen treatment due to the emergency nature of the surgery.

Periorbital emphysema, although uncommon, has been reported in literature after both wet and dry diving. The proposed risk factors included facial trauma, repeated forceful Valsalva manoeuvres and recent upper respiratory tract infection.[6] In our patient, he just had the facial surgery the day before, and he performed repeated forceful Valsalva manoeuvres during his first HBOT session. The defect over the pterygopalatine fossa secondary to the surgery allows the passage of gas to the subcutaneous layer. The reported intraoral pressure during the Valsalva manoeuvre could reach up to 40 mmHg.[7] This high pressure further pushes gas to the subcutaneous layer, resulting in progressive swelling over the patient's right face and periorbital region. Subcutaneous emphysema has also been reported after other head and neck surgery, such as tonsillectomy.[8] Yet, in a randomised controlled trial looking into hyperbaric

Figure 3
Axial plain computed tomography of the head and face showed extensive subcutaneous emphysema at right masticator space, left parapharyngeal and left masticator space (with arrows pointing to affected region)

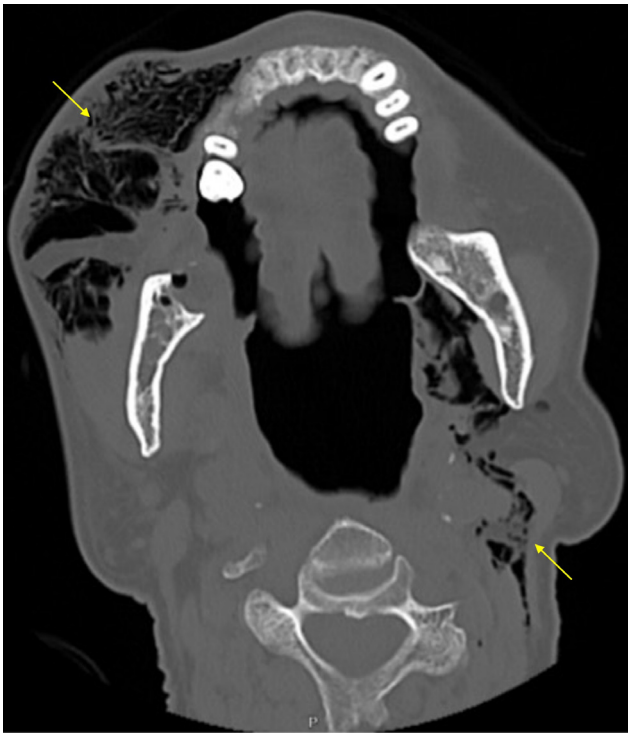
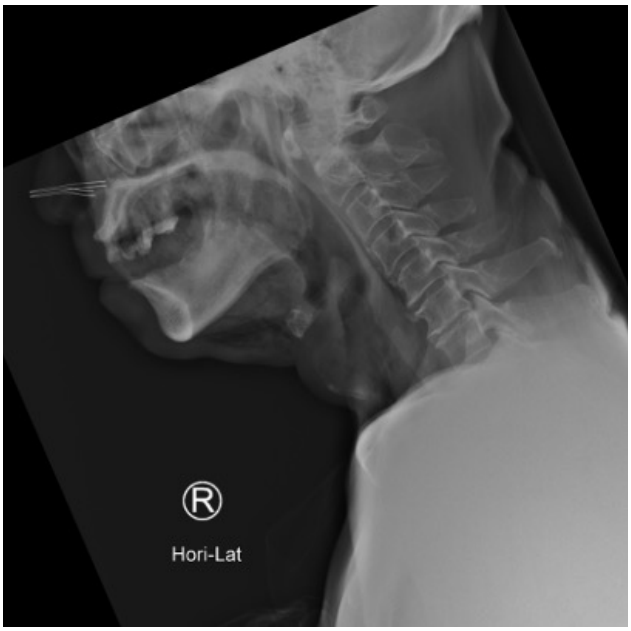


Figure 4
Lateral neck X-ray showing prevertebral gas



oxygen to prevent osteoradionecrosis of the irradiated mandible (HOPON study), which included 47 patients in the hyperbaric oxygen therapy arm, none developed head and neck surgery emphysema.[9]

According to Boyle's law, surgical emphysema is expected to further increase in both size and area as the pressure decreases during the decompression phase of HBOT. Gas in the retro-orbital area can lead to an increase in intra-ocular pressure, potentially resulting in orbital compartment syndrome. Blindness resulting from orbital emphysema had been reported.[10] Fortunately, this didn't occur in our patient, despite his severe periorbital emphysema.

If the patient developed pneumothorax or pneumomediastinum during the decompression, this could result in tension pneumothorax and tension pneumomediastinum. Orbital compartment syndrome could also occur during the decompression phase. Our contingency plan was an emergency stop should he develop any shortness of breath, chest pain, or eye pain. If pneumothorax was suspected, immediate needle thoracocentesis would be performed. Recompression was planned if the patient showed any signs of arterial gas embolism. If the patient developed rapidly worsening visual acuity and eye pain, we planned a lateral canthotomy and inferior cantholysis. After these potential emergencies settled, the decompression rate would be slowed to 3 kPa per minute to minimise the risk of sudden re-expansion of the gas phase.

Conclusions

Any communication between the nasopharyngeal tract and subcutaneous tissues could allow gas entry, especially if nasopharyngeal pressures are elevated by attempts to Valsalva. Bilateral myringotomy or grommet insertion could therefore prevent the development of facial and periorbital surgical emphysema by eliminating the need for the Valsalva manoeuvre. It may be difficult to predict who may need a prophylactic myringotomy to prevent this complication because the defect could be minor and hard to detect. Although a decision whether to perform prophylactic myringotomies in similar patient would be debatable, hyperbaric physicians should be alert for the potential development of facial and periorbital emphysema and have a contingency plan ready.

References

1 Heyboer M 3rd, Wojcik SM, Grant WD, Chambers P, Jennings S, Adcock P. Middle ear barotrauma in hyperbaric oxygen therapy. Undersea Hyperb Med. 2014;41:393–7. PMID: 25558548.

2 Tanya SM, Kulbay M, Kherani F, Mydlarski MB. Valsalva-induced orbital and subcutaneous periorbital emphysema secondary to lamina papyracea dehiscence: a case report. Case Rep Ophthalmol. 2025;16:202–206. doi: 10.1159/000544098. PMID: 40241927. PMCID: PMC11908810.

3 Paouris D, Barkociová J, Pavlík Š, Šebová I. Deep subcutaneous cervical emphysema related to mastoid

fracture in an adolescent patient - case report. Clin Case Rep. 2022;10(9):e6306. doi: 10.1002/ccr3.6306. PMID: 36110334. PMCID: PMC9465693.

4 Ceponis P, Keilman C, Guerry C, Freiberger JJ. Hyperbaric oxygen therapy and osteonecrosis. Oral Dis. 2017;23:141–51. doi: 10.1111/odi.12489. PMID: 27062390.

5 Marx RE, Johnson RP, Kline SN. Prevention of osteoradionecrosis: a randomized prospective clinical trial of hyperbaric oxygen versus penicillin. J Am Dent Assoc. 1985;111:49–54. doi: 10.14219/jada.archive.1985.0074. PMID: 3897335.

6 Tseng WS, Lee HC, Kang BH. Periorbital emphysema after a wet chamber dive. Diving Hyperb Med. 2017;47:198–200. doi: 10.28920/dhm47.3.198-200. PMID: 28868601. PMCID: PMC6159621.

7 Kumar CM, Van Zundert AAJ. Intraoperative Valsalva maneuver: a narrative review. Can J Anaesth. 2018;65:578–85. doi: 10.1007/s12630-018-1074-6. PMID: 29368315.

8 Barengo JH, Yuen SN, Kennedy P, Shott SR. Subcutaneous emphysema with pneumomediastinum after tonsillectomy: Case report and review of the literature. Int J Pediatr Otorhinolaryngol. 2020;131:109885. doi: 10.1016/j.ijporl.2020.109885. PMID: 31981917.

9 Shaw RJ, Butterworth CJ, Silcocks P, Tesfaye BT, Bickerstaff M, Jackson R, et al. HOPON (Hyperbaric Oxygen for the Prevention of Osteoradionecrosis): a randomized controlled trial of hyperbaric oxygen to prevent osteoradionecrosis of the irradiated mandible after dentoalveolar surgery. Int J Radiat Oncol Biol Phys. 2019;104:530–9. doi: 10.1016/j.ijrobp.2019.02.044. PMID: 30851351.

10 Fleishman JA, Beck RW, Hoffman RO. Orbital emphysema as an ophthalmologic emergency. Ophthalmology. 1984;91(11):1389–91. doi: 10.1016/s0161-6420(84)34135-5. PMID: 6514309.

Conflicts of interest and funding: nil

Submitted: 24 May 2025
Accepted after revision: 5 July 2025

Hyperbaric oxygen treatment of neonates: a case series

Gizem Kavram[1], Beril Yasa[1], Meltem Bor[1], Leyla Bilgin[1], Elmas Zeynep Ince[1], Bengusu Mirasoglu[2], Emine Asuman Coban[1]

[1] *Department of Pediatrics, Division of Neonatology, Istanbul University Istanbul Medical Faculty, Istanbul, Turkiye*
[2] *Department of Underwater and Hyperbaric Medicine, Istanbul University Istanbul Medical Faculty, Istanbul, Turkiye*

Corresponding author: *Dr Gizem Kavram, Department of Pediatrics, Division of Neonatology, Istanbul University Istanbul Medical Faculty, Istanbul, Turkiye*
ORCiD: *0000-0002-2014-3223*
drgizemtosun@gmail.com

Keywords
Case reports; Hyperbaric medicine; Newborn; Thrombosis; Wound healing

Abstract

(Kavram G, Yasa B, Bor M, Bilgin L, Ince EZ, Mirasoglu B, Coban EA. Hyperbaric oxygen treatment of neonates: a case series. Diving and Hyperbaric Medicine. 2025 30 September;55(3):284–288. doi: 10.28920/dhm55.3.284-288. PMID:40986926.) Hyperbaric oxygen therapy (HBOT) administers oxygen under high pressure, mainly for decompression illness, carbon monoxide intoxication, wound healing, infections, and acute peripheral arterial ischaemia. There has been limited use in newborn infants. This case series aims to highlight the potential role of HBOT in management of rare and challenging conditions encountered in the neonatal period. Although HBOT is widely available, its application in newborns remains limited and not well established. We present three neonatal cases: acute peripheral ischaemia; vascular compromise due to thrombosis and compartment syndrome; and a non-healing surgical wound following omphalocele repair. We aim to emphasise the potential clinical benefit and discuss the safety profile of HBOT in select life or limb threatening neonatal pathologies. These cases demonstrate that HBOT, when use as an adjunctive therapy, may contribute to tissue salvage and overall improved outcomes in critically ill neonates. Our intention is to raise awareness and contribute to the limited literature regarding neonatal HBOT, particularly in contexts where usual treatment options are insufficient.

Introduction

Hyperbaric oxygen therapy (HBOT) is a medical treatment administered in a high-pressure environment by breathing 100% oxygen intermittently or continuously.[1] A minimum pressure of 203 kPa (2.0 atmospheres absolute [atm abs]) is recommended, which can be increased to a maximum of 284–304 kPa (2.8–3 atm abs).[2] Patients can be treated independently or as a group with other patients. The duration of treatment for most indications is 1.5–2 hours per session, usually to a maximum of four sessions per 24 hours. Courses of 20–60 treatment sessions are usually administered, depending on the indication.[3]

Both inspired oxygen and pressure potentially have therapeutic effects by enhancing gas distribution and oxygen transportation to ischaemic tissues. It induces hyperoxia independent of haemoglobin, stimulating anti-inflammatory cytokines, growth factors, and antioxidants. This way, HBOT promotes angiogenesis, disrupts bacterial respiration, and reduces inflammation by enhancing antioxidant formation and regulatory T-cell turnover.[4–6]

HBOT is predominantly used in adults for conditions like decompression illness, carbon monoxide poisoning, acute peripheral ischaemia, chronic wounds, and soft tissue infections.[7] Although its use in newborns is limited, there are previous case reports of treating non-healing wounds, necrotising enterocolitis, thrombosis, carbon monoxide poisoning, and hypoxic-ischaemic encephalopathy in which improvement seemed associated with application of HBOT.[8–13] Animal studies suggest neuroprotective effects in hypoxic encephalopathy, and reducing apoptosis and the impact of oxygen radicals within 72 hours post-damage.[14]

Use of HBOT in neonates and infants is an area where there is extremely limited experience and expertise worldwide. This small case series aims to present our experience using HBOT during the neonatal period. In all three cases, parents provided written informed consent for inclusion of their child's case history and images in this manuscript.

All three patients were treated in a multiplace chamber (Hipertech ZYRON 12, 2008) and a specially designed baby incubator was used for all (Figure 1). A continuous oxygen flow was provided in the incubator through a gas inlet and outlet. All treatments were accompanied by an inside attendant and a paediatric physician. Attendants intervened through the lid of the incubator when needed. Thermoregulation was provided by covering the baby with multilayer cotton sheets in the incubator, and if needed heated Mediflex® bags were placed under the sheets,

Figure 1
Adapted incubator for treating neonates (left) and in use in the hyperbaric chamber (right)

avoiding direct contact between the baby and the bags. Pacifiers were used during compression and decompression for enabling middle ear equalisation and patients were regularly examined by a paediatric specialist after each session for middle ear barotrauma. Tympanostomy tubes were not needed.

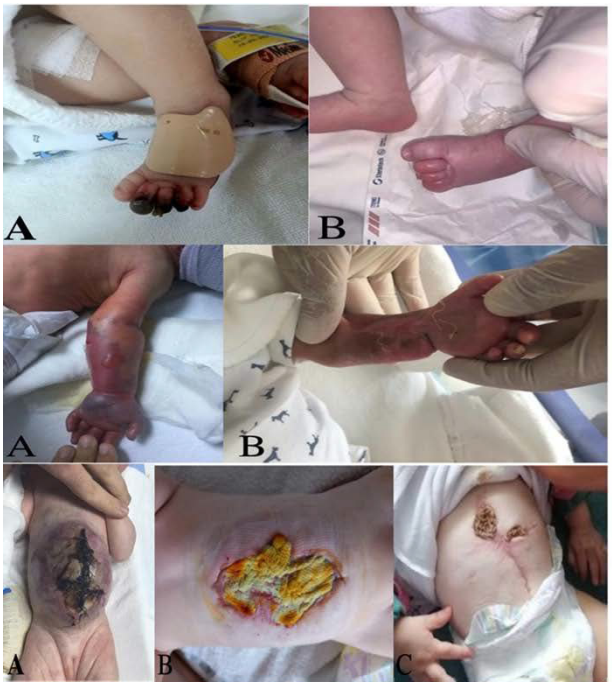
Case one

A 3,160 g girl, delivered by caesarean section at 36 weeks due to placenta previa, presented at day 21 of life with suspected peripheral arterial thrombosis involving toes on the left foot. She had three previous hospitalisations for jaundice and sepsis. Her mother and grandmother tested positive for COVID-19 infection one week before admission. Examination revealed bruising and necrosis on the left foot's second, third, and fourth toes (Figure 2). Arterial and venous Doppler ultrasound showed patent vessels and normal blood flow, ruling out current thrombosis. Metabolic and haematological investigations revealed normal homocysteine (7 $\mu mol \cdot L^{-1}$), vitamin B12 (579 $pg \cdot mL^{-1}$), and folate (12 $ng \cdot mL^{-1}$) levels. Lipid profile showed elevated triglycerides (200 $mg \cdot dL^{-1}$), low HDL (20 $mg \cdot L^{-1}$), normal LDL (47 $mg \cdot dL^{-1}$), and total cholesterol (107 $mg \cdot dL^{-1}$). Coagulation factors included factor 7 (65% of normal activity), factor 8 (224% of normal activity), factor 9 (78% of normal activity), factor 11 (40% of normal activity), and factor 12 (64% of normal activity). Protein C and S levels were normal. Factor V Leiden, methylenetetrahydrofolate reductase, prothrombin 20210 mutations, antiphospholipid and anticardiolipin antibodies were negative. Enoxaparin (two x 550 U subcutaneously) and pentoxifylline (intravenous, 30 $mg \cdot kg^{-1} \cdot day^{-1}$ as a six hour infusion per day, for five days) treatments were initiated. Plastic and reconstructive surgery recommended amputation, whereas underwater and hyperbaric medicine suggested HBOT. Despite a negative COVID-19 PCR test, IgG and IgM antibodies were positive. HBOT was administered twice daily for the first five days and then once daily for 21 sessions (243 kPa [2.4 atm abs], two hours each). The patient was discharged on the 37th day of life with complete healing without loss of extremity or function. Eye and hearing examinations yielded the expected results, and neurological evaluations detected no abnormalities (Table 1).

Case two

A 2,690 g girl born by elective caesarean section at 39 weeks gestation presented with a circulatory disorder in the left forearm on the first postnatal day. Physical examination revealed ecchymosis, bullous, exfoliative lesions, and a demarcation line on the left forearm (Figure 2). No radial pulse was detected in the left arm, and Moro and grasp reflexes were absent. Doppler ultrasound and CT angiography confirmed significant vascularisation loss in subclavian, axillary, and distal arteries and veins, with no blood flow observed in brachial, radial, and ulnar arteries and their branches. Sepsis could not be ruled out and antibiotic treatment with teicoplanin and cefotaxime was started. It was recommended to administer fresh frozen plasma in addition to heparin infusion, pentoxifylline infusion (30 $mg \cdot kg^{-1} \cdot day^{-1}$ infused for six hours per day, five days) and iliomedin treatments. The patient underwent HBOT three times a day for first week and twice a day for the following week. During the ongoing treatment, fasciotomy was decided upon due to a suspicion of compartment syndrome (10th day of life). Laboratory examinations to determine the cause of thrombosis were all within the normal range except for elevated protein S and factor 8 levels [homocysteine (7 $\mu mol \cdot L^{-1}$), vitamin B12 (579 $pg \cdot mL^{-1}$), folate (12.92

Figure 2
Photo montage of the three cases reported here



(Case 1).
A: Necrosis of the toes before treatment. **B:** Appearance of the toes one week after the treatment.

(Case 2).
A: Ecchymosis and bullae on the left forearm after birth.
B: Left arm healed with contracture of the wrist.

(Case 3).
A: Necrosis and circulation disorder around the 'mesh' on the 4th day of omphalocele surgery. **B:** Disappearance of necrotic tissue and regeneration at discharge.
C: Wound healing without flap surgery one year after discharge.

Table 1
Summary of neonatal cases treated with hyperbaric oxygen therapy in our clinic

Case	Indication	Birth week Age HBOT started	Dose/duration	Side effects	Result
One	Thrombosis	Week 36/40 21st day of life	243 kPa, two hours 21 sessions	Nil	Normal neurological exam at follow-up
Two	Thrombosis	Week 39/40 First day of life	243 kPa, two hours 28 sessions	Nil	Not followed up
Three	Wound	Week 36/40 34th day of life	243 kPa, two hours 15 sessions	Nil	Primary wound healing maintained at two years age

ng·mL^{-1}), protein C levels (47.7%), protein S levels (39.9%), Factor 8 (224.4%)]. Elevated factor 8 levels are associated with an increased risk of venous thromboembolism, such as deep vein thrombosis or pulmonary embolism. This is one of the most clinically significant implications of high factor 8 levels. Despite the development of mild contracture, the extremity was successfully preserved, and the patient was discharged on the 46th day with recommendations for physical therapy. Eye and hearing examinations yielded the expected results, and neurological evaluations detected no abnormalities (Table 1).

Case three

A 2,540 g girl, diagnosed with omphalocele antenatally was born by emergency caesarean section at 34 weeks gestation. The omphalocele sac, measuring 6 x 8 cm, was treated with wet sterile gauze upon delivery, and the baby was transferred to the neonatal intensive care unit. Surgery was performed to return the intestines to the abdomen and reposition the liver on the 5th and 21st day of life respectively, after which the abdomen was closed with a 'mesh'. Ampicillin and gentamicin antibiotic therapy which was started in the preoperative period was applied for six days and switched to vancomycin and meropenem due to an increase in acute phase reactants. Vancomycin-meropenem was completed at the fourteenth day of treatment. Micafungin was added on the sixth postoperative day (36th day of life) after the growth of *Candida* species was observed in the tracheal aspirate culture. The antifungal treatment was completed in 15 days. On the 32nd day of life (after the second operation), the patient was referred to underwater medicine due to skin loss around the incision line and compromised tissue circulation. Beginning at the 34th day of life, HBOT treatment was given eight hourly on the first day. It was planned to receive treatment twice a day for the next three days and once a day thereafter. HBOT was administered at (243 kPa [2.4 atm abs], two hours each). Fifteen sessions were administered

until the postnatal forty-fourth day. This was associated with regeneration of the area of skin loss around the incision line (Figure 2). Eye and hearing examinations yielded the expected results, and neurological evaluations detected no abnormalities. The patient was discharged on the 66th day of life (Table 1).

Discussion

Prior to these cases, in Turkey, a seven-day-old infant with carbon monoxide poisoning received HBOT, consisting of three 30-minute sessions daily at 243 kPa; however, one of the newborns included in our study was younger. Average neurological outcome was noted at discharge and six months of age.[12]

Thrombosis, while rare in neonates, poses a heightened risk in critically ill newborns, often leading to ischaemia-related limb loss.[14,15] HBOT has emerged as a promising intervention for neonatal extremity ischaemia due to thrombosis.[10,11] Wiebers et al. successfully administered HBOT together with thrombolysis and anticoagulation to a term male newborn with bilateral lower extremity thrombosis of undetermined aetiology, averting amputation in one extremity.[10] Similarly, HBOT in conjunction with heparin and fresh frozen plasma effectively treated a preterm newborn with purpura fulminans, yielding no adverse effects.[11] HBOT was administered for extremity ischaemia due to thrombosis in our cases one and two, both of which were discharged without any loss of extremity or motor function. While long-term outcomes were unreported, these infants remained fully recovered at the six-month follow-up.

Although peripheral arterial ischaemia is a common indication for HBOT in adults, there is limited literature on neonatal cases. The youngest known HBOT recipient for arterial gas embolism was a three-month-old baby who received HBOT for cerebral air embolism following a Glenn shunt operation for congenital heart disease, effectively resolving embolism. Despite bilateral tympanic membrane haemorrhage during treatment, the patient exhibited regular hearing examination at discharge.[16]

In another case report, a 17-month-old male patient with Noonan syndrome, idiopathic thrombocytopenic purpura, and bilateral undescended testicles developed haematoma and oedema in the scrotum and penis the day after bilateral orchiopexy and circumcision. Ischaemic appearances were observed on the penile and scrotal skin on the second postoperative day. Enoxaparin sodium and fresh frozen plasma were started on the recommendation of haematology. HBOT was initiated considering the possibility of tissue necrosis. A rapid healing was observed within five days. It was concluded that HBOT may be considered as an additional treatment option in patients with similar conditions.[17]

A retrospective study by Kangal et al. reported the outcomes and described difficulties encountered in infants 12 months old or younger undergoing HBOT; a rare patient population in this therapeutic intervention. Demographic data, clinical presentation, HBOT indication, chamber type, oxygen delivery method, total number of treatments, outcome and complications were extracted from clinical records. A total of 54 infants were included in the study. The patients' median age was 3.5 (range 0–12) months. The major HBOT indication was acute carbon monoxide intoxication ($n = 32$). A total of 275 HBOT treatments were administered, mostly performed in multiplace chambers ($n = 196$, 71%). Only one patient (2%) required mechanical ventilation. Acute signs were fully resolved in the most patients ($n = 40$, 74%). No complications related to HBOT were reported. In conclusion, it was suggested that HBOT may be a safe and effective treatment for infants.[18]

In another report, eight patients, six with hypoxic ischaemic encephalopathy and two with necrotising enterocolitis underwent HBOT at 203 kPa (2 atm abs). Neonatologists provided respiratory support during treatment. No adverse effects were observed in ophthalmological and central nervous systems. Neurodevelopmental examinations at three and six months post-treatment were standard for all patients. Long-term follow-up was conducted for two patients, both exhibiting normal neurological examinations at age five, with one case reporting mild attention deficit.[9]

HBOT is widely used for wound healing in adults but its use is limited in newborns.[7] HBOT has been successfully used in treating newborns after cardiac surgery and wound treatment due to necrosis of the glans penis.[13] In our case three, HBOT was administered to a patient with extensive necrosis post-omphalocele surgery. Following HBOT, reperfusion occurred in necrotic areas, demarcation lines formed, and wound healing was achieved without flap surgery.

In our experience, the limited use of HBOT in neonates is mainly due to concerns about the potential side effects associated with hyperoxia and challenges related to safe transportation, thermoregulation, and sedation during treatment. While term newborns possess relatively robust antioxidant systems, HBOT may pose risks, including respiratory morbidities and retinopathy, particularly for infants with a gestational age of less than 34 weeks and a birth weight under 1,200 g due to their immature antioxidant systems.[9]

HBOT side effects encompass barotrauma, visual auditory impairments, and potential oxygen toxicity to the central nervous system and lungs. While research on the neuroprotective effects of HBOT in adults has expanded, data on its long-term impacts on the developing nervous system in newborns are scarce.[19] None of our three patients experienced any side effects related to HBOT. Eye and hearing examinations yielded the expected (normal) results, and neurological evaluations detected no abnormalities.

Conclusions

HBOT shows promise for ischaemic and thrombotic events, but data on its use and risks in newborns are limited. Reporting long-term outcomes could improve its safety for challenging cases. In conclusion, HBOT can be a beneficial treatment option for difficult complications seen in neonates, including acute peripheral ischaemia, non-healing wounds and compromised flaps as in our case of omphalocele.

References

1 Singh S, Gambert SR. Hyperbaric oxygen therapy: a brief history and review of its benefits and indications for the older adult patient. Annnals of Long-Term Care. 2014;22(7-8):37–42.

2 Huang ET, editor. Undersea and Hyperbaric Medical Society hyperbaric medicine indications manual. 15th ed. West Palm Beach (FL): Best Publishing Company; 2023.

3 Thom SR. Hyperbaric oxygen: its mechanisms and efficacy. Plast Reconstr Surg. 2011;127(Suppl 1):131S–141S. doi: 10.1097/PRS.0b013e3181fbe2bf. PMID: 21200283. PMCID: PMC3058327.

4 Ortega MA, Fraile-Martinez O, García-Montero C, Callejon-Pelaez E, Saez MA, Alvarez-Mon M, et al. A general overview on the hyperbaric oxygen therapy: applications, mechanisms and translational opportunities. Medicina (Kaunas). 2021;57(9):864. doi: 10.3390/medicina57090864. PMID: 34577787. PMCID: PMC8465921.

5 Kocaman Ürütük F. Current approach to hyperbaric oxygen therapy. Istanbul Medical Journal. 2020;21(4):234–41. doi: 10.4274/imj.galenos.2020.89725.

6 Tejada S, Batle JM, Ferrer MD, Busquets-Cortes C, Monserrat-Mesquida M, Nabavi SM, et al. Therapeutic effects of hyperbaric oxygen in the process of wound healing. Curr Pharm Des. 2019;25:1682–93. doi: 10.2174/1381612825666190703162648. PMID: 31269879.

7 Mathieu D, Marroni A, Kot J. Tenth European Consensus Conference on Hyperbaric Medicine: recommendations for accepted and non-accepted clinical indications and practice of hyperbaric oxygen treatment. Diving Hyperb Med. 2017;47:24–32. doi: 10.28920/dhm47.1.24-32. PMID: 28357821. PMCID: PMC6147240.

8 Vazquez RL, Spahr RC. Hyperbaric oxygen use in neonates. A report of four patients. Am J Dis Child. 1990;144:1022–4. doi: 10.1001/archpedi.1990.02150330082027. PMID: 2396616.

9 Sánchez EC. Use of hyperbaric oxygenation in neonatal patients: a pilot study of 8 patients. Crit Care Nurs Q. 2013;36:280–9. doi: 10.1097/CNQ.0b013e318294e95b. PMID: 23736667.

10 Wiebers J, Purdy I, Lieber M, Milisavljevic V. Hyperbaric oxygen in treatment of neonatal arterial thromboembolism of lower extremities. J Perinatol. 2006;26:777–9. doi: 10.1038/sj.jp.7211606. PMID: 17122788.

11 Yılmaz A, Kaya N, Meriç R, Bayramli Z, Öroğlu B, Celkan TT et al. Use of hyperbaric oxygen therapy of purpura fulminans in an extremely low birth weight preterm: A case report. J Neonatal Perinatal Med. 2023;16:339–42. doi: 10.3233/NPM-200428. PMID: 32741781.

12 Turan C, Yurtseven A, Baki Beyter M, Saz EU. A 7-day-old male infant with carbon monoxide poisoning treated with hyperbaric oxygen. Pediatr Emerg Care. 2020;36:e360–e361. doi: 10.1097/PEC.0000000000001612. PMID: 30335685.

13 Polak N, Fishelev G, Lang E, Wang Z, Neheman A, Ben Haim Y, et al. Hyperbaric oxygen as salvage therapy for neonates suffering from critical ischemia of the glans penis after circumcision. Urology. 2021;149:e48–e51. doi: 10.1016/j.urology.2020.09.006. PMID: 32956687.

14 Zhu M, Lu M, Li QJ, Zhang Z, Wu ZZ, Li J, et al. Hyperbaric oxygen suppresses hypoxic-ischemic brain damage in newborn rats. J Child Neurol. 2015;30:75–82. doi: 10.1177/0883073814530500. PMID: 24762865.

15 Makatsariya A, Bitsadze V, Khizroeva J, Vorobev A, Makatsariya N, Egorova E, et al. Neonatal thrombosis. J Matern Fetal Neonatal Med. 2022;35:1169–77. doi: 10.1080/14767058.2020.1743668. PMID: 32204642.

16 LeDez KM, Zbitnew G. Hyperbaric treatment of cerebral air embolism in an infant with cyanotic congenital heart disease. Can J Anaesth. 2005;52:403–8. doi: 10.1007/BF03016284. PMID: 15814756.

17 Dereli D, Çakiroğlu S, Köse AA, Tokar B. Hyperbaric oxygen treatment in bilateral orchiopexy and post-circumcision haematoma in a thrombocytopenic patient with Noonan syndrome. Diving Hyperb Med. 2024;54:133–6. doi: 10.28920/dhm54.2.133-136. PMID: 38870956. PMCID: PMC11444912.

18 Ozgok Kangal K, Mirasoglu B. Hyperbaric oxygen treatment for infants: retrospective analysis of 54 patients treated in two tertiary care centres. Diving Hyperb Med. 2024;54:9–15. doi: 10.28920/dhm54.1.9-15. PMID: 38507905. PMCID: PMC11227964.

19 Ahmadi F, Khalatbary AR. A review on the neuroprotective effects of hyperbaric oxygen therapy. Med Gas Res. 2021;11:72–82. doi: 10.4103/2045-9912.311498. PMID: 33818447. PMCID: PMC8130666.

Conflicts of interest and funding: nil

Submitted: 2 October 2024
Accepted after revision: 19 July 2025

Localised tissue necrosis from marine puncture injury: first aid, definitive management and review of the literature

Deon A Viljoen[1], Jessica Andrews[2]

[1] *Occupational Underwater Medicine, NSW, Australia*
[2] *Molecular Biotechnology, Joondalup, WA, Australia*

Corresponding author: *Dr Deon Viljoen, Occupational Underwater Medicine, 39A Pell Street, Merewether, NSW, 2291, Australia*
ORCiD: *0009-0000-0124-6350*
deonvil20@gmail.com

Keywords
Case reports; First aid; Marine envenomation; Microbiology; Necrotising infections; Risk management; Surgery

Abstract

(Viljoen DA, Andrews J. Localised tissue necrosis from marine puncture injury: first aid, definitive management and review of the literature. Diving and Hyperbaric Medicine. 2025 30 September;55(3):289–293. doi: 10.28920/dhm55.3.289-293. PMID: 40986927.)
We discuss a case of tropical marine envenomation from an unidentified marine creature via a penetrating injury to the left ring finger resulting in localised tissue necrosis. We present an evidence-based review of marine puncture wounds including first aid, hot water immersion therapy, antibiotic selection and definitive management.

Introduction

Infections arising from soft tissue puncture wounds in marine environments can be difficult to manage and may result in substantial tissue injury, particularly where the injury involves cytotoxic envenomation from a marine creature. We report a case of a puncture wound suspected to arise from a marine creature interaction with probable cytotoxic venom introduction and discuss some of the important clinical principles it illustrates.

Case report

The patient gave written consent for reporting of their case and images.

A 56-year-old female diver was undertaking an underwater photography dive at Ambon Harbour, Indonesia. At a depth of 10 metres sea water, when reaching backwards with her left hand to assist with stability, she experienced immediate and intense pain in her left ring finger following the delivery of a presumed cytotoxic venom into the pulp space and volar aspect of the distal phalanx of her left ring finger. She was unable to identify the source of injury.

Within 10 minutes, and back on the boat, the finger was immersed into fresh hot water, as hot as could be tolerated in the absence of a thermometer, while the boat returned to land. Hot water was regularly added throughout the 30-minute boat trip to maintain heat. Upon land, the injury was re-immersed into hot water, again maintained by regular input of hot water for a further 60 minutes. To manage pain, the diver was given panadeine/codeine and the finger was cleaned and irrigated with fresh water before being coated in Kenacomb ointment and dressed with a bandage. Figure 1 demonstrates the injury at day one.

Cephalexin 500 mg was administered every 12 hours for five days and the patient was monitored for systemic symptoms. Within 36 hours, the pain had subsided, and the diver chose to remain on site and continue with the underwater photography expedition. The left hand was kept protected by a surgical glove and the injury remained localised to the pulp space and volar aspect of the distal and middle phalanges of the left ring finger.

At day five, the diver flew home where she consulted with a general practitioner and was referred on to a surgeon. An area of necrosis was observed within the pulp space on day eight and by day nine the necrosis had spread into the volar aspect of the middle phalanx of the affected finger. Figure 2 demonstrates the progression of tissue necrosis from day eight to day nine.

A surgical debridement was performed by the surgeon on day nine. A detailed description of the surgery and the amount of necrotic tissue is not available. The finger was allowed to heal for four months without further surgical intervention. Figure 3 shows the injury site shortly after surgical debridement. By day 112 the finger had fully healed with full functionality restored, however minor scarring and mild loss of sensation over the volar aspect of the distal

Figure 1
Image taken on Day one of the patient's left ring finger following the delivery of cytotoxic venom from an unidentified marine creature into the pulp space and volar aspect of the distal phalanx

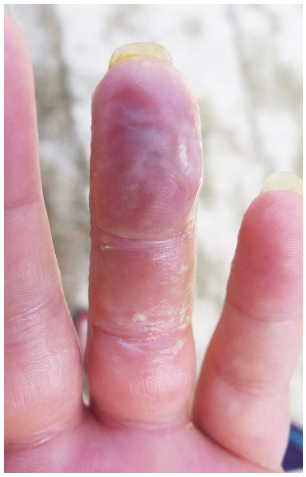


Figure 2
Image taken on Day eight (left) and image taken on Day nine (right) to demonstrate the spread of tissue necrosis over a 24 hour period

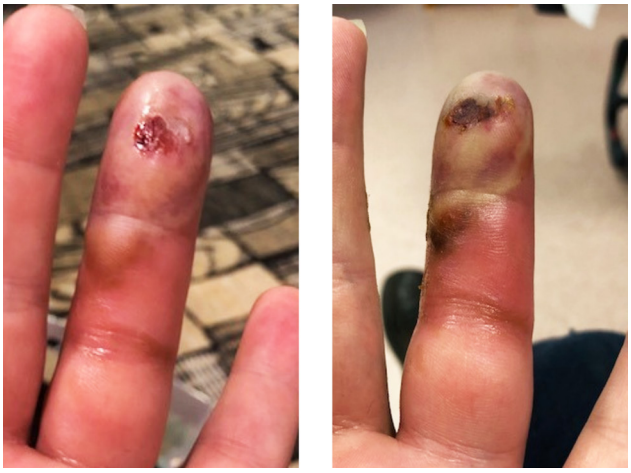


Figure 3
Image taken on Day 13 showing the injury site shortly after surgical debridement

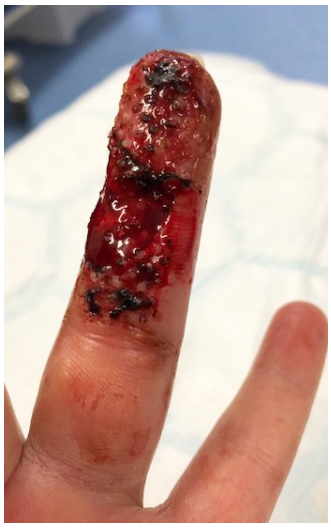
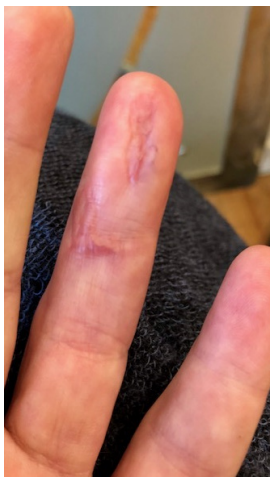


Figure 4
Image taken on Day 112 after the injury was allowed to heal without further intervention; full functionality was restored, however minor scarring and mild loss of sensation over the volar aspect of the distal phalanx was reported



phalanx was reported. Figure 4 shows the affected finger at day 112 after the wound was allowed to heal without further intervention.

Discussion

Many marine creatures, particularly in tropical waters, can cause human envenomation.[1] It is reasonable to expect the prevalence of marine envenomation may increase with the possible rising sea temperatures due to climate change, and the growing popularity of marine recreational activities.[2] It is therefore important that clear advice about risk-mitigation strategies[3] and appropriate first aid techniques are documented.

RELEVANT MARINE CREATURES IN INDONESIA

Indonesia's rich marine biodiversity includes numerous species capable of causing puncture envenomation to humans, posing significant medical concerns for coastal communities and divers. Notably, venomous fish such as stonefish (*Synanceia spp.*) and lionfish (*Pterois spp.*) possess dorsal spines that can deliver potent neurotoxins, resulting in intense pain, swelling, and in severe cases, systemic effects. Scorpionfish (Scorpaenidae) and rabbitfish (Siganidae) are similarly hazardous, with venomous spines capable of inflicting deep puncture wounds. Additionally, stingrays common in Indonesian waters have barbed tails containing venom glands, which can cause lacerating injuries

complicated by toxin-induced tissue necrosis and secondary infections. Other marine creatures of concern include sea urchins, whose sharp spines may break off in the skin, introducing venom and foreign material, and certain species of catfish, which have venomous fin spines. Together, these marine hazards underscore the need for local awareness, prompt wound management, and appropriate medical treatment to mitigate the risk of severe envenomation in Indonesia's tropical marine environments.

FIRST AID STRATEGIES

When possible, positive identification of the marine organism can assist downstream medical care considering the risk of systemic reactions. Hot water immersion (HWI) is widely recommended as first aid for puncture envenomations from marine creatures such as stonefish, stingrays, and catfish; however, its efficacy and exact mechanism of action remain subjects of ongoing debate.[4,5] The prevailing theory suggests that the application of hot (but non-scalding) water, typically at temperatures between 40°C and 45°C, leads to thermal denaturation of heat-labile venom proteins, thereby reducing their toxic effects. Nonetheless, emerging evidence indicates that the primary benefit may instead stem from heat-induced modulation of nociceptors and pain pathways in the peripheral and central nervous systems, resulting in transient analgesia rather than direct venom inactivation.[4] Clinical reports and small studies support the analgesic effect of HWI, yet robust randomised trials remain limited, and optimal treatment parameters, including the ideal temperature and duration, are not clearly standardised.[1,4,5] Generally, immersion for 30 to 90 minutes or until significant pain relief is achieved is advised, but risks such as thermal burns must be carefully managed.[6,7] Consequently, while HWI remains a practical and low-cost intervention for marine stings, further research is needed to clarify its true mechanism, refine protocols, and confirm its effectiveness across different venomous species. The importance of clear advice for patients who might continue HWI therapy unsupervised at home has been stressed.[6] To mitigate the risk of iatrogenic thermal injury water temperatures should be limited to 40–45°C, or as hot as the patient can stand with a non-injured limb, and exposure time should preferentially be limited to 30-minute intervals with 30 minutes deferrals, or otherwise no more than 90 minutes of continuous immersion.[6]

Following HWI therapy for pain relief and toxin denaturation, the injury should be thoroughly irrigated with fresh water and inspected for foreign particles under sterile conditions by a medical practitioner. The patient should be monitored for systemic symptoms that may require cardio-respiratory support.[8] It is important to inform the patient about the possible symptoms caused by a retained foreign body that may require urgent medical attention such as surgical removal and targeted antibiotics.[9] These include:

- Persistent localised pain: ongoing or worsening pain at the wound site that does not resolve with expected healing time;
- Chronic swelling: localised edema that persists or worsens despite initial wound management;
- Palpable lump or mass: a firm nodule or foreign body that can sometimes be felt under the skin;
- Sinus tract formation: development of a draining sinus or non-healing wound with intermittent discharge;
- Non-healing ulcer: delayed or failed wound closure, often with granulation tissue that fails to epithelialise;
- Recurrent or persistent infection: recurrent cellulitis or abscess formation at the site, despite adequate antibiotic therapy;
- Chronic inflammation or granuloma: foreign body reaction leading to a localised inflammatory mass or granulomatous tissue;
- Restricted movement: if near a joint or tendon, the retained fragment may cause pain with movement or reduced range of motion; and
- Neuropathic symptoms: paraesthesia or localised numbness if the retained object impinges on a nerve.

The requirement of sutures for open wounds should be determined by the attending physician's risk assessment.[10]

The use of prophylactic antibiotics for marine sting envenomation remains a debated aspect of management, as not all puncture wounds require antimicrobial therapy. Current evidence suggests that prophylactic antibiotics may be indicated for high-risk wounds, including deep punctures (e.g., from stingrays or stonefish spines), wounds with retained foreign bodies or devitalised tissue, injuries in immunocompromised hosts, and wounds in anatomically high-risk sites such as the hands or feet. Prophylactic regimens should provide broad coverage for common marine pathogens, including *Staphylococcus aureus*, *Streptococcus* spp. and Gram-negative organisms such as *Vibrio* spp.[10,11] Single-agent options include doxycycline (effective against *Vibrio* spp. and some MRSA strains), or fluoroquinolones such as ciprofloxacin or levofloxacin.[10,11] Alternatively, combination therapy may be warranted for broader coverage: for example, doxycycline plus a third-generation cephalosporin (e.g., ceftazidime) or doxycycline plus trimethoprim-sulfamethoxazole for MRSA plus *Vibrio* risk.[10] Once signs of established infection are present, such as worsening pain, cellulitis, purulent discharge, or systemic signs, empiric therapy should cover the same spectrum. Oral options for mild infections include doxycycline, trimethoprim-sulfamethoxazole plus a beta-lactam (e.g., amoxicillin-clavulanate), or fluoroquinolones. For severe or rapidly progressive infections, especially in suspected *Vibrio vulnificus* cases, intravenous agents such as doxycycline plus ceftazidime, or a carbapenem, may be required. Tetanus prophylaxis should also be ensured, and surgical consultation considered for retained spines or necrotic tissue.[8] Ultimately, antibiotic choice should be guided by local resistance patterns and individual patient

factors, with early microbiological cultures recommended when possible. Sterile wound care and regular inspection should be maintained until reasonable healing is confirmed, since tissue necrosis is a possible complication as seen in our case report. In this case prompt surgical debridement was effective in the healing process of the affected appendage and only mild scarring and loss of sensation resulted.

In remote settings where definitive medical care is limited, timely evacuation must be considered for marine envenomation injuries that present with clinical features indicating significant local or systemic complications. Stonefish stings, for example, are notorious for excruciating pain and the potential for progressive tissue necrosis, secondary bacterial infection, or cardiovascular compromise due to venom effects; evacuation is warranted if pain is unrelieved by first aid measures such as hot water immersion, or if antivenom administration is indicated but unavailable. Rapidly progressing cellulitis, myositis, or necrotic changes around the puncture wound, particularly when associated with highly virulent marine pathogens such as *Vibrio vulnificus* also demand urgent transfer to a facility capable of providing intravenous antibiotics, surgical debridement, and advanced wound care. Systemic signs and symptoms of sepsis, including fever, hypotension, tachycardia, or altered mental status, should prompt immediate evacuation due to the risk of septic shock and multi-organ failure. Additionally, the development of compartment syndrome, severe uncontrollable pain, neurovascular compromise, or signs of deep-space hand infections such as tenosynovitis or septic arthritis require surgical intervention not feasible in austere environments. In all cases, early recognition and prompt evacuation can be lifesaving, highlighting the importance of risk assessment and structured evacuation protocols for divers, fishermen, and coastal communities operating far from advanced medical services.

Surgical consultation should be promptly sought in cases of marine sting envenomation when there is clinical suspicion of retained foreign material, progressive soft tissue infection, or complications requiring operative management. Injuries from creatures such as stingrays, stonefish, and sea urchins often involve barbed spines or spicules that can fragment and remain embedded in the wound, serving as a nidus for persistent inflammation, granuloma formation, or secondary infection. If retained spines are suspected, evidenced by persistent pain, a palpable foreign body, or failure of the wound to heal, imaging (such as plain radiographs or ultrasound) is indicated, and surgical exploration may be necessary to remove the foreign material. Surgical debridement is also indicated when there is rapidly spreading cellulitis unresponsive to initial antibiotic therapy, deep space infections such as tenosynovitis or septic arthritis, abscess formation, or signs of necrotising soft tissue infection, particularly when marine pathogens such as *Vibrio vulnificus* are involved. Additionally, signs of compartment syndrome, including severe pain out of proportion to examination, tense swelling, or neurovascular compromise, necessitate urgent fasciotomy. Early surgical input can reduce the risk of chronic complications, such as functional impairment, and improve outcomes by ensuring adequate wound care, debridement of necrotic tissue, and appropriate drainage of purulent collections when indicated.

PREVENTION AND RISK MITIGATION

Protective swimwear that covers all or most of the skin, including gloves, maintaining buoyancy, avoiding contact with the seabed and marine life wherever possible, and staying vigilant about your surroundings are recommended strategies to avoid marine envenomation.[3] Restrictive bands such as rings on fingers should also be removed before entering the sea as they will pose a further risk in the event of marine envenomation and resultant swelling of fingers.

Conclusions

Divers should take the necessary precautions before entering the ocean and remain vigilant whilst in the water. In the event of marine envenomation, appropriate application of hot water immersion therapy to the affected area is recommended to improve clinical outcomes. Medical attention should be sought as soon as practical to allow the injury to be cleaned and inspected, and the patient may be prescribed prophylactic antibiotics particularly targeting gram-negative bacterial species. The patient should be monitored for systemic symptoms, and the affected area should be cleaned and inspected regularly until satisfactory healing can be confirmed and the possibility of tissue necrosis can be ruled out or managed. Surgical debridement of necrotic tissue should be performed when indicated.

References

1 Krzyżak J, Korzeniewski K. Marine creatures dangerous for divers in tropical waters. Int Marit Health. 2021;72(4):283–92. doi: 10.5603/IMH.2021.0052. PMID: 35146740.

2 Cisneros-Montemayor AM, Sumaila RU. A global estimate of benefits from ecosystem-based marine recreation: potential impacts and implications for management. J Bioecon. 2010;12:245–68. doi: 10.1007/s10818-010-9092-7.

3 Todd J, Edsell M. A diver's guide to subaquatic envenomation in the Mediteranean. Diving Hyperb Med. 2019;49:225–8. doi: 10.28920/dhm49.3.225-228. PMID: 31523798. PMCID: PMC6881212.

4 Atkinson PRT, Boyle A, Hartin D, McAuley D. Is hot water immersion an effective treatment for marine envenomation? Emerg Med J. 2006;23:503–8. doi: 10.1136/emj.2005.028456. PMID: 16794088. PMCID: PMC2579537.

5 Wilcox CL, Yanagihara AA. Heated debates: Hot-water immersion or ice packs as first aid for cnidarian envenomations? Toxins (Basel). 2016;8(4):97. doi: 10.3390/toxins8040097. PMID: 27043628. PMCID: PMC4848624.

6 Lewis CJ, Wood F, Goodwin-Walters A. Iatrogenic thermal burns secondary to marine sting treatment. J Burn Care Res. 2020;41:878–81. doi: 10.1093/jbcr/iraa042. PMID: 32141503.

7 Abdul Jalil KI, Qayyum MT. Iatrogenic thermal burn after

hot water immersion for weever fish sting treatment: a case report. Scars Burn Heal. 2020;14:6:2059513120944045. doi: 10.1177/2059513120944045. PMID: 32974056. PMCID: PMC7493226.

8 Aubin A, Chee P, editors. Marine wounds and stings [Internet]. New South Wales, Australia: DermNet; 2023. [cited 2023 Dec 17]. Available from: https://dermnetnz.org/topics/marine-wounds-and-stings.

9 Levine MR, Gorman SM, Young CF, Courtney DM. Clinical characteristics and management of wound foreign bodies in the ED. Am J Emerg Med.2008;26:918–22. doi: 10.1016/j.ajem.2007.11.026. PMID: 18926353.

10 Briotti J, Jayamaha JY, Keogh A. Marine penetrating injury to the shoulder of uncertain origin. Wilderness Environ Med. 2021;32:235–9. doi: 10.1016/j.wem.2021.01.012. PMID: 33839016.

11 Cebrián-López J, Jover-Díaz F, Infante-Urrios A, Piqueras-Vidal PM, Ortiz de la Tabla-Duccasse V. Post-traumatic wound infection after diving caused by Vibrio alginolyticus: a case report. Diving Hyperb Med. 2025;55:199–202. doi: 10.28920/dhm55.2.199-202. PMID: 40544150.

Conflicts of interest and funding: nil

Submitted: 27 February 2024
Accepted after revision: 19 July 2025

Obituary

Professor Des Gorman

Des Gorman, one of our field's most iconic figures, passed away on 2 July 2025 at age 71. Des was well known to SPUMS members, particularly those who were around in the nineties and 2000s when he was one of the world's most influential figures in diving medicine. He served as SPUMS president for a substantial period in this era.

Des was a kiwi of Māori descent (Ngāti Kuri and Ngāpuhi) who grew up in modest circumstances in Auckland. He was possessed of an extraordinary star-bright intellect and graduated from medical school at Auckland with multiple prizes and awards. Like many of us, he had been a passionate diver at a young age and this drove his early career decisions in the direction of diving and hyperbaric medicine. After a period in the Australian Navy (Des's father was Australian) he became director of the hyperbaric medicine unit at Royal Adelaide Hospital (RAH). Largely on the back of momentum created by Des's ground-breaking PhD research into arterial gas embolism pathophysiology, the RAH unit became the region's premiere academic centre for our discipline. Other themes in Des's academic and clinical careers, such as a research interest in carbon monoxide poisoning (later the subject of his MD thesis) and clinical occupational medicine, emerged during this period.

Des moved back to New Zealand at the beginning of the 1990s to become director of the new Slark Hyperbaric Unit (SHU) at the Royal New Zealand Navy Hospital. Just as he had done at the RAH, Des palpably energized the environment at the SHU, attracting new medical officers such as Alison Drewry, Anthony Holley and myself, and motivating incumbents such as James Woodfine, Chris Strack, John Monash, Courtenay Kenny and John Monighatti, all of whom made academic contributions to the field or trained in occupational medicine under Des's guidance. It was a vibrant, influential period in the unit's life. Des attracted many overseas visitors from the Australian and Omani militaries, all of whom spent six months to a year learning from him. Des had a particularly strong relationship with the Royal Navy of Oman and he remains a revered figure among many of their now senior medical officers.

Des transitioned from the Navy to academia in the late 2000s taking up a personal chair in Occupational Medicine at the University of Auckland. He, together with Professor Mike Davis, founded a successful diploma course in diving and hyperbaric medicine through the university. From his academic platform Des became increasingly involved in national health strategy formulation and management. He had a leadership role in the Health Workforce New Zealand group and was a strong advocate for innovation and equity in New Zealand healthcare. He became something of a household name for his thought-provoking commentaries on various healthcare issues, particularly during the COVID 19 pandemic.

I wish to invoke writer's privilege in providing a somewhat personal perspective on my journey with Des. I had never contemplated undertaking a PhD as part of my academic career prior to meeting him. However, I was massively inspired by his ability to speak so authoritatively on topics I was passionate about. Des convinced me that the basis for this was knowledge through original research and that the path to my goal of emulating him was to do a PhD as he had done. I followed his advice, under his supervision. I would like to think I was a vaguely competent student, but one place I needed help was writing. Of all the things Des taught me, writing scientific prose was the most important. Manuscript after manuscript got 'Gormanized' (covered in red ink) but it was educational gold. Des inspired and guided my early academic career and I am deeply grateful for that.

Of the many possible anecdotes, my most enduring professional memory of Des was the 2004 'remote decompression sickness' workshop in Sydney where David Doolette and I were trying to facilitate a sensible international consensus on what to do with mild or equivocal symptoms of DCS in remote locations. This required a substantial shift in entrenched dogma to establish a new practice paradigm. There was a real possibility of failing to reach consensus. Luckily, Des chaired the exhausting half day consensus debate in what proved to be an absolute masterclass of decisive chairmanship, thinking on your feet, and his particular brand of irresistible clinical logic. The consensus was established, and the resulting practice change has proved robust over 20 subsequent years. This was arguably the most consequential contribution to diving medicine I have been involved in, and without Des it likely would not have happened.

Des leaves behind his wonderful family; wife Christine, daughters Anna, Sarah, and Emily, and eight grandchildren. On behalf of SPUMS I offer them our sincere condolences, and our thanks for sharing Des with us over so many years and achievements. Kia Kaha.

Professor Simon J Mitchell
Editor, Diving and Hyperbaric Medicine

SPUMS
South Pacific Underwater Medicine Society

Notices and news

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

SPUMS President
Neil Banham

It is with sadness that I report the recent passing of Des Gorman, a previous SPUMS President and a long-time member of ExCom. Des was awarded his SPUMS Diploma (DipDHM) in 1989 and was President for two terms from 1990–1995. On behalf of SPUMS, our condolences to his family, friends and colleagues. An Obituary for Des appears in this issue.

I have just returned from the 18th Annual Scientific Meeting (ASM) of the Asian Hyperbaric and Diving Medicine Association (AHDMA) which was held in Da Nang, Vietnam. The ASM was co-hosted by the Vietnam Maritime Health Association (VINAMAHA), had over 200 delegates and was a great success. This year is AHDMA 20th anniversary, being established in 2005.

The conference was notable in that current or past Presidents of the major diving and hyperbaric medicine societies were represented, including myself (SPUMS), Enoch Huang (UHMS), Jacek Kot (EUBS), Andrew Ng (AHDMA), Professor Nguyen Truong Son (VINAMAHA), Tarun Sahni (Hyperbaric Society of India) as well as Francois Burman representing Divers Alert Network (DAN).

I am very grateful to Dr Yim Shao En, Andrew Ng, Prof Son and their team for an excellent conference and their superb hospitality.

The SPUMS 54th ASM in 2026 will be in Palau, Micronesia at the Palasia Hotel. Our ASM is being convened by Doug Falconer and Ian Gawthrope. There will be a new moon bump head parrotfish spawning event on the 13th/14th May – an enormous spectacle and not to be missed!

SPUMS 54th Annual Scientific Meeting
Palasia Hotel, Palau
10–15 May 2026
Theme: *Free diving*

Qantas are currently flying to Palau, departing Brisbane on Saturday mornings and returning Sunday morning. As of early August, 75 seats had been sold to SPUMS members for this flight. Registration, then booking of accommodation, diving and a post conference liveaboard diving trip (via Diveplanit) are now available on the SPUMS website.

The updated SPUMS Medical (6th edition 2025) is now available on the SPUMS website South Pacific Underwater Medicine Society - SPUMS-Full Medical.

David Smart and I have revised the document as a whole, with Lizzie Elliott driving the Paediatric diving section (new Appendix D) and Drew Heffernan and Cathy Meehan updating Diabetes and Diving.

The ANZHMG Introductory Course in Diving and Hyperbaric Medicine will next be held from mid-February 2026, again in Fremantle. The 2026 course is full with a waitlist, so I strongly suggest that you register your interest early for 2027 if you are considering attending. https://spums.au/index.php/education/spums-approved-courses-for-doctors.

Scholarships for trainees to attend this course are available thanks to the generosity of the Australasian Diving Safety Foundation (ADSF). Please contact John Lippmann at johnl@adsf.org.au for more information. ADSF has also kindly sponsored SPUMS membership for a year for course participants.

Data entry into the Australasian Decompression Illness (DCI) Registry has now been active for more than a year (from 1st July 2024). Almost all Australasian hyperbaric facilities are currently participating, with the remainder hopefully completing the bureaucracy to participate soon. The Registry is hosted by Monash University and generously funded by ADSF and collects data on all divers treated for DCI. In the near future, data will be available for research purposes. This data set will be a useful resource for those seeking to complete their SPUMS Diploma thesis.

Finally, I would like to thank members of ExCom for their hard work and support, as well as Nicky Telles our Editorial Manager and Web Assistant for her tireless efforts.

Dr Neil Banham
President, SPUMS

Clinical Professor David Smart, AM. Biography

Professor David Smart AM, announced his retirement from clinical practice this year. A previous biography had a number of minor errors, which have now been corrected.

Key highlights

Emergency medicine specialist, and specialist in diving and hyperbaric medicine, past president and life member, South Pacific Underwater Medicine Society.

Leader in Diving and Hyperbaric Medicine in Australia, the South Pacific and Internationally, working in the field since 1985, with a continuous commitment to diving safety and education for over 30 years.

Leader in the establishment of Diving and Hyperbaric Medicine Specialist training with the Australian and New Zealand College of Anaesthetists – Diploma of Advanced Diving and Hyperbaric Medicine.

Published over 80 peer reviewed papers in peer reviewed journals.

Awarded Member of the Order of Australia (AM) for services to diving and hyperbaric medicine and professional societies.

Major awards

1984 AMA Prize for the top graduate – University of Tasmania School of Medicine
1990 Buchanan Prize winner, Australasian College for Emergency Medicine. Top graduate October Fellowship Exams, Australasia.
1999 John Gilroy Potts Award – Australasian College for Emergency Medicine
Best scientific paper in World Literature by a College Fellow
2003 Foundation 20 Medal – Australasian College for Emergency Medicine – contribution to the development of Emergency Medicine during its first 20 years in Australasia
2003 National Finalist Australian Healthcare Association Baxter Healthcare Innovation Awards. Improving Health Outcomes for Tasmania's Aquaculture Industry Divers
2004 Order of the International Federation of Emergency Medicine, and Fellowship, for contribution to the development of Emergency Medicine Internationally
2005 Undersea and Hyperbaric Medicine Society USA Craig Hoffman Award for contribution to Diving Safety in Tasmania, Australia.
2013 Derek Craig Award for research and contribution to professional diving safety. Australian Diver Accreditation Scheme.
2013 Australian Diver Accreditation Scheme
2013 Undersea and Hyperbaric Medicine Society USA: Awarded Fellowship of Undersea and Hyperbaric Medicine. Awarded for international contribution and acknowledgement of achievement and high standards of expertise and practice to the field of Undersea and Hyperbaric Medicine
2015 Undersea and Hyperbaric Medicine Society USA. Award for excellence in Commercial Diving. Outstanding contributions to the commercial diving industry in the area of increased productivity or performance of the working diver. Practical application of biomedical knowledge and science to the solution of problems encountered in diving operations.
2015 Australasian College for Emergency Medicine International Development Grant Award. Establishment of sustainable Emergency Life Support and Serious Illness in remote Environments Courses in Fiji.
2019 Member of the Order of Australia (AM) for services to diving and hyperbaric medicine and professional societies.
2025 Life Membership, South Pacific Underwater Medicine Society.

Qualifications

Bachelor of Medical Science, University of Tasmania.
1984 Bachelor of Medicine and Bachelor of Surgery (Hons-1), University of Tasmania.
1989 Diploma of Diving and Hyperbaric Medicine, South Pacific Underwater Medicine Society.
1991 Fellow of the Australasian College for Emergency Medicine.
1995 Fellow of the Australasian College of Tropical Medicine.
2000 Fellow, Australian Institute of Company Directors.
2003 Certificate in Diving and Hyperbaric Medicine Australian and New Zealand College of Anaesthetists.
2005 Doctor of Medicine, University of Tasmania.
2017 Diploma of Advanced Diving and Hyperbaric Medicine, Australian and New Zealand College of Anaesthetists.

Emergency Medicine career

1991–2025 Fellow Australasian College for Emergency Medicine.
1991 Staff specialist emergency medicine, The Queen Elizabeth Hospital Adelaide, South Australia.
1992–1993 Emergency Medicine Fellow, Fremantle and Rockingham-Kwinana Hospitals, Western Australia.
1994–1998 Director of Emergency Medicine Royal Hobart Hospital, Tasmania.
1994–1997 Consultant to Statewide Aeromedical Retrieval Service, Tasmania.
1994–2006 Member Court of Examiners Australasian College for Emergency Medicine.
1994–1997 State Censor for Tasmania, Australasian College for Emergency Medicine.
1996–2014 Director of Emergency Medicine, Calvary Private Hospital Hobart.
1998–2003 Chair, Scientific Committee Australasian College for Emergency Medicine.

1995–1997 Supervisor of Postgraduate Medical Training, Southern Tasmania. Tasmanian Postgraduate Medical Institute.
1997–2003 Councillor for Tasmania, Australasian College for Emergency Medicine.
2001 Convener, 18th Annual Scientific Meeting of the Australasian College for Emergency Medicine, incorporating the 22nd Annual Scientific Meeting of the Australasian Society for Emergency Medicine *Emergency Medicine in the Market Place*. September 30 – October 4.
2004 Scientific Convener, 10th International Conference on Emergency Medicine, Cairns Australia. June 6–10 (Winner of national meeting of the year awarded by Meetings and Events Australia).

Diving and hyperbaric medicine career

1985–88 Medical hyperbaric chamber attendant Royal Hobart Hospital.
1989 Hyperbaric on call doctor, Royal Hobart Hospital
1992–1993 Hyperbaric Fellow, Fremantle Hospital Western Australia.
1994–1997 On call consultant Department of Diving and Hyperbaric Medicine, Royal Hobart Hospital.
1998–2021 Medical Director, Department of Diving and Hyperbaric Medicine, Royal Hobart Hospital.
2003–2023 Supervisor of Diving and Hyperbaric Medicine Training, Royal Hobart Hospital.
2003–2023 Examiner in Diving and Hyperbaric Medicine, Australian and New Zealand College of Anaesthetists.
2017–2023 Chief Examiner in Diving and Hyperbaric Medicine, Australian and New Zealand College of Anaesthetists.
1996–2025 Consultant to State, National and International Diving Industries on medical and occupational safety in diving, and recreational diving safety.

Over 30 registrars trained in Diving and Hyperbaric Medicine over past 20 years, 14 achieved Dip DHM and 11 achieved DipAdvDHM (ANZCA)

SPUMS roles

1999–2025 Executive Member SPUMS
Australian Standards Representative
2001–2014 Chairman Australian and New Zealand Hyperbaric Medicine Group
2002–2013 SPUMS Education Officer and Censor
2014–2020 SPUMS President
2020–2025 SPUMS Immediate Past president
2008–2020 Diving and Hyperbaric Medicine Journal Academic Board
2020–2025 Diving and Hyperbaric Medicine Journal Governance committee

SPUMS convener roles

2009 Diving, Flying and Space exploration. Future Synergies in Diving Accident Management. 38th Annual Scientific Meeting of the South Pacific Underwater Medicine Society. Iririki Island Vanuatu.
2023 Diver health and ocean health amid the storm clouds of climate change. 51st Annual Scientific Meeting of the South Pacific Underwater Medicine Society. Cairns Australia.
2024 A plunge into recreational diving and diver health. 52nd Annual Scientific Meeting of the South Pacific Underwater Medicine Society. Pacific Harbour, Fiji.

Contributions to diving safety in Tasmania and Australia

Diving Medicine Specialist and Medical director, Department of Diving and Hyperbaric Medicine Royal Hobart Hospital – managed between 50 and 80 diving accidents annually in Tasmania, including 25-30 cases of decompression illness annually.
Co-author of Tasmania's Emergency management and retrieval protocol for Diver Emergencies – updated second yearly for two decades.
Teaching of Tasmanian recreational diver seminars annually – Combined Dive Clubs weekend Bicheno Tasmania in June, and Diver safety and awareness evenings run for the general public at Royal Hobart Hospital, and Marine safety events with Marine and Safety Tasmania.
Medical Doctorate Thesis published in 2005: Expired carbon monoxide as a marker of poisoning and its application in determining treatment endpoints.
Active campaigner for preventing carbon monoxide poisoning in the community and in divers.
Production of Diver safety videos with Marine and Safety Tasmania:
Recipe for Safe Diving from Tasmania (2003)
https://www.youtube.com/watch?v=1IZiGA4MEdA
(Hookah Diving Safety 2013)
https://www.mast.tas.gov.au/guides/carbon-monoxide-co-poisoning
(CO poisoning 2017)
Contributor to Australian Resuscitation Council guidelines on first aid management of injured divers.
Member Australian Standards Diving Safety Committees from 1999-2020 for Australian and New Zealand Standards AS/NZS 2299.1, 2299.2, 4005.1, 2815.1, 2815.2, 2815.3, 2815.4, 2815.5 and 2815.6, 4005.1-4 and 4774.2 (including revisions).

Research papers on diver safety and accident management

Working with Tasmania's Aquaculture Industry for over 30 years – leading to very significant reductions in decompression illness and improvements in diver safety
SPUMS Journal. 1990;20:159–65.
Papers and Proceedings of the Royal Society of Tasmania. 1999;133(1):77–83.

Diving and Hyperbaric Medicine. 2014;44(3):124–36.
Management of Diving Emergencies. Emergency Medicine 1997;9:42–44.
Health risk management in Tasmania's Abalone industry. Diving and Hyperbaric Medicine. 2010;40(2):83–87.
Australian standards for occupational and recreational divers. Change in the wind? Diving and Hyperbaric Medicine. 2010;40(3):160–1.
Diving related pulmonary oedema – dive accident investigation to assist the forensic pathologist. Diving and Hyperbaric Medicine. 2014;44(2):97–100.
Safety in hyperbaric environments – lithium batteries. Diving and Hyperbaric Medicine. 2011;41(3):165.
Epilepsy, scuba diving and risk assessment. Diving and Hyperbaric Medicine. 2013;43(1):37–41.
Management of inner ear barotrauma in divers and recommendations for returning to diving. Diving and Hyperbaric Medicine. 2014:44(4):208–22.
Occupational diving training in Australia. Diving and Hyperbaric Medicine. 2017;47(4):214–5.
A 20-year review of Diving Deaths in Tasmania. Diving and Hyperbaric Medicine. 2019;49(1):21–9.

Educational activities

Training of doctors nurses, paramedics, medical students and divers in dive safety, management of diving accidents and assessment of medical fitness to dive in order to prevent diving accidents.
ANZHMG Introductory Course in diving and Hyperbaric Medicine Faculty 1998 – present.
MOUM Course Royal Australian Navy 2002–2021
Medical Support of Offshore and Saturation Diving 2016 (Level 2D), convener.
Course instructor medical management of diving accidents – diver medical technician courses, ADAS Accredited, Occupational and Offshore Divers the Underwater Centre Tasmania, 1997–2017.
Course instructor at Tasmanian Hyperbaric Nursing courses from 1995–2024.
COVID Safety – use of AMRON Hoods for Oxygen administration at 1 ATA – Local Royal Hobart Hospital procedures.
COVID and diving fitness – President's report, Diving and Hyperbaric Medicine. 2020;50(2):188–90.

Presentations on diving safety

International Outreach – Teaching management of diving accidents to doctors and nurses in Fiji, 2002–2020 (last three years via Emergency Life Support courses).
Fiji Hyperbaric Facility – specialist advice regarding safety issues in previous Fiji hyperbaric facility, and assistance with the specifications/implementation of current CWMH Suva hyperbaric facility.
Stay Safe when you dive – educational publication. Tasmanian seafood industry.

Research on hyperbaric attendant safety

2009: Hyperbaric chamber attendant safety 1. Doppler analysis of decompression stress in multiplace chamber attendants. Diving and Hyperbaric Medicine. 2009;9(2):63–70.
2009: Hyperbaric chamber attendant safety 2. 14-year health report of multiplace chamber attendants. Diving and Hyperbaric Medicine. 2009;39(2):71–6.
SPUMS Representative: Australian Federal work health and safety legislation.
SPUMS Representative: review of Diving at work section of the Federal Legislation (2014–18).
Tasmanian Diving Industry safety.
Tasmanian Abalone Industry Diving Code of Practice review 2018–19.
Worksafe Tasmania – presentations on diving safety to Tasmanian occupational and scientific diving sector.
Advisor to CSIRO diving committee and Australian Antarctic division of diving safety.
Advisor to Aquaculture industry on diving health and safety standards.

Expedition work

Australian Antarctic Division Research Expedition #68 Casey Base 2014 – Medical Supervisor of Diving Operations, including setting up emergency protocols, recompression chamber facilities, overseeing dive safety and review of operational procedures.

Productions of national guidelines for diver health risk assessment and management of accidents

SPUMS Diving medical health risk assessment for diving (1999), (2011), (2020) and (2025).
Australian Resuscitation Council – Guideline 9.3.5 (2011) resuscitation of divers who have used compressed gas.

International collaborations – for medical guidance and prevention of diver injury

Stellenbosch Collaborative – content provider to the development of a modular course in Diving and Hyperbaric Medicine Stellenbosch University, South Africa. (2012).
Approved by Undersea and Hyperbaric Medicine Society.
Joint Position Statement on persistent foramen ovale and diving (2015) with United Kingdom Sports Diving Medical Committee – first author.
Patent Foramen Ovale and Fitness to Dive Consensus (2015) – with Diver Alert Network and Undersea and Hyperbaric Medical Society – speaker and panel discussion.
ISBN 978-1-941027-71-4.
Review of Cardiovascular Risk assessment for diving (2020) (Diving and Hyperbaric Medicine. 2020;50(3):273–77.
European Diving Technology committee invited contributor to review of medical training standards and fitness to dive standards (2025).

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 and Hyperbaric Medicine. 2024;54(4):344–349. doi: 10.28920/dhm54.4.344-349. PMID: 39675743. PMCID: PMC11779524.
South Pacific Underwater Medicine Society (SPUMS) position statement regarding paediatric and adolescent diving. Diving and Hyperbaric Medicine. 2024;54(4):338–43. doi: 10.28920/dhm54.4.338-343. PMID: 39675742. PMCID: PMC11779525.
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 and Hyperbaric Medicine. 2025;55(1):51–55. doi: 10.28920/dhm55.1.51-55. PMID: 40090026. PMCID: PMC12043516.

Educational book chapters covering diving accidents

Dysbarism – Textbook of Adult Emergency Medicine (Multiple editions).
Oxygen Therapy – Textbook of Adult Emergency Medicine (Multiple editions).

Diving environment

Development of SPUMS policies on environment and banning of single use plastics.

Convenor of SPUMS 51st Annual Scientific Meeting. Theme: Diver health and ocean health amid the storm clouds of climate change.

Related interests – marine toxinology

Participant in Institute of Marine and Antarctic Studies, University of Tasmania Marine Research on recreational Fisheries and sustainability.
Speaker on Marine food toxins and their effects on humans, and impact of ballast water delivering exotic marine species to remote environments.
Support for the diving operations of the Free Ocean Carbon Experiment (Antarctic Section), 2014–2015.
Scombroid fish poisoning: Med J Aust. 1992;157:748–51.
Faculty Member, International Clinical Toxinology Short Course – marine venoms and food poisons International and Australian Animal Venoms and Poisons).
Book chapter: Clinical toxinology of Shellfish poisoning, in clinical toxicology of animal venoms and poisons. Meier J, White J, CRC Press USA; 1995. ISBN 0-8493-4489-1.

Recreational Diver activity

Recreational scuba diver since 1983 – master diver qualification
Over 3,500 hours logged underwater
Recreational diving photographer

Photo: David and Annette Smart, at Government House Tasmania, for the Order of Australia Awards.

SPUMS Diploma in Diving and Hyperbaric Medicine

(Updated June 2025)

Requirements for candidates

For the Diploma of Diving and Hyperbaric Medicine (Dip DHM) to be awarded by the Society, the candidate must:

- be medically qualified;
- remain a current financial member of the Society for the duration of their candidacy for the Diploma;
- pay such administrative fees and charges (e.g., candidate registration fee) as may, from time-to-time, be approved by the Society's Executive;
- supply evidence of satisfactory completion of an examined two-week fulltime course in Diving and Hyperbaric Medicine at an approved facility. The list of such facilities may be found on the SPUMS website;
- have completed the equivalent (as determined by the Education Officer) of at least six months' fulltime clinical training in an approved Hyperbaric Medicine Unit;
- 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;
- 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 for 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 01 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 01 January 2026 a one-off Registration Fee of AUD $250.00 will be payable at the time of enrolment for 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 2025, the SPUMS Academic Board consists of:
Dr David Cooper, Education Officer
Associate Professor Simon Mitchell.

All enquiries and applications should be sent to:
Dr David Cooper
Email: *education@spums.org.au*

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

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 President (currently Dr Neil Banham)
SPUMS Immediate Past President (currently Professor David Smart)
SPUMS Education Officer (currently Dr David Cooper)
Diving and Hyperbaric Medicine Journal Editor (currently 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 brief synopsis (1–2 pages) of the project and be submitted to president@spums.org.au.

Closing date: 31 December 2025

Dr Neil Banham MBBS, FACEM, DipDHM, ANZCA DipAdvDHM
SPUMS President

Royal Australian Navy Medical Officers' Underwater Medicine Course

Dates: 13–24 October 2025

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 remains at AUD$1,355 (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)2-9647-5572*
Fax: *+61 (0)2-9647-511*
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 2026, dates will again be from mid to late February 2026 for two weeks.

Dates: 16–27 February 2026
Venue: Hougoumont Hotel, Fremantle, Western Australia
Cost: AUD$3,300.00 (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

SPUMS

South Pacific Underwater Medicine Society

54TH ANNUAL SCIENTIFIC MEETING

Palasia Hotel, Palau

10–15 May 2026

Theme: "Free diving"

Register now at:
https://spums.au/index.php/asm-registration

Notices and news

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

President's report

Jean-Eric Blatteau

A farewell note from the President of the EUBS

As my mandate as President of the European Underwater and Baromedical Society comes to a close at our Annual Scientific Meeting in Helsinki this September, I would like to share a few personal reflections.

I have had the privilege of taking over this responsibility in the aftermath of the COVID period, a time when our society had to adapt rapidly. Instead of our traditional face-to-face meetings, we organised two webinars, which turned out to be a real success and allowed our members to stay connected and engaged despite the difficult circumstances. Fortunately, we were soon able to return with great joy to our in-person congresses in wonderful locations such as Prague, Porto, Brest, and now Helsinki. Each of these meetings was a true celebration of science, friendship, and our shared passion for diving and hyperbaric medicine.

Over the past years, EUBS has strengthened its position as a leading society in our field. A particularly important step was the closer integration with the European Committee for Hyperbaric Medicine (ECHM), that is now formally recognised in our Constitution. This has been done with full respect of the unique role and missions of the ECHM, including the organisation of the next Consensus Conference in hyperbaric medicine, which is eagerly awaited by our community. Together, EUBS and ECHM have also produced several joint position statements, such as on the use of low-pressure chambers in humans and on the European Code of Good Practice for HBOT.

I also very much hope that we will soon be able to organise another joint congress with SPUMS. The previous TRICON meetings were outstanding successes, both scientifically and socially, and I believe they represent the spirit of collaboration and friendship that drives our societies.

Looking beyond our own community, it is clear that the world around us is facing great challenges. Armed conflicts and humanitarian crises sadly continue, and with them come devastating injuries and long-term consequences, from complex wounds and crush injuries to psychological trauma such as post-traumatic stress disorder. Hyperbaric medicine, though often associated with diving and chronic conditions, may also have a role to play in supporting the recovery of such patients. As a scientific society, we have a responsibility not only to advance our discipline in its traditional domains, but also to explore how HBOT might contribute to addressing the medical and humanitarian needs of our time.

The future of the EUBS is therefore both exciting and important. I believe our congresses will continue to grow as a place for science, but also for education – with more workshops, more hands-on training, and more opportunities for younger colleagues. I also encourage all of you to contribute to our excellent common journal, *Diving and Hyperbaric Medicine*, which continues to thrive under the tireless leadership of Professor Simon Mitchell.

I would also like to warmly congratulate our new President, Professor Bengusu Mirasoglu. She is an internationally recognised expert in hyperbaric medicine, and she demonstrated remarkable leadership in her work in Istanbul, especially during the tragic earthquake in Türkiye. She will be supported by our new Vice-President, Dr Anders Kjellberg from Stockholm, whose innovative research on HBOT in COVID patients has been particularly inspiring.

None of this would have been possible without the constant dedication and hard work of Dr Peter Germonpré and the entire Executive Committee, who work tirelessly, often behind the scenes, to keep our society moving forward.

For me, it has been an immense honour to serve as President of the EUBS. I am deeply grateful for the trust you have placed in me and for the friendships that have grown along the way. I now pass on the torch with pride, and with confidence that the EUBS will continue to thrive, not only as a scientific society, but also as a community ready to contribute to the great medical and humanitarian challenges of our time.

With gratitude and warmest regards,

Professor Jean-Eric Blatteau
President, EUBS

EUBS Notices and news

EUBS Member-at-Large elections

The EUBS Elections again held electronically, using the ElectionRunner software. 55% of our members voted, which is above average compared to previous years – however it could still be improved.

We have elected a new Vice-President: Dr Anders Kjellberg was approved by a vast majority of votes. He will immediately take office and follow the path of Dr Bengusu Mirasoglu, our new President.

We had to elect a Member-at-Large for the four-year period 2025–2029, but as Dr Kjellberg can not combine the position of Member-at-Large with Vice-Presidency, there will be two new Members-at-Large this year. There were three candidates: Dr Pieter Bothma from the UK, Dr Gamze Sumen from the Turkey and Dr Benoît Desgraz from Switzerland. The candidate who scored best was Dr Benoît Desgraz, and he will become the Member-at-Large 2025; the second elected candidate, Dr Pieter Bothma will replace Dr Kjellberg and will serve for three years, until 2028. Both will take office as from our General Assembly on 6 September 2025. We will be saying goodbye to our 2021 Member-at-Large, Dr Evangelos Papoutsidakis, for having served faithfully on the ExCom for four years. We thank Vangelis for his efforts for our society and hope be able to count on him 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 email: secretary@eubs.org.

EUBS2025 Annual Scientific Meeting, Helsinki, Finland, from 1–6 September 2025

As this issue of Diving and Hyperbaric Medicine journal is published, we will have had the pleasure to unite again in Helsinki, from 1–6 September 2025. 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 49th Annual Scientific Meeting of EUBS will have been a great success.

During our General Assembly, a change in our Constitution and Bylaws will have been approved, officialising the close cooperation and integration of the activities and roles of ECHM and EUBS. The new version of the EUBS Constitution and Bylaws can be found on the EUBS website. This merger has been prepared since our 2019 Scientific Meeting, and now that the change is official, we can work to make this a streamlined and efficient scientific 'machine' ! The very first concerted action will, in fact, be the organisation of the next Consensus Conference on Indications for HBO therapy, in 2026, joint with our next Annual Scientific Meeting.

Indeed, next year, the EUBS meeting will be in Geneva, Switzerland, from 14–19 September 2026, so please keep these dates already free in your busy agendas. Also, plan to have some days off before and after the conference to enjoy the Geneva area, extra beautiful that time of the year.

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.

Courses and meetings

Scott Haldane Foundation

As an institute dedicated to education in diving medicine, the Scott Haldane Foundation has organised more than 320 courses all over the world, over the past 33 years. SHF is targeting on an international audience with courses worldwide.

Below is the schedule of upcoming SHF-courses in 2025.

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

Second half of 2025

8–15 November	In-depth course What a Diving doctor MUST know (level 2d) Bali, Indonesia
15–22 November	In-depth course What a Diving doctor MUST know (level 2d) Bali, Indonesia
On request	Internship HBOt (level 2d) NL/Belgium

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

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

EUBS and SPUMS members are able to access the German Society's large database of publications in diving and hyperbaric medicine. EUBS members have had this access for many years. For SPUMS members access will be available soon for you, GTÜM has a new website and access is being created specifically for you. There will be a link in the 'members only' area of the SPUMS website. This should be available in the next month, so keep an eye out.

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

Historical Diving Society Australia - Pacific

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

Biennial Joint Conference of The United Kingdom Diving Medical Committee and The British Hyperbaric Association

Monday 3 November

- Anticoagulants and diving
- Habitat Diving
- DDRC fitness to dive audit
- Health and Safety Executive Update
- Cardiovascular update

Tuesday 4 November

- Ear Nose and Throat fitness topics
- TOE for PFO detection
- Gas Toxicity
- Case Reports
  - DCI and ADHD
  - importance of early recompression
  - DCS after a shallow dive
  - iatrogenic gas embolism
- Safe use of batteries in chambers
- BHA Helpline audit

Wednesday 5 November

- Pelvic osteo-radionecrosis
- Gas Embolism
  - urgency of recompression
  - delayed presentation
  - rat model
  - what do we need to do?
  - prevention
  - from a 2ml syringe
  - from CO2
  - Pro-Con Debate: 1 or more treatments?
- Critically ill patients treated by LHM Healthcare
- Heimlich valve alternative

Register to attend or for remote access at
https://sites.google.com/view/ukdmcbha2025/home

Diving and Hyperbaric Medicine: Instructions for authors

(Short version – updated June 2024)

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

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

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

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

Types of articles: DHM welcomes contributions of the following types:

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

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

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

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

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

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

Supplements to a particular issue are occasionally published for purposes deemed appropriate by the editor. These may accommodate articles / treatises that are too long for the main journal or collections of articles on thematic areas. There is no open portal for submission of such material and any plans or suggestions for supplements should be discussed with the Editor before writing.

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

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

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

Instructions for authors (full version 2024 – this document)
DHM Keywords 2023
DHM Mandatory submission form 2024
Trial design analysis and presentation
Conflict of interest statement
English as a second language
Guideline to authorship in DHM 2015
Samples of formatted references for authors of journal articles (last reviewed 2024)
Recommendations for the conduct, reporting, editing, and publication of scholarly work in medical journals 2024
Helsinki Declaration revised 2013
Is ethics approval needed?

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 AUD5,000.00.

There are two categories of scholarships:

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

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