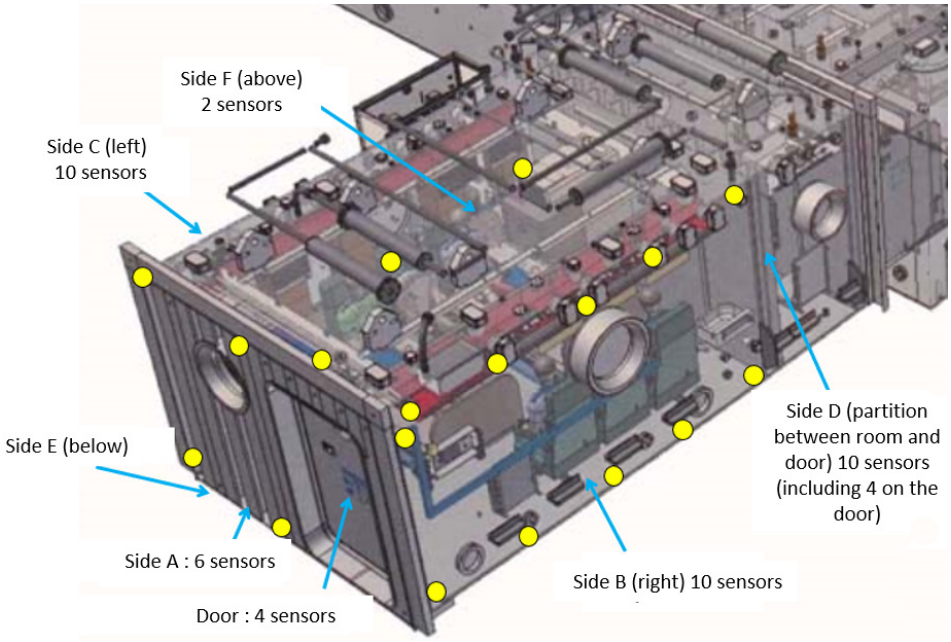
Diving and Hyperbaric Medicine

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

SPUMS *Volume 54 No. 3 September 2024* EUBS



Pneumatic testing of hyperbaric chambers

Necrotising infection clinical composite endpoint

HBOT for spondylodiscitis

Methylphenidate and oxygen toxicity

Return to diving after COVID-19

Challenging cases for diving medical examiners

Arterial dissection in divers

Tympanocentesis prior to submarine escape

Evaluation of a hyperbaric ventilator

Investigation of diving fatalities

Large lungs and barotrauma risk

Five case reports

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

CONTENTS

Diving and Hyperbaric Medicine Volume 54 No. 3 September 2024

154 The Editor's offering

Original articles

155 Validation of necrotising infection clinical composite endpoint in a retrospective cohort of patients with necrotising soft tissue infections
Victoria Bion, Dylan Jape, Rachel Niesen, Margaret Angliss, Frank Bruscino-Raiola, Biswadev Mitra, Bridget Devaney

162 The role of hyperbaric oxygen treatment in the management of spondylodiscitis
Kübra Canarslan Demir, Burak Turgut, Gözde B Sarıyerli Dursun[1], Fatma S Konyalioğlu, Taylan Zaman

168 Methylphenidate and the risk of acute central nervous system oxygen toxicity: a rodent model and observational data in human divers
Ivan Gur, Yehuda Arieli, Yinnon Matsliah

176 Medical examination of divers after COVID-19 infection: a prospective, observational study using published (original and revised) guidelines for evaluation
Charlotte Sadler, Anna Lussier, Ian Grover, Karen Van Hoesen, Peter Lindholm

184 Retrospective analysis of challenging cases for medical examiners of diving
Inge Reus, Erik van de Sande, Rienk Rienks, Thijs Wingelaar

188 Arterial dissection in scuba divers: a potential adverse manifestation of the physiological effects of immersion
Neal W Pollock, John Lippmann, John Pearn, John Hayman

196 Role of tympanocentesis in the prevention of middle ear barotrauma induced by fast buoyant ascent escape from 200 m underwater
Xu Liu, Hengrong Yuan, Jieying Peng, Guanghao Zhu, Nan Wang, Yukun Wen, Hongliang Zheng, Yiqun Fang, Wei Wang

Technical reports

204 Acoustic emission, an innovative diagnosis tool for therapeutic hyperbaric chambers: or how to requalify safely using pneumatic pressure test
Johann Catty, Olivier Seguin, Jean-Laurent Juillie, Daniel Mathieu, Erika Parmentier-Decrucq

212 Evaluation of a new hyperbaric oxygen ventilator during pressure-controlled ventilation
Cong Wang, Qiuhong Yu, Yaling Liu, Ziqi Ren, Ying Liu, Lianbi Xue

217 The investigation of diving accidents and fatalities
John Lippmann, James Caruso

Short communication

225 Large lungs in divers: a risk for pulmonary barotrauma?
Robert A van Hulst, Pieter-Jan AM van Ooij

Case reports

230 Maxillary sinus barotrauma with infraorbital nerve paraesthesia after breath-hold diving
Kubra Canarslan Demir, Zeliha Yücel

233 Lateral ST-elevation myocardial infarction from systemic air embolism after CT guided lung biopsy
Aung Myo Htay, Emma Wilson

237 Bispectral index with density spectral array (BIS-DSA) monitoring in a patient with inner ear and cerebral decompression sickness
Gerald Schmitz, Sharon Aguero

242 Decompression sickness in surface decompression breathing air instead of oxygen
Jan Risberg, Helle Midtgaard

249 Hyperbaric oxygen treatment (HBOT) in a case of traumatic chondronecrosis of the cricoid cartilage
Subhranshu Kumar, HBS Chaudhry, Chandrasekhar Mohanty, Sourabh Bhutani, Muhammed Risham, Kshitij Lanjekar

Letter to the Editor

252 Hyperbaric medicine and climate footprint
Alice Varichon, Rodrigue Pignel, Sylvain Boet

Errata

253 Correction: Formulating policies and procedures for managing diving related deaths: a whole of state engagement from frontline and hospital services in Tasmania
Elizabeth J Elliott, Karl Price, Bernard Peters

SPUMS notices and news

254 The President's report
Neil Banham

255 Notices and news

EUBS notices and news

259 The President's report
Jean-Eric Blatteau

260 Notices and news

262 Courses and meetings

263 Diving and Hyperbaric Medicine: Instructions for authors
(Short version – updated June 2024)

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

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

---

DIVING AND HYPERBARIC MEDICINE

https://www.dhmjournal.com/

Diving and Hyperbaric Medicine **is published online jointly by the South Pacific Underwater Medicine Society and the European Underwater and Baromedical Society**
E-ISSN 2209-1491; ABN 29 299 823 713

The Editor's offering

I write having just departed Brest, France, after a spectacularly successful EUBS Annual Meeting. I offer my congratulations to Francois Guerrero and Pierre Lafère on a superb organisational job and a stimulating scientific program.

This third issue of DHM in 2024 contains another eclectic mix of 16 articles of which 10 primarily relate to diving and six to hyperbaric medicine matters.

On the hyperbaric side, Victoria Bion and colleagues present another paper from Bridget Devaney's group that seeks to lay the groundwork for a major trial of hyperbaric oxygen in necrotising soft tissue infections. In this iteration they report their validation of a clinical composite study endpoint. Kübra Canarslan Demir and colleagues report a series of refractory spondylodiscitis patients who achieved a good outcome associated with hyperbaric oxygen treatment (HBOT). The authors are appropriately cautious with their conclusions, but the apparent benefit of HBOT in this sporadic, difficult to treat problem is certainly of interest. Johan Catty and colleagues report on acoustic emission technology employed to facilitate pneumatic (rather than hydrostatic) testing of hyperbaric chambers. Pneumatic testing substantially simplifies the logistics and cost of periodic chamber pressure tests. Having previously reported testing of a new hyperbaric ventilator in volume control mode, Cong Wang and colleagues resubmit this device to testing in pressure control mode. Finally, there are case reports of HBOT used to treat iatrogenic coronary arterial gas embolism arising from lung biopsy, and traumatic chondronecrosis of the traumatic cricoid cartilage.

On the diving side, Ivan Gur and colleagues evaluated the extent to which methylphenidate, commonly used to treat attention deficit disorder, might enhance the risk of oxygen toxicity. In a cohort of human divers there was no signal suggesting increased risk, and in an animal experiment methylphenidate seemed to increase seizure latency. Charlotte Sadler and colleagues applied their previously published guidelines for assessing return to diving to a cohort of patients who had suffered COVID-19 infection. The vast majority of divers were designated suitable for return to diving and the guidelines were judged easy to use. Continuing in a 'fitness for diving' theme, Inge Reus and colleagues reviewed a cohort of cases referred to the Netherlands central board of experts by medical examiners of diving to identify issues commonly considered challenging. Interpretation of multiple comorbidities, guidelines and diagnostic data pertaining to circulatory, respiratory and neurological disorders were the most common problems, and may be targets for continuing medical education. Neal Pollock and colleagues identify cases of arterial dissection reported to have occurred in proximate relationship to diving. Although these lesions are notoriously difficult to confidently attribute to any particular event, the authors plausibly suggest that vascular changes in diving (such as a rise in blood pressure) may be contributory. Xu Liu and colleagues report that tympanocentesis in rats prior to a simulated rapid 200 m compression / decompression (to simulate a swimming submarine escape) prevents the middle ear barotrauma that otherwise invariably occurs. Parenthetically, in their preamble they report that a successful 194.6 m human simulation has taken place in China; an extraordinary feat. John Lippmann and James Caruso review the perennially problematic area of investigation of diving accidents and fatalities. This article contains multiple strategic and technical points of advice for practitioners undertaking reviews of circumstances and autopsies following diving accidents. Rob van Hulst and Pieter-Jan van Ooij report a fascinating study in which they investigate the incidence of blebs and bullae in military divers with large lungs (based on spirometry), and relate these findings to the risk of pulmonary barotrauma. Finally, there are three diving related case reports describing maxillary sinus barotrauma with infraorbital nerve palsy, the use of the bispectral index electroencephalogram device in monitoring a patient with cerebral and inner ear decompression sickness, and decompression sickness following dives performed using the surface decompression on oxygen method. The latter paper by Jan Risberg and Helle Midtgaard reports an unfortunate error in which divers undertaking surface decompression were supplied with air instead of oxygen over the course of a substantial period of diving. Despite this, only two cases of decompression sickness occurred.

One of the biggest challenges to timely processing of manuscripts for the journal is finding suitable reviewers. There is a substantial element of 'reviewer fatigue' in the community at present and having a larger body of reviewers to draw on would be extremely useful. At the EUBS meeting I made a general call for members with an academic interest in undertaking occasional reviews for the journal to notify me of their name, institution, country, email address and particular areas of expertise. I restate that request here. Anyone with an interest in helping the journal in this regard can email me at editor@dhmjournal.com.

Simon Mitchell
Editor, Diving and Hyperbaric Medicine Journal

Cover photo: Acoustic sensor array layout for pneumatic testing of a hyperbaric chamber. See Catty et al. in this issue.

Original articles

Validation of necrotising infection clinical composite endpoint in a retrospective cohort of patients with necrotising soft tissue infections

Victoria Bion[1], Dylan Jape[2], Rachel Niesen[2], Margaret Angliss[3], Frank Bruscino-Raiola[3], Biswadev Mitra[4,5], Bridget Devaney[1,4,5]

[1] *Department of Intensive Care and Hyperbaric Medicine, Alfred Health, Melbourne, Australia*
[2] *School of Medicine, Monash University, Melbourne, Australia*
[3] *Plastics, Hand and Faciomaxillary Surgery Unit, Alfred Health, Melbourne, Australia*
[4] *School of Public Health and Preventative Medicine, Monash University, Melbourne, Australia*
[5] *Emergency and Trauma Centre, Alfred Health, Melbourne, Australia*

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

Keywords
Fournier's gangrene; Hyperbaric research; Necrotising fasciitis; Necrotising infections; Organ dysfunction scores

Abstract
(Bion V, Jape D, Niesen R, Angliss M, Bruscino-Raiola F, Mitra B, Devaney B. Validation of necrotising infection clinical composite endpoint in a retrospective cohort of patients with necrotising soft tissue infections. Diving and Hyperbaric Medicine. 2024 30 September;54(3):155–161. doi: 10.28920/dhm54.3.155-161. PMID: 39288918.)
Introduction: Rapidly progressive necrotising soft tissue infections (NSTIs) are associated with high mortality and morbidity. Low incidence and disease heterogeneity contribute to low event rates and inadequately powered studies. The Necrotising Infections Clinical Composite Endpoint (NICCE) provides a binary outcome with which to assess interventions for NSTIs. Partly with a view towards studies of hyperbaric oxygen treatment in NSTIs we aimed to validate NICCE in a retrospective cohort of NSTI patients.
Methods: Eligible patients were admitted between 2012 and 2021 to an adult major referral hospital in Victoria, Australia with surgically confirmed NSTI. The NICCE and its constituents were assessed in the whole cohort ($n = 235$). The cohort was divided into two groups using the modified sequential organ failure assessment (mSOFA) score, with an admission mSOFA score ≥ 3 defined as high acuity.
Results: Baseline characteristics of the whole ($n = 235$), the high ($n = 188$) and the low acuity cohorts ($n = 47$) were similar. Survival rates were high (91.1%). Patients with an admission mSOFA ≥ 3 were less likely to meet NICCE criteria for 'success' compared to the lower acuity cohort (34.1% and 64.7% respectively). Meeting NICCE criteria was significantly associated with lower resource utilisation, measured by intensive care unit days, ventilator days, and hospital length of stay for all patients and for those with high acuity on presentation.
Conclusions: The NICCE provides greater discriminative ability than mortality alone. It accurately selects patients at high risk of adverse outcomes, thereby enhancing feasibility of trials. Adaptation of NICCE to include patient-centred outcomes could strengthen its clinical relevance.

Introduction

Necrotising soft tissue infections (NSTI) are a group of severe, rapidly progressive infections of subcutaneous tissue, fascia or muscle associated with high mortality and morbidity. Incidence varies globally with rates as high as 15 per 100,000 in Thailand[1] and as low as 0.3 per 100,000 in high income countries such as the USA[2] and Norway.[3] The majority of published mortality rates vary between 20–30% with 15% of patients experiencing disability, sequelae and amputation.[4] At the Alfred Hospital, a quaternary hospital in Melbourne, Australia, the mortality rate for patients with a diagnosis of NSTI at hospital discharge was reported to be 14%.[5]

Standard treatment of NSTI involves empiric antimicrobials, early and repeated surgical debridement of non-viable tissue, intensive care support, and use of adjuvant therapies.[6] Patients may require amputation in the early phase of the disease, followed by complex reconstructive procedures and extensive rehabilitation in nearly half of survivors.[7] Whilst early surgical intervention has been shown to reduce mortality and the number of operations required to control the disease,[8–10] there is limited high level evidence for any treatment modality for NSTI.[11]

The Alfred Hospital provides a state-wide service for hyperbaric oxygen treatment (HBOT). This has become an established component of NSTI treatment at many centres. Support for HBOT is based on proposed physiological mechanisms, case reports, retrospective case-control studies, and a systematic review and meta-analysis published in 2021 which concluded that HBOT reduced the odds of dying from NSTI (odds ratio 0.44 [95% CI 0.33–0.58]).[12] Research into the efficacy of HBOT in NSTI has continued including a recent Danish nationwide population-based observational study that demonstrated a significant association between HBOT and improved 30 day survival.[13] There are no randomised controlled trials to support or refute use of HBOT for NSTI. A 2015 Cochrane review cited the following reasons for insufficient randomised controlled trial data; heterogeneity in the disease process, disease severity, anatomical location and management of NSTI, as well as low disease incidence.[14]

Given the low incidence of NSTI, randomised controlled trials evaluating interventions should include outcome measures with higher event rates than mortality alone in order to improve the feasibility of successful recruitment and to adequately power future studies. It would also be important to include patient-centered and clinically important outcome measures.[15]

In 2017, Bulger et al, developed the Necrotising Infections Clinical Composite Endpoint (NICCE) as a standardised outcome measure to enable comparison between studies and meta-analyses, and as a means of measuring intervention efficacy in future randomised controlled trials.[16] The NICCE criteria incorporate local tissue injury, systemic organ dysfunction and mortality to produce a binary measure of 'success' or not. Whilst other diagnostic composite tools have been developed and assessed with variable success,[17] to our knowledge, the use of a composite outcome score has not been evaluated previously.

The NICCE appears promising in that it demonstrated both internal component consistency as well as face and criterion validity in the US patient cohort in which it was developed and tested. The score has not been validated in an Australian patient cohort to date and if valid would provide a standardised means of assessing NSTI outcomes in future studies. The aim of this study was to validate NICCE in a retrospective cohort of patients with NSTI at an adult major referral hospital in Victoria, Australia.

Methods

The study protocol was reviewed and approval to proceed granted by the Alfred Hospital Human Research and Ethics Committee (Project ID 704/20). The requirement to seek informed consent from patients or persons responsible was waived.

We performed a retrospective cohort analysis of NSTI patients admitted to the Alfred Hospital between 2012 and 2021. Case records were obtained for patients with a diagnosis of NSTI from the intensive care and hyperbaric services' database and plastic surgery databases. Inclusion criteria required patients to have a diagnosis of NSTI confirmed surgically. Cases of clostridial myonecrosis were included. Ambiguous cases were reviewed by a minimum of two authors to ensure that inclusion criteria were met. Cases without a diagnosis of NSTI or where disease control was achieved at the primary hospital prior to transfer were excluded.

Baseline variables, NICCE components, and outcome variables were collected from electronic medical records and entered into a standardised data collection tool using REDCap software. Baseline variables included age, sex, admission weight, site of infection, secondary referral, and admission modified sequential organ failure assessment (mSOFA). A high acuity subset was identified using an admission mSOFA score of three or greater. This subset was analysed in the method described by Bulger et al.[16] We also included data for the low acuity group (mSOFA < 3) for further comparison as high acuity patients were over-represented in the total cohort. Rationale for use of the mSOFA score has been described previously.[16]

To simplify the data collection process, given that the public healthcare system in Victoria utilises multiple different electronic medical record systems, we opted to collect admission data from the point of admission to the Alfred Hospital, regardless of transfer status. We expected a large number of cases to have a primary admission elsewhere prior to transfer and therefore included primary hospital debridements in our analysis. All operative findings, including those from the primary hospital, were included where available from the Alfred Hospital medical records. Patients repatriated to their referral hospital with disease controlled and a trend towards recovery, or those that were stepped down were assumed to be alive at day 28. Those that were repatriated to another hospital's intensive care unit were considered to have an inconclusive outcome.

Outcome variables collected included NICCE components (listed below) and resource utilisation as determined by number of intensive care unit days, ventilator days and hospital length of stay. Intensive care unit-free and ventilator-free days were calculated out of 28 for the study period. In addition to following the process used by Bulger et al.[16] we collected data on the use of HBOT and number of hyperbaric treatments completed.

The NICCE outcome and resource utilisation measures for both the high acuity (mSOFA ≥ 3) and the low acuity (mSOFA < 3) subsets were also compared to the whole cohort.

NICCE VARIABLES COLLECTED

- Alive at day 28
- Three or less debridements before day 14
- No amputation beyond first debridement*
- Modified SOFA** score at day 14 of one or less

Achievement of the NICCE requires all criteria to be met.
*debridement was defined as removal of necrotic tissue, not just operative exploration
**mSOFA components: peripheral oxygen saturation to inspired oxygen fraction (SpO_2/FiO_2) ratio, blood pressure and use of vasopressors or inotropes, Glasgow Coma Scale (GCS), creatinine.

DATA ANALYSIS

Baseline characteristics of the whole cohort and the high and low acuity subsets (mSOFA ≥ 3 or mSOFA < 3 respectively) were summarised using medians and interquartile ranges (IQR). Individual components of NICCE are presented as a percentage of the cohort meeting each criterion, as well as the percentage meeting all NICCE components.

The Wilcoxon rank test was used to compare indicators of resource allocation between cases that met NICCE criteria and those that did not in both the whole cohort and the high acuity subset. Indicators of resource utilisation presented include intensive care unit-free days, ventilator-free days, and hospital length of stay. A result was considered significant if the *P*-value was less than 0.05. All analyses were conducted using Stata v 15.1 (College Station, TX, USA).

Results

Two hundred and thirty-three cases of NSTI were identified from the hyperbaric database, of which 20 were excluded due to prior disease control at referral centre ($n = 4$), absence of surgical confirmation of disease or alternate diagnosis ($n = 14$) and administrative error ($n = 2$). An additional 23 cases were identified in the plastic surgery database, of which one was excluded for having disease control prior to transfer to our centre.

An mSOFA score of ≥ 3 was observed in 188 (80%) of the cohort, and defined as the high-acuity cohort. Baseline characteristics of the whole cohort ($n = 235$), the high acuity cohort ($n = 188$) and the low acuity cohort ($n = 47$) are presented in Table 1. Age, sex, admission weight, and site of infection were similar between the three groups. Most patients were transferred from other hospitals (89.4%), and 64.3% had an endotracheal tube placed prior to admission at the Alfred Hospital. Transferred patients were admitted to the Alfred Hospital a median of one day after initial presentation to the primary hospital. Two hundred and nineteen patients (93.2%) received HBOT. The median number of HBOT sessions was five (IQR 4–7). Patients who returned to their

Table 1
Baseline characteristics of the study cohort; IQR – interquartile range; mSOFA – modified Sequential Organ Failure Assessment; SD – standard deviation

Variable	Total population ($n = 235$)	Population with initial mSOFA ≥ 3 ($n = 188$)	Population with initial mSOFA < 3 ($n = 47$)
Age (years), mean (SD)	55.2 (15.9)	56.0 (15.9)	51.9 (15.6)
Male Female	147 (62.5%) 88 (37.5%)	119 (63.3%) 69 (36.7%)	28 (59.6%) 19 (40.4%)
Admission weight (kg) mean (SD)	93.1 (29.0)	93.7 (29.4)	90.4 (27.1)
Site of infection Extremity Perineum Head and/or neck Other Multiple sites	 136 (57.9%) 79 (33.6%) 9 (3.8%) 43 (18.3%) 30 (12.8%)	 109 (58.0%) 61 (32.4%) 7 (3.7%) 37 (19.7%) 24 (12.8%)	 27 (57.4%) 18 (38.3%) 2 (4.3%) 6 (12.8%) 6 (12.8%)
Referred from another hospital	210 (89.4%)	171 (91.0%)	39 (83.0%)
Number of days in primary hospital, median (range)	1 (1–2)	1 (1–2)	2 (1–6)
Intubated on admission	151 (64.3%)	151 (80.3%)	0
Admission mSOFA, median (IQR)	9 (4–12)	10 (7–12)	0 (0–1)

Table 2
Components of NICCE endpoint by cohort; † – data missing for 1 patient; ‡ – complete data available for 173 patients; § – complete data available for 172 .patients; mSOFA – modified Sequential Organ Failure Asssessment

Variable	Total population (*n* = 235)	Population with initial mSOFA ≥ 3 (*n* = 188)	Population with initial mSOFA < 3 (*n* = 47)
≤ 3 debridements	158 (67.2%)	121 (64.4%)	37 (78.7%)
Amputation beyond first debridement †	14 (6.0%)	12 (6.4%)	2 (4.3%)
mSOFA ≤ 1, day 14 ‡	104 (44.3%)	72 (38.3%)	32 (68.1%)
28-day survival	214 (91.1%)	169 (89.9%)	45 (95.7%)
NICCE composite endpoint – all criteria met §	69 (40.1%)	47 (34.1%)	22 (64.7%)

Table 3
Relationship between NICCE and resource utilisation; data are median (interquartile range); ICU – intensive care unit; LOS – length of stay; mSOFA – modified Sequential Organ Failure Asssessment

Groups / parameters	All NICCE criteria met	Did not meet all NICCE criteria	*P*-value
All patients	**(*n* = 69)**	**(*n* = 103)**	
ICU days, median	4 (3–8)	14 (8–19)	< 0.001
ICU free days, median	24 (20–25)	14 (9–20)	< 0.001
Ventilator days, median	3 (0–6)	9 (5–13)	< 0.001
Ventilator-free days, median	25 (22–28)	19 (15–23)	< 0.001
Hospital LOS, median, days	21 (17–28)	33 (2–555)	< 0.001
Admission mSOFA ≥ 3		**(*n* = 91)**	
ICU days, median	6 (4–9)	15 (9–23)	< 0.001
ICU free days, median	22 (19–24)	13 (6–19)	< 0.001
Ventilator days, median	4 (3–7)	9 (6–14)	< 0.001
Ventilator-free days, median	24 (21–25)	19 (14–22)	< 0.001
Hospital LOS, median, days	21 (18–28)	34 (25–57)	< 0.001

primary hospital either after reconstruction had begun, or with ward level care (79 patients) were assumed not to have any further debridements or amputations, and to have survived to day 28, whilst two patients repatriated with ongoing intensive care unit-level care were considered to have insufficient data to determine all NICCE criteria. This only resulted in missing data for 11 patients.

Individual NICCE components and those achieving overall NICCE criteria for 'success' are displayed in Table 2. Patients with initial mSOFA ≥ 3 were less likely to meet all NICCE criteria compared to the lower acuity cohort (34.1% and 64.7% respectively). The differences observed between the low and high acuity groups for all NICCE criteria as well as the individual component of mSOFA at day 14 were substantially larger than the individual components of debridement, amputation, or 28-day survival.

Meeting all NICCE criteria was associated with lower resource utilisation for all patients as well as for those with high acuity at baseline (Table 3). Meeting all NICCE criteria was associated with reduced intensive care unit length of stay, fewer ventilator days, and reduced hospital stay (Figures 1–3). This remained significant when comparing ICU-free and ventilator-free days which would account for deaths prior to the 28-day study period.

Discussion

The NICCE demonstrated good discriminative ability in comparison to the individual components of debridement, amputation, or 28-day survival. In our analysis, mSOFA score at day 14 appears as discriminative as the overall NICCE outcome, suggesting that this component may perform as well as the composite measure. This could

Figure 1
Intensive care unit (ICU) length of stay (days) among patients who met NICCE criteria compared to those that did not in the whole cohort

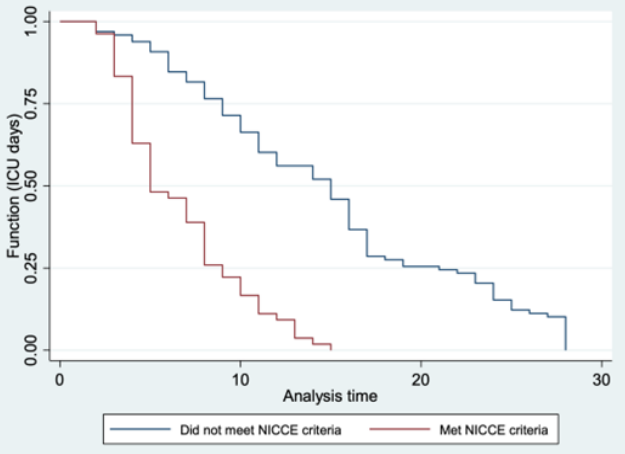


Figure 2
Number of days requiring invasive ventilation out of 28-day period among patients who met NICCE criteria compared to those that did not in the whole cohort

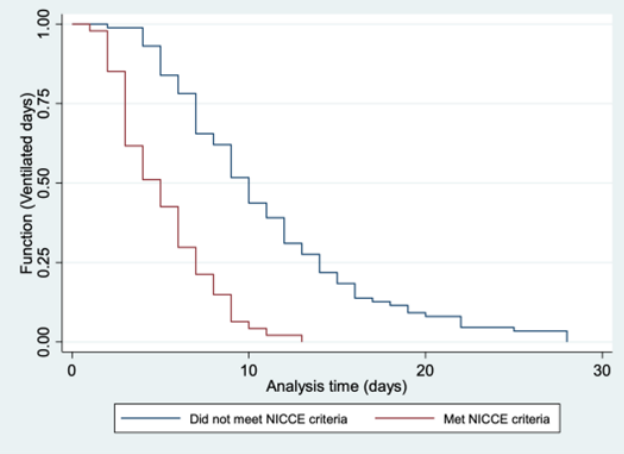
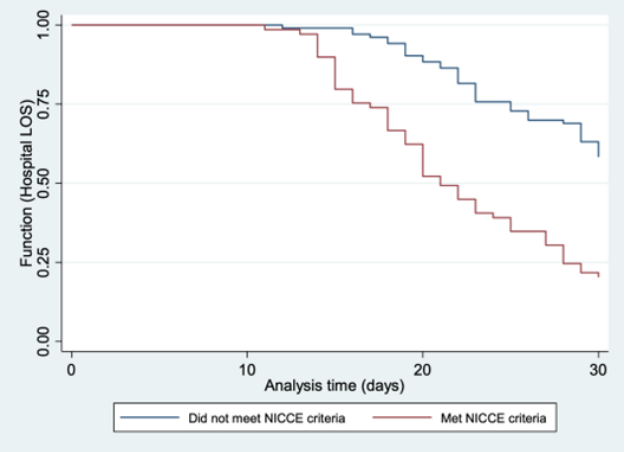


Figure 3
Hospital length of stay (LOS) (days) among patients who met NICCE criteria compared to those that did not in the whole cohort



indicate that mSOFA at day 14 could provide a less complicated, and as effective an endpoint compared to NICCE. Conversely, mSOFA at day 14 is likely to be seen as less important to patients than other components such as operations, amputation, and survival. Among patients with mSOFA ≥ 3, the proportion of patients meeting NICCE criteria in our cohort was similar to that reported elsewhere (33%).[16] The NICCE composite endpoint provided greater discriminative ability than mortality alone.

The number of cases included from the Alfred Hospital was similar to the cohort reported by Bulger et al.[16] (235 and 238 respectively). Eighty percent (188/235) of the Alfred Hospital cohort had an admission mSOFA score ≥ 3 compared with 35% in the Bulger cohort (69/198), indicating a higher acuity cohort at the Alfred Hospital. High acuity cases in the Bulger cohort were identified from one of the trial datasets, not both, resulting in the smaller denominator above.

The study by Bulger et al.[16] utilised data from two pre-existing trial datasets with extensive predetermined exclusion criteria, including two different age cut-offs, comorbidities such as human immunodeficiency virus (HIV) infections and end stage organ dysfunction, as well as extremis on admission. These criteria exclude patients with high acuity on admission. Whilst the Bulger dataset included multiple centres, the extensive exclusion criteria limits applicability in NSTI cohorts, who often present with organ dysfunction in septic shock. Despite being a single-centre study, it is likely that with fewer exclusion criteria, our study more accurately reflects a real-world NSTI population and is more generalisable.

For patients transferred from other centres, the Alfred Hospital admission data does not represent the first time-point of their clinical admission and occurred a median of one day after primary admission. To be considered for transfer to our quaternary centre, patients would generally be required to demonstrate haemodynamic instability and/or progressive necrosis, and commonly require intensive care unit admission. The Alfred Hospital surgical teams generally recommend primary debridement of suspected NSTI at the referring centre prior to transfer where possible. Whether intubated to facilitate resuscitation or routinely at the time of primary surgical intervention at the referring hospital, these patients often remain intubated for transfer. Presence of an endotracheal tube raises the mSOFA score, and may over-estimate the acuity level of our cohort. On the other hand, having received initial treatment with surgery and antibiotics at the primary hospital and gaining a degree of source control, the use of Alfred admission data may under-estimate the initial acuity of transferred patients. The use of the intensive care and hyperbaric services and

the plastics surgery databases for identification of patients may have resulted in a number of Alfred NSTI patients not being included in the study sample, including those with NSTI of the trunk (general surgery or urology) or head and neck, who required neither intensive care support nor reconstructive surgical intervention. This may result in an over-estimate of the acuity level of our cohort. These factors did not appear to have a significant impact on the results, which remained consistent with those reported in the original NICCE validation study.

Meeting NICCE criteria of 'success' was associated with resource utilisation, and was consistent across all measures (ventilator-days, ICU-days, length of hospital stay). The NICCE could therefore be used in future studies to assess cost-efficacy of study interventions. Once surgical control and haemodynamic stability are achieved, and hyperbaric treatment is complete, patients often return to their primary hospital resulting in missing data for subsequent time-points in our analysis. This study demonstrates that NICCE is a feasible endpoint to measure with minimal missing data.

The NICCE does not apply weighting to its components. The debridements measure provides equivalent value to the survival measure, although it seems likely that patients and clinicians would place much higher value in the latter component. Similarly, NICCE does not measure long term comorbidity or functional status to provide a quality-of-life measure. The NICCE may therefore not be a good tool for the study of patient-centered outcomes. The NICCE was developed without patient consultation and future developments of NSTI outcome scoring might usefully include establishment of a patient advisory group to guide the inclusion of patient reported outcome measures.

The mortality rate presented in this study of 8.9% at 28 days appears at first glance to be an improvement from the overall mortality of 14.4% reported in a 2015 study at the same centre,[5] and to our knowledge is the lowest published mortality rate for NSTI outside of large-scale registry-based studies. This six percent improvement in survival needs to be interpreted with caution, however, as survival data was not collected beyond 28 days in this study (as required by NICCE), whereas in the previous study all known deaths at the time of data collection were included. Survivors of NSTI are known to have an ongoing increased risk of mortality subsequent to the early phases of the disease, as evidenced by all-cause mortality rates of 19% at 30 days, 25% at 90 days and 30% at one year reported in a recent, large, prospective multi-centre study.[18] A further potential limitation to the survival estimate in our study is the assumption that participants who were repatriated to their referring hospital with ward level care prior to 28 days survived. If inaccurate this would falsely elevate the survival rate. A follow up study at our centre could clarify this trend toward improved survival over time, which may be attributed to better awareness and management of sepsis, high performing intensive care support, and consolidation of expertise in a high-volume centre. The routine use of HBOT in NSTI patients at our centre may also contribute to the high survival rates. This hypothesis is strengthened by a contemporary (2023) study which demonstrated almost identical mortality rates to ours of 9% in a group receiving HBOT,[13] but this is an area which requires further research. Current reporting of survival in NSTI is inconsistent.[19] A 2021 meta-analysis found that whilst mortality outcomes were universally reported, the time from admission to death was not.[12] It is critical that a Core Outcome Set for NSTI is developed for consistent reporting.[5]

The restriction of study populations to higher severity cases could further enable assessment of efficacy. In modern critical care medicine, improved outcomes of patients have paradoxically increased the degree of difficulty to demonstrate statistically significant outcomes for individual interventions.[20] One strategy has been development of large, multicentre trials that provide high quality evidence for the primary outcomes, but may be affected by limited generalisability and exploration of secondary outcomes. For example, the CRASH-2 trial enrolled over 20,000 patients to demonstrate a relative mortality benefit of 9% in favour of patients receiving tranexamic acid after trauma.[21] However, restricting the population to a clinically relevant cohort of patients with acute traumatic coagulopathy enabled power to detect a similar relative difference in outcomes using a sample size of 1300 patients.[22] Similarly, the population of patients with mSOFA ≥ 3 would seem to represent the more clinically important cohort as evidenced by differences in endpoints (Table 2).

Conclusion

The NICCE endpoint of 'success' provides a higher event rate than mortality alone, particularly among patients with higher disease severity as measured by the mSOFA score on admission. The resultant higher discriminative ability of NICCE should enable more accurate identification of patients who had higher risk of adverse outcomes, thereby enhancing the feasibility of interventional trials. The NICCE endpoint should be considered for future studies in the NSTI population. Adaptation of the weighting of components with patient input should be considered and evaluated alongside addition of specific patient-centered outcome measures to further improve correlation of a useful research outcome measures with clinically meaningful outcomes.

References

1 Khamnuan P, Chongruksut W, Jearwattanakanok K, Patumanond J, Tantraworasin A. Necrotizing fasciitis: epidemiology and clinical predictors for amputation. Int J Gen Med. 2015;8:195–202. doi: 10.2147/IJGM.S82999. PMID: 25999758. PMCID: PMC4437611.

2 Endorf FW, Klein MB, Mack CD, Jurkovich GJ, Rivara FP. Necrotizing soft-tissue infections: differences in patients treated at burn centers and non-burn centers. J Burn Care

Res. 2008;29:933–8. doi: 10.1097/BCR.0b013e31818ba112. PMID: 18997557. PMCID: PMC3042354.
3 Naseer U, Steinbakk M, Blystad H, Caugant DA. Epidemiology of invasive group A streptococcal infections in Norway 2010–2014: a retrospective cohort study. Eur J Clin Microbiol Infect Dis. 2016;35:1639–48. doi: 10.1007/s10096-016-2704-y. PMID: 27311458.
4 Stevens DL, Bryant AE. Necrotizing soft-tissue infections. N Eng J Med. 2017;377:2253–65. doi: 10.1056/NEJMra1600673. PMID: 29211672.
5 Devaney B, Frawley G, Frawley L, Pilcher DV. Necrotising soft tissue infections: the effect of hyperbaric oxygen on mortality. Anaesth Intensive Care. 2015;43:685–92. doi: 10.1177/0310057X1504300604. PMID: 26603791.
6 Giuliano A, Lewis F Jr, Hadley K, Blaisdell FW. Bacteriology of necrotizing fasciitis. Am J Surg. 1977;134:52–7. doi: 10.1016/0002-9610(77)90283-5. PMID: 327844.
7 Endorf FW, Supple KG, Gamelli RL. The evolving characteristics and care of necrotizing soft-tissue infections. Burns. 2005;31:269–73. doi: 10.1016/j.burns.2004.11.008. PMID: 15774280.
8 Bucca K, Spencer R, Orford N, Cattigan C, Athan E, McDonald A. Early diagnosis and treatment of necrotizing fasciitis can improve survival: an observational intensive care unit cohort study. ANZ J Surg. 2013;83:365–70. doi: 10.1111/j.1445-2197.2012.06251.x. PMID: 22989238.
9 Boyer A, Vargas F, Coste F, Saubusse E, Castaing Y, Gbikpi-Benissan G, et al. Influence of surgical treatment timing on mortality from necrotizing soft tissue infections requiring intensive care management. Intensive Care Med. 2009;35:847–53. doi: 10.1007/s00134-008-1373-4. PMID: 19099288.
10 Kobayashi L, Konstantinidis A, Shackelford S, Chan LS, Talving P, Inaba K, et al. Necrotizing soft tissue infections: delayed surgical treatment is associated with increased number of surgical debridements and morbidity. J Trauma. 2011;71:1400–5. doi: 10.1097/TA.0b013e31820db8fd. PMID: 21768906.
11 Hua C, Bosc R, Sbidian E, De Prost N, Hughes C, Jabre P, et al. Interventions for necrotizing soft tissue infections in adults. Cochrane Database Sys Rev. 2018;5(5):CD011680. doi: 10.1002/14651858.CD011680.pub2. PMID: 29851032. PMCID: PMC6494525.
12 Hedetoft M, Bennett MH, Hyldegaard, O. Adjunctive hyperbaric oxygen treatment for necrotising soft-tissue infections: A systematic review and meta-analysis. Diving Hyperb Med. 2021;51:34–43. doi: 10.28920/dhm51.1.34-43. PMID: 33761539. PMCID: PMC8081587.
13 Hedetoft M, Madsen MB, Hyldegaard O. Hyperbaric oxygen treatment in the management of necrotising soft-tissue infections: results from a Danish nationwide registry study. BMJ Open. 2023;13:e066117. doi: 10.1136/bmjopen-2022-066117. PMID: 36813488. PMCID: PMC9950903.
14 Levett D, Bennett MH, Millar I. Adjunctive hyperbaric oxygen for necrotizing fasciitis. Cochrane Database Sys Rev. 2015;1:CD007937. doi: 10.1002/14651858.CD007937.pub2. PMID: 25879088. PMCID: PMC6516968.
15 Memar MY, Yekani M, Alizadeh N, Baghi HB. Hyperbaric oxygen therapy: antimicrobial mechanisms and clinical application for infections. Biomed Pharmacother. 2019;109:440–7. doi: 10.1016/j.biopha.2018.10.142. PMID: 30399579.
16 Bulger EM, May A, Dankner W, Maislin G, Robinson B, Shirvan A. Validation of a clinical trial composite endpoint for patients with necrotizing soft tissue infections. J Trauma Acute Care Surg. 2017;83:622–7. doi: 10.1097/TA.0000000000001564. PMID: 28538644.
17 Cribb BI, Wang MTM, Kulasegaran S, Gamble GD, MacCormick AD. The SIARI Score: a novel decision support tool outperforms LRINEC score in necrotizing fasciitis. World J Surg. 2019;43:2393–400. doi: 10.1007/s00268-019-05061-4. PMID: 31214830.
18 Hedetoft M, Madsen MB, Madsen LB, Hyldegaard O. Incidence, comorbidity and mortality in patients with necrotising soft-tissue infections, 2005-2018: a Danish nationwide register-based cohort study. BMJ Open. 2020;10(10):e041302. doi: 10.1136/bmjopen-2020-041302. PMID: 33067303. PMCID: PMC7569942.
19 Wackett J, Devaney B, Chau R, Ho J, King N, Grewal J, et al. Reported outcome measures in necrotising soft tissue infections: a systematic review. Diving Hyperb Med. 2024;54:47–56. doi: 10.28920/dhm54.1.47-56. PMID: 38507909.
20 Hauser CJ, Boffard K, Dutton R, Bernard GR, Croce MA, Holcomb JB, et al. Results of the CONTROL trial: efficacy and safety of recombinant activated Factor VII in the management of refractory traumatic hemorrhage. J Trauma. 2010;69:489–500. doi: 10.1097/TA.0b013e3181edf36e. PMID: 20838118.
21 CRASH-2 trial collaborators; Shakur H, Roberts I, Bautista R, Caballero J, Coats T, et al. Effects of tranexamic acid on death, vascular occlusive events, and blood transfusion in trauma patients with significant haemorrhage (CRASH-2): a randomised, placebo-controlled trial. Lancet. 2010;376(9734):23–32. doi: 10.1016/S0140-6736(10)60835-5. PMID: 20554319.
22 Mitra B, Bernard S, Gantner D, Burns B, Reade MC, Murray L, et al. Protocol for a multicentre prehospital randomised controlled trial investigating tranexamic acid in severe trauma: the PATCH-Trauma trial. BMJ Open. 2021;11(3):e046522. doi: 10.1136/bmjopen-2020-046522. PMID: 33722875. PMCID: PMC7970250.

Conflicts of interest and funding: nil

Submitted: 28 December 2023
Accepted after revision: 28 June 2024

The role of hyperbaric oxygen treatment in the management of spondylodiscitis

Kübra Canarslan Demir[1], Burak Turgut[1], Gözde B Sariyerli Dursun[1], Fatma S Konyalioğlu[2], Taylan Zaman[1]

[1] *Department of Underwater and Hyperbaric Medicine, University of Health Sciences, Gulhane Training and Research Hospital, Ankara, Turkey*
[2] *Department of Public Health, University of Health Sciences, Gulhane Training and Research Hospital, Ankara, Turkey*

Corresponding author: *Dr Kübra Canarslan Demir, SBÜ-Gülhane Eğitim ve Araştırma Hastanesi, Sualtı Hekimliği ve Hiperbarik Tıp Kliniği, Etlik/Ankara, Turkey*
drcanarslan@hotmail.com

Keywords
Discitis; Osteomyelitis; Pain; Recovery of function; Safety; Visual analog scale

Abstract
(Canarslan Demir K, Turgut B, Sariyerli Dursun GB, Konyalioğlu FS, Zaman T. The role of hyperbaric oxygen treatment in the management of spondylodiscitis. Diving and Hyperbaric Medicine. 2024 30 September;54(3):162–167. doi: 10.28920/dhm54.3.162-167. PMID: 39288919.)
Introduction: This study analysed treatment outcomes in a patient cohort diagnosed with spondylodiscitis, who received adjunct hyperbaric oxygen treatment (HBOT) in addition to antibiotic therapy at our clinic. Important considerations included the timing of HBOT initiation on treatment success, and recurrence rates.
Methods: We retrospectively reviewed the records of all patients diagnosed with spondylodiscitis who received HBOT at the Underwater and Hyperbaric Medicine Clinic in Gulhane Training and Research Hospital, between 1 November 2016 and 25 October 2022. The patients received HBOT at 243.2 kPa for a total of 120 minutes per session, once daily for five days a week for a total of 30 sessions.
Results: Twenty-five patients with spondylodiscitis were evaluated before and after combination HBOT and targeted antibiotic treatment. After treatment, patients had lower median (range) visual analogue pain scores (8 [4–10] vs 3 [0–7], $P < 0.001$) and C-reactive protein (22.3 [4.3–79.9] mg·L^{-1} vs 6.8 [0.1–96.0] mg·L^{-1}, $P = 0.002$) and lower mean (standard deviation) white blood cell counts (8.8 [3.5] x 10^{9}·L^{-1} vs 6.1 [1.6] x 10^{9}·L^{-1}, $P = 0.002$). When patients were examined (median) 48 months (2–156 months) after the completion of treatment, there were no persistent cases of spondylodiscitis.
Conclusions: Combination HBOT with targeted antibiotic therapy effectively managed our cohort of patients diagnosed with spondylodiscitis. Hyperbaric oxygen treatment was safe, with no complications experienced. Moreover, HBOT may have helped to eliminate persistence and recurrence of symptoms with long term follow-up. A randomised controlled study with a larger number of patients is needed for more definitive conclusions.

Introduction

Spondylodiscitis is a clinical condition resulting from the infection of the intervertebral disc and adjacent vertebral structures.[1] The terms 'vertebral osteomyelitis', 'spinal discitis', 'disc infection', and 'spondylodiscitis' are used interchangeably due to the frequent involvement of both the disc and vertebrae and the difficulty in distinguishing between them.[2] Risk factors for spondylodiscitis include intravenous drug use, infective endocarditis, degenerative spinal disease, prior spinal surgery, diabetes mellitus, corticosteroid use, and other immunosuppressive conditions.[3] Between 1998 and 2013, the annual incidence of hospital admissions for spondylodiscitis in the United States increased from 2.9 per 100,000 to 5.4 per 100,000.[3] This situation prolongs the hospitalisation period of patients and creates a serious burden on both the patient and the healthcare system.[3]

Hyperbaric oxygen treatment (HBOT) is an intervention in which patients breathe 100% oxygen in a hyperbaric chamber that is pressurised to higher than atmospheric pressure (101.3 kPa). This elevates tissue oxygen levels in partially ischaemic and hypoxic tissues which enhances oxygen-dependent leukocyte functions by promoting the production of hydrogen peroxide and superoxide.[4] Hyperoxia also supports osteogenesis and neovascularization to replace damaged tissue with healthy bone. Neovascularisation facilitates the entry of immune cells, antibodies, and antibiotics into the infected area, while HBOT also promotes the removal of bone debris by improving osteoclastic activity.[4] Several publications suggest the beneficial effects of HBOT in the treatment of spinal infections.[5–8] However, randomised controlled studies specifically examining the effectiveness of HBOT in managing these clinical conditions have not yet been conducted.

This study retrospectively assessed treatment outcomes in a patient cohort diagnosed with spondylodiscitis, who received adjunct HBOT in addition to antibiotic therapy at our clinic.

Methods

The study protocol was approved by the Gulhane Training and Research Hospital Clinical Research Ethics Committee with decision number 2023/73 on 12 April 2023.

We retrospectively reviewed the records of all patients diagnosed with spondylodiscitis who received HBOT at the Underwater and Hyperbaric Medicine Clinic in Gulhane Training and Research Hospital between 1 November 2016 and 5 October 2022.

The following factors were recorded from patient files and the hospital automation system: age, gender, elapsed time between diagnosis and HBOT, localisation, comorbidities, medications used, number of HBOT sessions received, 10-point visual analog scale (VAS) for pain before and after HBOT, white blood cell count (WBC), C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), magnetic resonance imaging (MRI) results, and whether the disease recurred during patient follow-up. Additionally, any complications that occurred during therapy were noted.

The diagnosis of discitis was made when all three of the following criteria were present:

- Clinical symptoms and findings including recurrent low back pain, decreased spinal mobility, paravertebral muscle spasm, and positive bed-shaking test.
- Laboratory findings of elevated ESR and CRP values.
- MRI findings consistent with discitis being decreased signal intensity on T1-weighted images, increased signal intensity in both the disc space and adjacent vertebral bodies on T2-weighted images, and enhancement of the same areas with gadolinium.

In addition to analgesic therapy, an appropriate antimicrobial regimen was chosen for all patients based on the results of antimicrobial susceptibility tests and the recommendations of the infectious diseases specialist.

The patients received HBOT at 243.2 kPa for a total of 120 minutes per session, once daily for five days a week for 30 sessions. Once HBOT was completed, antibiotic treatment was stopped. Patients were mobilised with lumbo-sacral corsets when they felt comfortable.

STATISTICAL ANALYSIS

Data analysis was performed with the IBM® SPSS 25.0 program. Categorical variables are reported as numbers and percentages, while numerical variables are reported as mean (standard deviation [SD]) or median (range). The normality of numerical variables was checked using the Shapiro-Wilk test, histograms, and probability plots. The homogeneity of variances between groups was evaluated using Levene's test. For independent group comparisons of data exhibiting a normal distribution, the independent samples *t*-test was used. For independent group comparisons of non-normal data, the Mann-Whitney U test was used. For dependent group comparisons of data following a normal distribution, the paired samples *t*-test was used. For dependent group comparisons of non-normal data the Wilcoxon signed-rank test was used. In all analyses, a *P*-value of less than 0.05 was considered statistically significant. Because the time between diagnosis and HBOT did not follow a normal distribution, it was categorised based on a median value of 14 days as a cut-off point. The patients with a starting time of HBOT exceeding 14 days were grouped as 'long', while those with a starting time of 14 days or less were grouped as 'short'.

Table 1
Demographics, comorbidity and medication in the study cohort; BMI – body mass index; SD – standard deviation

Characteristic	***n* (%)**
Female	13 (52)
Male	12 (48)
Comorbidity absent	5 (20)
Comorbidity present	20 (80)
Two or fewer medications	9 (36)
More than two medications	16 (64)
Characteristic	**Mean (SD)**
Age (years)	52.9 (12.8)
BMI ($kg \cdot m^{-2}$)	28.3 (2.6)

Table 2
Disease and treatment characteristics in the study cohort; HBOT – hyperbaric oxygen treatment; MRI – magnetic resonance imaging

Etiology, *n* = 19, *n* (%)	
Vertebral tuberculosis	1 (5.27)
Previous vertebral surgery	18 (94.73)
Localisation, *n* = 24, *n* (%)	
Cervical	2 (8.3)
Lumbar 1-2/2-3/3-4	8 (33.3)
Lumbar 4-5/5-S1	10 (41.7)
Multiple disc involvement	4 (16.7)
Regression on MRI, *n* = 13, *n* (%)	
Absent	3 (23.1)
Present	10 (76.9)
Other parameters, Median (range)	
Time from diagnosis to initiation of HBOT (days)	14 (1–210)
Follow-up period (months)	48 (2–156)
Number of sessions	30 (10–40)

Table 3

Comparison of pain scores and laboratory parameters of patients before and after treatment; CRP – C-reactive protein; ESR – erythrocyte sedimentation rate; HBOT – hyperbaric oxygen treatment; SD – standard deviation; VAS – visual analog scale, WBC – white blood cells

Outcome measure	*n*	Before treatment Median (range)	After treatment Median (range)	*P*
Pain VAS (0–10)	25	8 (4–10)	3 (0–7)	< 0.001
CRP ($mg \cdot L^{-1}$)	24	22.3 (4.3–79.9)	6.8 (0.1–96.0)	0.002
ESR ($mm \cdot h^{-1}$)	16	32.0 (8.0–123.0)	35.0 (1.0–94.0)	0.171
Outcome measure	***n***	**Before treatment Mean (SD)**	**After treatment Mean (SD)**	***P***
WBC (cells x $10^9 \cdot L^{-1}$)	24	8.8 (3.5)	6.1 (1.6)	< 0.002

Results

Twenty-five patients with spondylodiscitis were evaluated before and after HBOT. Patients used antibiotics for an average of six weeks along with HBOT. Approximately half of the patients (n = 12) were over 50 years of age. Twenty patients (80%) had comorbid conditions. Diabetes mellitus was present in eight patients (32%), and ten patients (40%) were hypertensive. No patient experienced complications related to HBOT during the treatment sessions. The descriptive characteristics of the patients are reported in Tables 1 and 2.

At the end of the HBOT sessions, it was observed that patients had significantly lower pain VAS scores, WBC counts, and CRP levels compared to the measurements taken before treatment (Table 3).

The influence of the timing of initiation of HBOT on treatment outcomes was evaluated using VAS data collected pre- and post-treatment. No significant difference was found in the VAS change between the 'short' or 'long' groups, defined by the timing of initiation treatment.

Magnetic resonance imaging findings of 13 patients before and a median one month (range 0–24 months) after HBOT were compared revealing that 10/13 patients showed regression of the lesion. While no change was detected in the MRI findings of two patients one month after HBOT, no signs of spondylodiscitis were found in their MRIs taken 36 months later. The third patient had no other MRI scans.

When patients were examined 48 months (2–156 months) after the completion of HBOT, all 25 had resolution of their back pain and were clinically well.

Discussion

Pain is the most common symptom in patients with spondylodiscitis and is often disproportionate to clinical findings.[9,10] In our study, the mean VAS score of our patients was 8, which is not surprising. The VAS score of our patients decreased to 3 after 30 sessions of HBOT. Similarly In another study VAS scores in spondylodiscitis patients decreased from 8.8 to 2.2 after one month of combined HBOT and antibiotic therapy.[6] The combination of HBOT and antibiotic treatment may be beneficial in reducing the pain of spondylodiscitis patients.

Accurate diagnosis depends on a combination of clinical, laboratory, and imaging findings. High ESR and CRP values, as well as typical changes observed in MRI, are important parameters in establishing the diagnosis. They are used to follow the course of spondylodiscitis and to monitor the response to treatment.[11–14] A decrease in CRP values is significantly associated with clinical improvement. Other studies conclude HBOT reduces CRP in patients with spondylodiscitis.[6,15] In our study group, the average initial CRP level was high at 22.3 $mg \cdot L^{-1}$ but decreased to 6.8 $mg \cdot L^{-1}$ after treatment.

In recent years, MRI has become the preferred imaging modality for the diagnosis of spondylodiscitis due to its reported sensitivity and specificity of over 92%.[13,16] Others have shown that HBOT reduces inflammatory findings in MRI.[6,15] In our study, when comparing the pre-treatment and 3–4 months post-treatment MRI scans of 13 patients, a radiological reduction-healing in inflammatory appearance was observed. However, due to the limited number of patients with MRI scans both before and after HBOT, we were unable to perform statistical analysis in this regard. The improvement in MRI in 10 out of 13 patients suggested that our combination treatment might be beneficial. Studies with a larger number of patients are needed to reach definitive conclusions. No change was detected in the MRI findings of two patients one month after HBOT. However, no signs of spondylodiscitis were found in their MRIs taken 36 months later. We believe this may be related to the late reflection of clinical improvement on MRI, as reported elsewhere.[17]

The key principles for successful treatment of spinal infections include antibiotic therapy to eradicate the underlying infection, debridement of the spinal canal in the presence of neurological deficits or epidural abscess,

and fixation of the affected segment to maintain or restore spinal stability. Long-term parenteral antibiotic therapy and immobilisation adversely affect quality of life while significantly increasing the cost of care.[18,19] Additionally, major complications (e.g., colitis, kidney failure, allergic reactions) have been reported as side effects of long-term antibiotic therapy.[20] The duration of antibiotic therapy recommended in the literature for the treatment of spondylodiscitis varies. Repeated laboratory markers and the patient's clinical response are key parameters in determining the exact duration of antibiotic therapy.[11,21]

There are other studies where HBOT was used in addition to antibiotics in the treatment of spondylodiscitis.[6,15] In one of these,[15] at the end of HBOT or within the first month of follow-up, MRI revealed sufficient healing of the infection in 12 out of 13 patients. The author argued that HBOT may be beneficial in long hospitalisation stays, repeated surgeries, and morbidities.[15] In the other,[6] 22 spondylodiscitis patients were treated with antibiotics and HBOT, and they achieved infection control and recovery in all patients with a no recurrence. The authors concluded that HBOT is a beneficial adjunctive therapeutic measure in the management of spondylodiscitis.[6] In another relevant study, HBOT was administered in the treatment of neurosurgical infections after brain and spinal cord surgery.[7] Five out of seven patients with osteomyelitis and wound infections following spinal surgery showed improvement with combined treatment without the removal of foreign bodies.

Unfortunately, our study did not provide the opportunity to compare the group that received antibiotics and HBOT with a control group that received only antibiotics. However, a multicenter observational prospective study in which patients were treated only with antibiotics reported an average antibiotic duration of 14.7 weeks.[22] In another study involving 110 patients, the mean total duration of antibiotic therapy was 103.0 (standard deviation 40.4) days, with a range of 42 to 285 days.[23] Although our study recommended 40 sessions of HBOT, patients actually received 30 sessions. The results presented here represent the follow-up outcomes of our combination treatment, administered over an average period of six weeks. This combination therapy was associated with a statistically significant decrease in CRP levels, and the inflammatory changes on MRI decreased. This period was shorter than the periods cited above for antibiotic treatment alone. We believe that earlier improvement in WBC, CRP and MRI findings may be attributed to the adjunctive effects of HBOT, which is being used in addition to medical therapy in the treatment of various infections in bone and soft tissues.[4,7,23–25]

Upon examining our patients approximately 48 months after treatment, we observed no spondylodiscitis recurrence, and none of the patients reported experiencing back pain. In a study that reported long-term outcomes of vertebral osteomyelitis in 263 patients with 6.5 years of follow-up, relapse, persistent symptoms, and surgery rates were 14%, 31%, and 43%, respectively.[26] In another study involving 260 patients with vertebral osteomyelitis with long-term follow-up, neurological deficits and persistent back pain were seen in 16% and 32% of cases, respectively.[27] In our cohort, an absence of symptom persistence or recurrence was observed. Adjunctive HBOT may have contributed to these results.

Most infected tissues, including bones, are hypoxic due to ischaemia secondary to inflammation-induced tissue oedema.[25,28] Adequate oxygen delivery to ischaemic tissue is important for healing and mitigating infection.[21,29] In spondylodiscitis, oxygenation decreases due to inflammation. The bactericidal capacity of leukocytes is significantly impaired in a hypoxic environment.[4,30] Hyperbaric oxygen has been shown to increase oxygen tension in infected tissues, including bone.[29] Increased oxygen levels in ischaemic tissues stimulate the bactericidal effect of white blood cells. Hyperbaric oxygen also inhibits the growth of aerobic and facultative anaerobic bacteria by inducing various metabolic effects related to the synthesis of proteins, nucleic acids and essential cofactors of metabolic reactions.[24] Oxygen-based free radicals oxidise proteins and membrane lipids, cause DNA damage and inhibit metabolic functions essential for (bacterial) growth.[4] Lack of adequate vascular supply to the adult disc reduces the ability of the patient's immune system to fight infection.[21] Therefore, angiogenesis is of critical importance in wound healing. It has been shown that HBOT reduces ischaemia in tissues by inducing the formation of new capillaries.[31] Improved vascularity not only improves tissue oxygen tension and host defense but also facilitates the entry of leukocytes, antibodies, and antibiotics into the infected area.[25]

This study has several limitations. Firstly, our study is retrospective in nature, without a control group. We cannot confidently attribute the apparent benefit ascribed to adjunctive HBOT without a control group of similar patients to compare with. Our analysis was based on a limited number of patients. Additionally, our patients were a heterogeneous group in terms of age, infective organism, co-morbidities, and surgery. Despite the aforementioned limitations, this cohort of patients responded well to the combination therapy. Stronger conclusions would require more rigorous trials.

Conclusions

These results demonstrate that in our cohort of patients, the combination of HBOT and targeted antibiotic therapy was effective in the management of spondylodiscitis. Hyperbaric oxygen may have helped to eliminate the persistence and recurrence of symptoms over an average 48-month follow-up. Additionally, none of our patients experienced complications during treatment. Randomised controlled

studies with a larger number of patients are needed to reach more definitive conclusions.

References

1 Mandell GL, Dolin R, Bennett JE. Mandell, Douglas, and Bennett's principles and practice of infectious diseases. 7th ed. London: Churchill Livingstone; 2010.

2 Colmenero JD, Jiménez-Mejías ME, Sánchez-Lora FJ, Reguera JM, Palomino-Nicás J, Martos F, et al. Pyogenic, tuberculous, and brucellar vertebral osteomyelitis: a descriptive and comparative study of 219 cases. Ann Rheum Dis. 1997;56:709–15. doi: 10.1136/ard.56.12.709. PMID: 9496149. PMCID: PMC1752312.

3 Issa K, Diebo BG, Faloon M, Naziri Q, Pourtaheri S, Paulino CB, et al. The epidemiology of vertebral osteomyelitis in the United States from 1998 to 2013. Clin Spine Surg. 2018;31(2):E102–8. doi: 10.1097/BSD.0000000000000597. PMID: 29135608.

4 Jain KK. HBO Therapy in infections. In: Jain KK, editor. Textbook of hyperbaric medicine. 6th ed. Cham: Springer; 2018. p. 155–69.

5 Ahmed R, Severson MA, Traynelis VC. Role of hyperbaric oxygen therapy in the treatment of bacterial spinal osteomyelitis. J Neurosurg Spine. 2009;10:16–20. doi: 10.3171/2008.10.SPI08606. PMID: 19119927.

6 Kutlay M, Colak A, Simsek H, Yildiz S, Topuz K, Kaya S, et al. Antibiotic and hyperbaric oxygen therapy in the management of post-operative discitis. Undersea Hyperb Med. 2008;35:427–40. PMID: 19175198.

7 Larsson A, Engström M, Uusijärvi J, Kihlström L, Lind F, Mathiesen T. Hyperbaric oxygen treatment of postoperative neurosurgical infections. Neurosurgery. 2002;50:287–95. PMID: 11844263.

8 Onen MR, Yuvruk E, Karagoz G, Naderi S. Efficiency of hyperbaric oxygen therapy in ıatrogenic spinal ınfections. Spine (Phila Pa 1976). 2015;40:1743–8. doi: 10.1097/BRS.0000000000001065. PMID: 26192727.

9 Pilgaard S. Discitis (closed space infection) following removal of lumbar inervertebral disc. J Bone Joint Surg Am. 1969;51:713–6. PMID: 4891161.

10 Wirtz DC, Genius I, Wildberger JE, Adam G, Zilkens KW, Niethard FU. Diagnostic and therapeutic management of lumbar and thoracic spondylodiscitis-an evaluation of 59 cases. Arch Orthop Trauma Surg. 2000;120(5–6):245–51. doi: 10.1007/s004020050457. PMID: 10853888.

11 Fouquet B, Goupille P, Jattiot F, Cotty P, Lapierre F, Valat JP, et al. Discitis after lumbar disc surgery. Features of "aseptic" and "septic" forms. Spine (Phila Pa 1976). 1992;17:356–8. doi: 10.1097/00007632-199203000-00019. PMID: 1566171.

12 Frank AM, Trappe AE. The role of magnetic resonance imaging (MRI) in the diagnosis of spondylodiscitis. Neurosurg Rev. 1990;13:279–83. doi: 10.1007/BF00346365. PMID: 2280839.

13 Bavinzski G, Schoeggl A, Trattnig S, Standhardt H, Dietrich W, Reddy M, et al. Microsurgical management of postoperative disc space infection. Neurosurg Rev. 2003;26:102–7. doi: 10.1007/s10143-002-0241-x. PMID: 12962295.

14 Jiménez-Mejías ME, de Dios Colmenero J, Sánchez-Lora FJ, Palomino-Nicás J, Reguera JM, García de la Heras J, et al. Postoperative spondylodiskitis: etiology, clinical findings, prognosis, and comparison with nonoperative pyogenic spondylodiskitis. Clin Infect Dis. 1999;29:339–45. doi: 10.1086/520212. Erratum in: Clin Infect Dis. 1999;29:1611. PMID: 10476739.

15 Körpınar Ş. Could hyperbaric oxygen be a solution in the treatment of spinal ınfections? Medicina (Kaunas). 2019;55(5):164. doi: 10.3390/medicina55050164. PMID: 31137457. PMCID: PMC6571771.

16 Szypryt EP, Hardy JG, Hinton CE, Worthington BS, Mulholland RC. A comparison between magnetic resonance imaging and scintigraphic bone imaging in the diagnosis of disc space infection in an animal model. Spine (Phila Pa 1976). 1988;13:1042–8. doi: 10.1097/00007632-198809000-00012. PMID: 3206298.

17 Cheung WY, Luk KD. Pyogenic spondylitis. Int Orthop. 2012;36:397–404. doi: 10.1007/s00264-011-1384-6. PMID: 22033610. PMCID: PMC3282872.

18 Duarte RM, Vaccaro AR. Spinal infection: state of the art and management algorithm. Eur Spine J. 2013;22:2787–99. doi: 10.1007/s00586-013-2850-1. PMID: 23756630. PMCID: PMC3843785.

19 Lener S, Hartmann S, Barbagallo GMV, Certo F, Thomé C, Tschugg A. Management of spinal infection: a review of the literature. Acta Neurochir (Wien). 2018;160:487–96. doi: 10.1007/s00701-018-3467-2. PMID: 29356895. PMCID: PMC5807463.

20 Mann S, Schütze M, Sola S, Piek J. Nonspecific pyogenic spondylodiscitis: clinical manifestations, surgical treatment, and outcome in 24 patients. Neurosurg Focus. 2004;17(6):E3. doi: 10.3171/foc.2004.17.6.3. PMID: 15636573.

21 Silber JS, Anderson DG, Vaccaro AR, Anderson PA, McCormick P. NASS. Management of postprocedural discitis. Spine J. 2002;2:279–87. doi: 10.1016/s1529-9430(02)00203-6. PMID: 14589480.

22 Gouliouris T, Aliyu SH, Brown NM. Spondylodiscitis: update on diagnosis and management. J Antimicrob Chemother. 2010;65(Suppl 3):iii11–24. doi: 10.1093/jac/dkq303. PMID: 20876624.

23 Legrand E, Flipo RM, Guggenbuhl P, Masson C, Maillefert JF, Soubrier M, et al. Management of nontuberculous infectious discitis. treatments used in 110 patients admitted to 12 teaching hospitals in France. Joint Bone Spine. 2001;68:504–9. doi: 10.1016/s1297-319x(01)00315-3. PMID: 11808988.

24 Whelan HT, Kindwall EP. Hyperbaric medicine practice. Flagstaff (AZ): Best Pub Co; 2017.

25 Fischer B. Handbook of hyperbaric oxygen therapy. Berlin: Springer-Verlag; 1988.

26 McHenry MC, Easley KA, Locker GA. Vertebral osteomyelitis: long-term outcome for 253 patients from 7 Cleveland-area hospitals. Clin Infect Dis. 2002;34:1342–50. doi: 10.1086/340102. PMID: 11981730.

27 Gupta A, Kowalski TJ, Osmon DR, Enzler M, Steckelberg JM, Huddleston PM, et al. Long-term outcome of pyogenic vertebral osteomyelitis: a cohort study of 260 patients. Open Forum Infect Dis. 2014;1(3):ofu107. doi: 10.1093/ofid/ofu107. PMID: 25734175. PMCID: PMC4324221.

28 Hunt TK, Hopf HW. Wound healing and wound infection. What surgeons and anesthesiologists can do. Surg Clin North Am. 1997;77:587–606. doi: 10.1016/s0039-6109(05)70570-3. PMID: 9194882.

29 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.

30 Mader JT, Brown GL, Guckian JC, Wells CH, Reinarz JA. A mechanism for the amelioration by hyperbaric oxygen of experimental staphylococcal osteomyelitis in rabbits. J Infect Dis. 1980;142:915–22. doi: 10.1093/infdis/142.6.915. PMID: 7462700.

31 Marx RE, Ehler WJ, Tayapongsak P, Pierce LW. Relationship of oxygen dose to angiogenesis induction in irradiated tissue. Am J Surg. 1990;160:519–24. doi: 10.1016/s0002-9610(05)81019-0. PMID: 2240387.

Conflicts of interest and funding: nil

Submitted: 25 July 2023
Accepted after revision: 7 June 2024

Methylphenidate and the risk of acute central nervous system oxygen toxicity: a rodent model and observational data in human divers

Ivan Gur[1], Yehuda Arieli[2], Yinnon Matsliah[3]

[1] *Rambam Medical Center, Haifa, Israel*
[2] *Israel Naval Medical Institute, Israel Defence Force Medical Corps, Haifa, Israel*
[3] *Department of Pediatric Neurology and Developmental Medicine, Carmel Medical Center, Haifa, Israel*

Corresponding author: *Dr Ivan Gur, Department of Internal Medicine C, Rambam Medical Center, 4 HaAlia Street, Haifa 3109601, Israel*
i_gur@rambam.health.gov.il

Keywords
ADHD; Attention deficit hyperactivity disorder; Diving; Hyperbaric oxygen; Seizures

Abstract
(Gur I, Arieli Y, Matsliah Y. Methylphenidate and the risk of acute central nervous system oxygen toxicity: a rodent model and observational data in human divers. Diving and Hyperbaric Medicine. 2024 30 September;54(3):168–175. doi: 10.28920/dhm54.3.168-175. PMID: 39288920.)

Introduction: The effects of methylphenidate, a stimulant often prescribed for the treatment of attention-deficit/hyperactivity disorder (ADHD), on the development of central nervous system oxygen toxicity (COT) have not been experimentally evaluated.

Methods: The records of all pure-oxygen-rebreather divers evaluated at our institution from 1975–2022 were assessed. Cases of COT were defined as a new onset of tinnitus, tunnel vision, myoclonus, headache, nausea, loss of consciousness, or seizures resolving within 15 minutes from breathing normobaric air, and matched 4:1 with similar controls. Any medications issued to the diver in the preceding three months, including methylphenidate, were recorded. In the animal arm of this study, male mice were exposed to increasing doses of methylphenidate orally, with subsequent exposure to hyperbaric O_2 until clinically evident seizures were recorded.

Results: Seventy-five cases of COT were identified in divers, occurring at a median of 80 (range 2–240) minutes after dive initiation at a median depth of 5 m (2–13). Hypercarbia was documented in 11 (14.7%) cases. Prescription of methylphenidate in the preceding three months was not associated with increased risk (OR 0.72, 95% CI 0.16–3.32) of COT. In mice, increasing methylphenidate exposure dose was associated with significantly longer mean COT latency time being 877 s (95% CI 711–1,043) with doses of 0 mg·kg^{-1}; 1,312 s (95% CI 850–1,773) when given 0.75 mg·kg^{-1}; and 1,500 s (95% CI 988–2,012) with 5 mg·kg^{-1} ($F = 4.635$, $P = 0.014$).

Conclusions: Observational human data did not demonstrate an association between methylphenidate and an increased risk of COT. Methylphenidate exposure in mice prolongs COT latency and may have protective effects against COT.

Introduction

First synthesised in 1944, methylphenidate has been at the heart of pharmacotherapeutic approaches in the treatment of attention deficit for the past half a century. Diagnosed in over 2% of the adult population in the industrialised world and over 7% of children, attention deficit disorders (ADD) are the most common neurobehavioral disorders. The quick rise in the diagnosis of attention-deficit/hyperactivity disorders (ADHD) throughout the western world during the past two decades has made methylphenidate among the ten most prescribed medications in young adults.[1] Limited data suggest the prevalence of both ADD and methylphenidate use to be significant in divers.[2,3] The reuptake inhibition of dopamine (as well as norepinephrine and to a lesser extent, an agonist effect on the serotonin 5HT1A receptor) is the main central nervous system (CNS) effect of methylphenidate. Clinically, neurostimulator effects predominate when methylphenidate is administered in both human and animal subjects. While generally considered safe in children and adults, even with coinciding epilepsy, these recommendations are based mainly on registry-based data.[4] Some observational studies showed a signal towards increased risk of seizures early after the initiation of methylphenidate.[5,6]

Correlated with cerebral size and complexity, mammals, and particularly humans, are at increased risk of CNS oxygen toxicity (COT). While specific presentations may vary, generalised seizures and loss of consciousness are the most feared complications. In view of the potential for fatal drowning after sudden incapacitation underwater, coupled with the yet to be fully understood effects of immersion on COT risk, the maximal inspired PO_2 exposure limits are usually set to 284–304 kPa (2.8–3 atmospheres absolute [atm abs]) in dry conditions,[7] and 142–182 Kp (1.4–1.8 atm abs) while submerged.[8] However, personal susceptibility to COT is highly variable. Circadian rhythm,

exertion, environmental conditions such as ambient temperature or darkness, CO_2 levels, and exposure to various substances like caffeine or phenylephrine are just some of the modifiable factors to have been shown to alter susceptibility to COT.[9]

The effects of methylphenidate on COT susceptibility have not been experimentally evaluated thus far. Currently considered safe, these guidelines are based solely on expert opinion.[10] A paucity of observational information and no systematically evaluated analyses have been published on the potential effects of methylphenidate on COT.[11]

We aimed to investigate the potential influence of methylphenidate exposure on COT in a rodent model, coupled with an observational analysis of a large cohort of professional oxygen and mixed gas divers.

Methods

The human study was approved by our Institutional Ethics Committee (approval #2280-2022). The requirement for consent was waived by the ethics committee due to the retrospective nature of this study. The animal use protocol prepared for this study was reviewed and approved by our AAALAC-accredited Institutional Animal Care and Use Committee (approval #03-3415).

HUMAN SUBJECTS AND CASE DEFINITION

The medical records of all pure-O_2 rebreather military divers evaluated by the Israeli Naval Medical Institute (INMI) from 1 January 1975 to 31 December 2022 were included. Medical evaluation conducted bi-annually, includes a full list of medications, past medical history, otolaryngological and ophthalmological exams, a full cardiological workup (including a resting electrocardiogram, examination by a board-certified cardiologist, and ergometry for all candidates over the age of 35), a chest X-ray once every five years, spirometry, urinalysis, and complete blood count. In addition, all incidents involving a mild to moderate suspicion of oxygen toxicity (the onset of at least one of the following symptoms: tinnitus, tunnel vision, myoclonus, new headache, or nausea) are mandatorily reported to INMI, with severe cases (involving loss of consciousness and/or seizures) actively investigated including a full forensic equipment evaluation by our specialised laboratory.

For the purposes of this study, to be defined as COT, any case must have: 1) occurred during a dive using a pure O_2 rebreather system; 2) manifested as a new onset of tinnitus, tunnel vision, myoclonus, headache, nausea, loss of consciousness or seizures only after the dive began; 3) resolved within 15 minutes when normobaric air was administered (in case of a seizure, this maximum recovery period referred to the tonic-clonic phase and excluded any postictal manifestations such as decreased consciousness or confusion); and 4) no other explanation (including hypoxia from any cause or trauma) was found on an active investigation. Controls were matched at a 4:1 ratio with divers from similar recruitment years to allow for maximal matching of potential confounders like age, diving experience, and exposure profiles. Medication exposure was determined by reviewing dispensary records, which due to the centralised (single payer and single provider) nature of military healthcare, meticulously document any medication issued, including those considered 'over the counter'. Any prescription given over the previous three-month period was recorded as positive for drug exposure (e.g., methylphenidate, acetaminophen, etc). During the study years, only immediate-release methylphenidate prescribed for the indication of ADHD was allowed. Other stimulants, as well as methylphenidate prescription for other indications (sleep disorders, off-label, etc.), were not permitted by military regulations in the study population.

ANIMALS AND PHARMACOTHERAPY

Mice were chosen as previous evidence suggests this species exhibits lower variability in COT latency when compared to other rodents, particularly rats.[12] Twenty male Institute of Cancer Research mice (mean 32.1 [SD 2.6] g, range 28–38 g), (Harlan Laboratories, Indianapolis, IN, USA) aged eight weeks were included in this study. Methylphenidate was given orally, after the following preparation: 1) Immediate-release methylphenidate was dissolved in 20 µL of sterile water to achieve a total administered dose of 0.75 $mg \cdot kg^{-1}$, previously demonstrated to correspond to human therapeutic doses, achieving the target plasma concentration of 6–10 $ng \cdot mL^{-1}$ within 15 minutes.[13] 2) Another dose of 5$mg \cdot kg^{-1}$ was chosen to investigate a potential dose effect. This higher concentration was chosen for being at the cusp of previously reported LD_{50} for mice[6] and pharmacodynamic investigations in both rodent and human models.[1,13] After examining different feeding methods, and striving to avoid gavage or the need for orogastric cannulation and the associated stress-related increase in the risk of COT,[9] dissolving the administered dose in peanut butter following 16 hours of starvation was found to be the most effective method of drug administration. This approach was supported by previous evidence of unaltered pharmacokinetics of methylphenidate with regards to feeding status.[12] This also allowed for standardisation of caloric intake prior to hyperbaric oxygen exposure, since reduced caloric intake and starvation were linked with decreased risk of COT.[7] Seizures were defined as the onset of regular tonic-clonic twitches (limbs, head, or tail). Any isolated myoclonia (limb twitch, tail hardening, etc) were disregarded.

HYPERBARIC CHAMBER SETUP AND EXPOSURE PROFILES

Video surveillance was continuous throughout the hyperbaric exposure, as were chamber conditions including temperature, humidity and O_2 concentration (Servomex®, Crowborough,

East Sussex, UK). Following the administration of dissolved immediate release methylphenidate or water (placebo) as described above, the mouse was placed in a well-ventilated cage, preventing heat and CO_2 accumulation as hypercarbia is a strong predictor of COT.[7] Compression was initiated 90 minutes after the drug was administered, allowing for peak blood concentration to be reached. Compression to 506 kPa (5 atm abs) was achieved over 10 minutes, followed by the saturation of the hyperbaric chamber with 100% O_2. This hyperbaric oxygen exposure was continued until seizures were observed, at which point air was flushed through the chamber and the chamber was decompressed to normobaric pressure at 101.3 kPa (1 atm abs) per minute. Chamber temperature was maintained at 25–30°C.

Five mice served as controls throughout the experiment (exposure group A). Eight were exposed to a placebo followed by 0.75 mg·kg^{-1} and then 5 mg·kg^{-1} of methylphenidate (group B). Seven were exposed to a placebo, followed by 5 mg·kg^{-1} and then 0.75 mg·kg^{-1} of methylphenidate (group C). The study design is presented in Figure 1. Exposures were spaced seven days apart, allowing for potential effects of previous hyperbaric exposure and the resultant decrease in seizure latency to wear off.[9]

STATISTICAL ANALYSIS

Human case-control study

Standard descriptive statistics were used to summarise population characteristics. The low prevalence of methylphenidate exposure in the human (observational) portion of this study allowed for the odds ratio (OR) to serve as a reasonable estimator of risk. Adjustment for possible interactions was achieved by constructing a multivariate logistic regression model using Pearl and Reed's method.[14]

Animal model

Fisher's least significant difference (LSD) correction was applied when applicable to adjust for multiple comparisons for the mice data. Analysis of variance was performed when comparing COT latency time after normal distribution was ascertained by means of QQ plot visual analysis as well as skewness and kurtosis ≤ |2|. A repeated measures general linear model was constructed to allow for within-group comparison of the effects of varying methylphenidate exposure on the same animal. Levene's test for homoscedasticity and an unpaired *t*-test were performed on exposure groups B and C in matching drug doses. Groups B and C were pooled (by drug dose) only if Levene's test yielded nonsignificant ($P > 0.01$) results. A 2-sided $P < 0.05$ was considered statistically significant for all tests. All calculations were performed using SPSS software version 24.0.

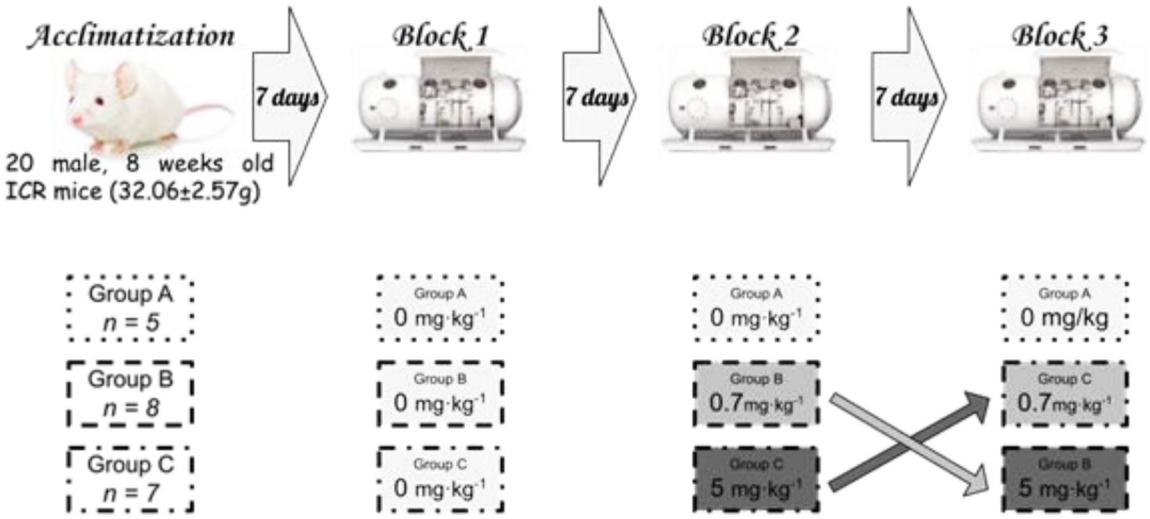
Results

HUMAN CASES AND CONTROLS

A total of 75 cases of COT in humans were identified as matching our case definition. The median latency time, defined as time from dive initiation to the appearance of symptoms, was 80 minutes (IQR 26–135) (range 2–240). The median bottom depth was 5 metres of sea water (IQR 4–6) and the median water temperature was 22°C

Figure 1

A schematic representation of the animal study protocol; in each block, mice were compressed to 506 kPa (5 atm abs) over 10 mins, followed by pure O_2 breathing until seizures were observed. All doses refer to methylphenidate administered orally. ICR – institute of cancer research



(IQR 18–25) (range 13–30). In 44 COT cases (58.7%) the diver was appropriately dressed as mandated by our diving physiology research laboratory guidelines.[15] Hypercarbia (mainly attributed to scrubber dysfunction) was recorded in 11 cases (14.7%). Diving profiles involved a constant depth ('square') in 42 cases (56%), whereas 20 (26.7%) involved a gradual ascent ('repet-up') and 13 (17.3%) were not homogeneous ('hang-off' or 'yo-yo' dives). Exertion was maximal (estimated above 15 metabolic equivalents) in 25 (33%) cases, moderate (fin swimming) in 32 (42.7%), and minimal (using a propulsion vehicle) in 18 (24%). These findings, along with a comparison to matched controls in baseline characteristics, are presented in Table 1 and Table 2.

Comparing cases of COT to controls, the OR for methylphenidate exposure at any dose during the antecedent three months (but not within 24 hours before diving) was 0.72 (95% CI 0.16–3.32). Adjusted for age, body mass index, diving experience, smoking, history of attention deficit disorder or allergic rhinitis, and recent (within three

Table 1

Baseline characteristics of central nervous system oxygen toxicity (COT) cases and matched controls; [1] Any tobacco use within prior six months; [2] History of mild vasomotor rhinitis or allergic rhinitis with no active disease or treatment during the past two years; [3] Attention deficit disorder, with or without hyperactivity, diagnosed by a certified psychiatrist/neurologist in accordance with DSM-V criteria, with no significant functional limitation or comorbidity, irrespective of the need for psychopharmacotherapy; [4] Asymptomatic mild impairments, such as kyphosis < 50 degrees, scoliosis < 20 degrees, over two years from simple uncomplicated fracture with no sequela, partial meniscectomy/meniscal tear with no sequelae or functional limitation and mild pes planus; [5] Refractive deficit of ± 1.75 diopter (spherical or astigmatism) or less, provided uncorrected visual acuity is 6/9 (20/30) or better; [6] Provided disease deemed inactive and serology negative for the past six months; [7] Varicocele or hydrocele provided no functional limitation is present (including surgery with no sequelae completed more than two years prior); [8] Any medications prescribed within the prior six months

Parameter	**Cases** ***n*** **= 75**	**Controls** ***n*** **= 300**	***P***
Age (years), Median (Range)	20 (18–27)	21 (18–27)	
18–21 years, *n* (%)	52 (69.3)	203 (67.7%)	0.782
22–25 years, *n* (%)	18 (24.0)	79 (26.3%)	0.680
> 25 years, *n* (%)	5 (6.7)	18 (6.0%)	0.830
Morphometrics, Mean (standard deviation)			
Height, cm	178.0 (5.3)	178.4 (5.8)	0.626
Weight, kg	75.4 (6.1)	76.6 (7.9)	0.198
Body mass index, $kg \cdot m^{-2}$	23.8 (1.3)	24.1 (1.9)	0.210
Total pure O_2 diving experience, *n* (%)			
< 100 hours	25 (33.3)	91 (30.3)	0.615
100–500 hours	30 (40.0)	103 (34.3)	0.359
> 500 hours	20 (26.7)	106 (35.3)	0.155
Background diagnoses, *n* (%)			
Smoking[1]	5 (6.7)	23 (7.7)	0.768
Allergic rhinitis/sinusitis[2]	2 (2.7)	3 (1.0)	0.260
Attention deficit[3]	7 (9.3)	36 (12.0)	0.517
Significant orthopedic history[4]	9 (12.0)	42 (14.0)	0.651
Refractive error/correction[5]	3 (4.0)	5 (1.7)	0.211
Celiac[6]	2 (2.7)	4 (1.3)	0.410
Varicocele[7]	3 (4.0)	7 (2.3)	0.423
Medications previously prescribed[8], *n* (%)			
Methylphenidate	2 (2.7)	11 (3.7)	0.672
Decongestants	7 (9.3)	22 (7.3)	0.562
Antihistamine	9 (12.0)	34 (11.3)	0.871
NSAIDs	22 (29.3)	95 (31.7)	0.696
Acetaminophen	46 (61.3)	178 (59.3)	0.752

Table 2

Central nervous system oxygen toxicity (COT) dive characteristics; [1] Estimated METs > 15; [2] see (Ofir et al. 2019)[15]; [3] Any indication of potential hypercarbia or CO_2 scrubbing dysfunction, including deficient soda lime, damaged scrubber container or any damaged one-way valves

Parameter	*n* (%)
Time of day	
05:00–22:59	32 (42.7)
23:00–04:59	43 (57.3)
COT latency, minutes	
< 60	28 (37.3)
60–119	21 (28.0)
120–179	15 (20.0)
≥ 180	11 (14.7)
Exertion	
Diver propulsion vehicle	18 (24.0)
Swimming	32 (42.7)
Maximal exertion[1]	25 (33.3)
Diving profile	
Square	42 (56.0)
Repet-up	20 (26.7)
Hang-off	13 (17.3)
Maximal depth, metres	
< 4	15(20.0)
4–6	43(57.3)
> 6	17(22.7)
Water temperature, °C	
< 19	22 (29.3)
19–23	22 (29.3)
> 23	31 (41.3)
Appropriate garment[2]	44 (58.7)
Other	
Hypercarbia[3]	11(14.7)

months) use of decongestants, antihistamines or analgesics the adjusted OR for methylphenidate exposure was 0.87 (95% CI 0.14–5.29), as presented in * Supplementary Table 1. Reviewing individual cases, no medications were used in the 24 hours preceding the COT event.

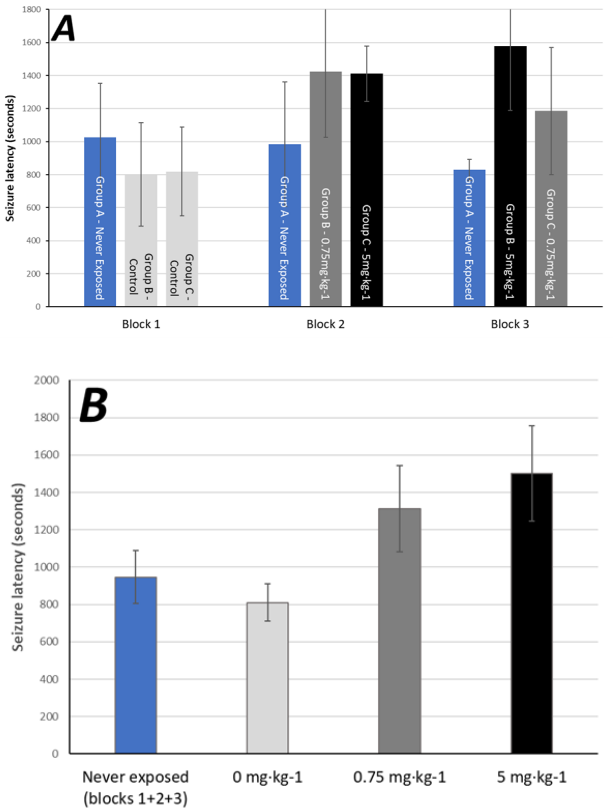
RODENT MODEL

Increasing methylphenidate exposure in mice was associated with significantly longer COT latency time (F = 4.635, $P = 0.014$). Pooling COT latency times by methylphenidate exposure dose showed a mean latency of 1,500 s (95% CI 988–2,012) when mice were given 5 mg·kg^{-1}, 1,312 s (95% CI 850–1,773) for 0.75 mg·kg^{-1}, 809 s (95% CI 607–1,010) for 0 mg·kg^{-1} (treatment group B and C) and 946 s (95% CI 660–1231) for controls (treatment group A). These are presented in Figure 2.

Post-hoc analysis indicated latency time to be significantly longer comparing the 5 mg·kg^{-1} dose to controls (877 s vs 1,500 s, LSD corrected Md 622 s [95% CI 183–1,061], $P = 0.006$), while comparing 0.75 mg·kg^{-1} to controls approached statistical significance (1,312 s vs 1,500 s, LSD corrected Md 434 s [95% CI -93–961], $P = 0.052$). These findings were unchanged when adjusted to animal weight and chamber temperature by constructing a general linear

Figure 2

A. Seizure latency with varying methylphenidate exposure; mean central nervous system oxygen toxicity (COT) latency time (from reaching chamber pressure of 506 kPa [5 atm abs] to the occurrence of tonic-clonic twitches) is presented by study group and block. B. Latencies are grouped by methylphenidate dose and also compared to mice never exposed to methylphenidate (study group A). Error bars represent 95% confidence intervals. The difference between average latency times for the 5 mg·kg^{-1} group compared with those of the 0 mg·kg^{-1} (control) group reached statistical significance ($P = 0.015$)



*** Footnote:** Supplementary Table 1 is available on the DHM Journal website.

Table 3

Hyperbaric oxygen-induced central nervous system oxygen toxicity (COT) seizure latency (seconds) in mice; Analysis of variance (ANOVA) was performed to investigate potential variability in average COT latency times between the different exposure groups, both when all cases were included and when only looking at the subset of mice treated with different doses of methylphenidate (groups B and C). A general linear model accounting for animal weight and chamber temperature is presented in the bottom portion. The righthand portion depicts post hoc analyses (corrected for multiple comparisons) investigating the difference in COT latency times between specific exposure groups, both overall and in the exposed animals (groups B and C). CI – confidence interval; MD – mean difference

Overall		Post hoc (corrected by Fisher's least significant difference)								
		0.75 vs 0 mg·kg^{-1}			5 vs 0 mg·kg^{-1}			5 vs 0.75 mg·kg^{-1}		
F	*P*	MD	95% CI	*P*	MD	95% CI	*P*	MD	95%CI	*P*
Analysis of variance, all cases included										
4.635	0.014	434.3	-4.78–873.38	0.052	622.83	183.75–1,061.92	0.006	188.53	318.48–695.54	0.460
Only treatment groups B and C (repeated measure analysis of variance)										
3.416	0.042	502.6	-49.07–1,054.27	0.073	691.13	139.46–1,242.81	0.015	188.53	363.14–740.21	0.494
General linear model adjusted for weight and chamber temperature										
4.308	0.018									

model (F = 4.308, *P* = 0.018). A repeated measure analysis of variance including only mice exposed to methylphenidate (treatment groups B and C) showed similar trends (F = 3.416; *P* = 0.042; post-hoc 5 mg·kg^{-1} vs control MD 691s, [95%CI 139–1,242], *P* = 0.015). Analysing mice unexposed to methylphenidate (treatment group A) showed no significant difference in COT latency (F = 0.756, *P* = 0.542). All measurements are presented in Table 3.

Discussion

Our analysis found no increased risk of COT to be associated with chronic methylphenidate exposure (with no documented use of the drug in the 24 hours before diving) in humans, albeit the low prevalence of methylphenidate exposure underpowered our ability to detect minor increases in risk. Moreover, since no divers reported taking any medications in the vicinity of documented COT events, our human data cannot shed light as to the short-term safety of methylphenidate use with regard to COT risk. Considering the likely underreporting in the highly motivated group of military divers, we deem the immediate exposure data (beyond pharmacy records) to be of low reliability. Finally, the low incidence of COT in human divers limits our ability to truly estimate the potential effect of methylphenidate withdrawal on the risk of COT.

The rodent model, undertaken in an attempt to bridge this knowledge gap, seems to establish a certain protective property of methylphenidate on the risk of COT. Our data suggests an association between methylphenidate dose and increasing COT latency.

Increased sympathomimetic activity may increase the risk of COT, as evident from the association between strenuous exercise, stress, circadian rhythm disturbance and hypothermia.[7] This notion is consistent with our findings of the epidemiological characteristics of documented COT events. Sympathomimetic agents, such as pseudoephedrine, were also shown in rodent models to increase the risk of COT.[9] The weak increase in CNS norepinephrine as a result of methylphenidate administration[16] is therefore an unlikely explanation for such COT protective effects.

We believe the mechanism may be related to increasing levels of dopamine. Methylphenidate was repeatedly shown to significantly increase dopamine levels in various brain regions in different rodent and primate models.[4,6,13,16] Striatal dopamine levels were previously shown to decrease under increasing partial pressures of oxygen, with critically low levels resulting in COT-induced seizures in rats.[17,18] Caffeine, a substance shown to increase striatal dopamine receptor availability, has been demonstrated to delay convulsions in rats.[19] Methylphenidate's dopaminergic effects thus may help explain its neuroprotective properties in delaying critical COT-inducing low levels of dopamine. Such a mechanism would not apply to prolonged use of other stimulants, e.g., methamphetamines, shown to cause degeneration of dopaminergic terminals in the striatum,[20] which might increase the risk of COT.

Of note, dopamine hypoactivity has been suggested as a potential pathophysiological pathway at the core of ADHD. For instance, expression of the dopamine transporter was shown (using single photon emission tomography) to be 70% higher in the striatum of patients with ADHD compared with controls.[21] Such a deficit may increase inappropriate connectivity to the prefrontal cortex. Treatment with methylphenidate has been shown to decrease the dopamine transporter density.[22] In other words, divers with ADHD who are currently not treated with methylphenidate (e.g., did not take this medicine in the hours leading to the dive) may thus have dopaminergic hypoactivity, previously shown to increase COT susceptibility.[7] Our data do not

suggest an increased risk of COT in patients with ADHD who are prescribed methylphenidate, all of whom stop the medication at least 24 hours before the dive. However, we are underpowered and methodologically ill equipped to answer this interesting question – namely, are patients on methylphenidate at increased risk of COT if they dive without this medication.

LIMITATIONS

This study has several important limitations. The relative rarity of COT confounded us to the case-control design in human subjects and only to pure O_2 rebreather divers. Since these systems are used almost exclusively by elite, very young, highly trained, and overwhelmingly male divers, the generalisability of our findings to other populations of mixed-gas divers or HBO patients is limited. The animal arm of this study is limited by the great differences between our rodent model and cerebral complexity in humans, easily evident by the immensely higher PO_2 needed to induce COT in our model animals. Importantly, we were unable to investigate the effects of immersion in our model animals. This is important since immersion is consistently shown to increase COT susceptibility, practically halving the COT threshold in humans.[7]

Conclusions

Observational human data suggests methylphenidate is not associated with an increased risk of COT. Methylphenidate exposure in mice increases COT latency, and may have protective effects against COT.

References

1 Storebø OJ, Pedersen N, Ramstad E, Kielsholm ML, Nielson SS, Krogh HB, et al. Methylphenidate for attention deficit hyperactivity disorder (ADHD) in children and adolescents – assessment of adverse events in non-randomised studies. Cochrane Database Syst Rev. 2018;5(5):CD012069. doi: 10.1002/14651858.CD012069.pub2. PMID: 29744873. PMCID: PMC6494554.

2 Van Wijk CH, Meintjes WAJ. Adult attention-deficit/hyperactivity disorder prevalence among commercial divers in South Africa. Diving Hyperb Med. 2020;50:164–7. doi: 10.28920/dhm50.2.164-167. PMID: 32557419. PMCID: PMC7481110.

3 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.

4 Leeman-Markowski BA, Adams J, Martin SP, Devinsky O, Meador KJ. Methylphenidate for attention problems in epilepsy patients: safety and efficacy. Epilepsy Behav. 2021;115:107627. doi: 10.1016/j.yebeh.2020.107627. PMID: 33360744. PMCID: PMC7884102.

5 Man KKC, Lau WCY, Coghill D, Besag FMC, Cross JH, Ip P, et al. Association between methylphenidate treatment and risk of seizure: a population-based, self-controlled case-series study. Lancet Child Adolesc Health. 2020;4:435–43. doi: 10.1016/S2352-4642(20)30100-0. PMID: 32450123.

6 NTP-CERHR monograph on the potential human reproductive and developmental effects of methylphenidate. NTP CERHR MON. 2005;(15):v, vii-I-2, II-xi-147. PMID: 17180168.

7 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.

8 Wingelaar TT, van Ooij P-JAM, 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.

9 Pilla R, Held HE, Landon CS, Dean JB. High doses of pseudoephedrine hydrochloride accelerate onset of CNS oxygen toxicity seizures in unanesthetized rats. Neuroscience. 2013;246:391–6. doi: 10.1016/j.neuroscience.2013.04.035. PMID: 23624060.

10 Querido AL, van Hulst RA. Diving and attention deficit hyperactivity disorder. Diving Hyperb Med. 2019;49:41–7. doi: 10.28920/dhm49.1.41-47. PMID: 30856666. PMCID: PMC6526049.

11 Van Wijk CH, Meintjes WAJ. Associations between adverse underwater events and ADHD diagnosis among military divers. J Atten Disord. 2021;25:848–50. doi: 10.1177/1087054719864654. PMID: 31319735.

12 Amarelle L, Quintela L, Hurtado J, Malacrida L. Hyperoxia and lungs: what we have learned from animal models. Front Med (Lausanne). 2021;8:606678. doi: 10.3389/fmed.2021.606678. PMID: 33768102. PMCID: PMC7985075.

13 Balcioglu A, Ren J-Q, McCarthy D, Spencer TJ, Biederman J, Bhide PG. Plasma and brain concentrations of oral therapeutic doses of methylphenidate and their impact on brain monoamine content in mice. Neuropharmacology. 2009;57(7-8):687–93. doi: 10.1016/j.neuropharm.2009.07.025. PMID: 19631228. PMCID: PMC2783341.

14 Pearl R, Reed LJ. On the rate of growth of the population of the United States since 1790 and its mathematical representation. Proc Natl Acad Sci U S A. 1920;6(6):275–88. doi: 10.1073/pnas.6.6.275. PMID: 16576496. PMCID: PMC1084522.

15 Ofir D, Yanir Y, Eynan M, Arieli Y. Evaluating the thermal protection provided by a 2–3 mm wet suit during fin diving in shallow water with a temperature of 16–20°C. Diving Hyperb Med. 2019;49:266–75. doi: 10.28920/dhm49.4.266-275. PMID: 31828745. PMCID: PMC7039775.

16 Cöngölolu A, Türkbay T, Doruk A, Topal T. Long-term methylphenidate treatment causes increased superoxide dismutase activity and unchanged lipid peroxidation in rat brain. Klinik Psikofarmakoloji Bülteni. 2006;2(16):79–83.

17 Lavoute C, Weiss M, Risso JJ, Rostain JC. Examination of the role of NMDA and GABAA receptors in the effects of hyperbaric oxygen on striatal dopamine levels in rats. Neurochem Res. 2017;42:1116–22. doi: 10.1007/s11064-016-2145-0. PMID: 28032294.

18 Lavoute C, Weiss M, Risso J-J, Rostain J-C. Alteration of striatal dopamine levels under various partial pressure of oxygen in pre-convulsive and convulsive phases in freely-moving rats. Neurochem Res. 2014;39:287–94. doi: 10.1007/s11064-013-1220-z. PMID: 24362638.

19 Bitterman N, Skapa E, Gutterman A. Starvation and dehydration attenuate CNS oxygen toxicity in rats. Brain Res. 1997;761:146–50. doi: 10.1016/s0006-8993(97)00442-3. PMID: 9247077.

20 Liu B, Traini R, Killinger B, Schneider B, Moszczynska A. Overexpression of parkin in the rat nigrostriatal dopamine system protects against methamphetamine neurotoxicity. Exp Neurol. 2013;247:359–72. doi: 10.1016/j.expneurol.2013.01.001. PMID: 23313192. PMCID: PMC4321803.

21 Fusar-Poli P, Rubia K, Rossi G, Sartori G, Balottin U. Striatal dopamine transporter alterations in ADHD: pathophysiology or adaptation to psychostimulants? A meta-analysis. Am J Psychiatry. 2012;169:264–72. doi: 10.1176/appi.ajp.2011.11060940. PMID: 22294258.

22 Aster H-C, Romanos M, Walitza S, Gerlach M, Mühlberger A, Rizzo A, et al. Responsivity of the striatal dopamine system to methylphenidate-a within-subject I-123-β-CIT-SPECT study in male children and adolescents with attention-deficit/hyperactivity disorder. Front Psychiatry. 2022;13:804730. doi: 10.3389/fpsyt.2022.804730. PMID: 35492708. PMCID: PMC9046584.

Conflicts of interest and funding

This study was funded by the Israel Ministry of Defense – Directorate of Defense Research and Development (IMOD DDR&D). No conflicts of interest were declared.

Submitted: 5 February 2024
Accepted after revision: 7 June 2024

Medical examination of divers after COVID-19 infection: a prospective, observational study using published (original and revised) guidelines for evaluation

Charlotte Sadler[1], Anna Lussier[1], Ian Grover[1], Karen Van Hoesen[1], Peter Lindholm[1]

[1] *Division of Hyperbaric Medicine and Wound Care, Department of Emergency Medicine UCSD University of California, San Diego, USA*

Corresponding author: *Associate Professor Charlotte Sadler, Division of Hyperbaric Medicine and Wound Care, Department of Emergency Medicine, University of California, San Diego, San Diego, CA, USA*
ORCiD: *0000-0002-3373-6597*
csadler@health.ucsd.edu

Keywords
Diving medicine; Fitness to dive; Medicals – diving; Respiratory; Occupational diving

Abstract
(Sadler C, Lussier A, Grover I, Van Hoesen K, Lindholm P. Medical examination of divers after COVID-19 infection: a prospective, observational study using published (original and revised) guidelines for evaluation. Diving and Hyperbaric Medicine. 2024 30 September;54(3):176–183. doi: 10.28920/dhm54.3.176-183. PMID: 39288921.)
Introduction: The COVID-19 pandemic raised significant concerns about fitness to dive due to potential damage to the pulmonary and cardiovascular systems. Our group previously published guidelines (original and revised) for assessment of these divers. Here, we report a prospective, observational study to evaluate the utility of these guidelines.
Methods: Recreational, commercial, and scientific divers with a history of COVID-19 were consented and enrolled. Subjects were evaluated according to the aforementioned guidelines and followed for any additional complications or diving related injuries.
Results: One-hundred and twelve divers (56 male, 56 female, ages 19–68) were enrolled: 59 commercial, 30 scientific, 20 recreational, two unknown (not documented), one military. Cases were categorised according to two previous guidelines ('original' $n = 23$ and 'revised' $n = 89$): category 0 ($n = 6$), category 0.5 ($n = 64$), category 1 ($n = 38$), category 2 ($n = 2$), category 3 ($n = 1$), uncategorisable due to persistent symptoms ($n = 1$). One hundred divers (89.3%) were cleared to return to diving, four (3.6%) were unable to return to diving, four (3.6%) were able to return to diving with restrictions, and four (3.6%) did not complete testing. Regarding diving related complications, one diver had an episode of immersion pulmonary oedema one year later and one diver presented with decompression sickness and tested positive for COVID-19.
Conclusions: Most divers who presented for evaluation were able to return to diving safely. Abnormalities were detected in a small percentage of divers that precluded them from being cleared to dive. Guidelines were easily implemented by a variety of clinicians.

Introduction

The emergence of the novel SARS-CoV-2 virus and subsequent COVID-19 pandemic raised significant concerns in the diving community regarding fitness to dive due to potential damage to the pulmonary and cardiovascular systems. Early reports in the pandemic suggested divers who recovered from COVID-19 may not be able to dive again due to permanent lung damage or that they would be at risk for pulmonary barotrauma or decompression sickness as a result of the lung injury from the infection.[1] There was also concern that divers could potentially have abnormal gas diffusion as a result of the chronic lung disease or complications from viral myocarditis.[2,3]

As the disease spread rapidly around the world, it became apparent that a uniform approach to assessing divers after recovering from COVID-19 before returning to dive would be necessary. These guidelines would need to be sensitive enough to protect divers from serious injury, but not overly restrictive or cost prohibitive. Our group published guidelines and then revised them two years later as vaccines were developed and the virus evolved.[4,5]

These guidelines are intended to be used on divers who have fully recovered, are asymptomatic from the symptoms of COVID-19, and have returned to their baseline exercise tolerance. The primary aim of this study was to evaluate the outcomes of COVID-19 infection in a cross-section of divers using the aforementioned guidelines. A secondary aim was to evaluate the feasibility of their use and implementation and to identify opportunities to improve and possibly modify the guidelines.

Methods

This study is an IRB approved (#201437), prospective, observational study. Subjects were enrolled beginning in January 2021 through June 2023. All divers at least 18 years of age who presented to our dive clinic during this time (including commercial, recreation, scientific, and military divers) with a history of COVID-19 were eligible for enrollment. A COVID-19 history was confirmed with positive testing, either at home or in a hospital or clinic setting. Positive COVID-19 antibody testing (without history of vaccination) could also be used to confirm diagnosis. Divers who presented to our emergency department with a diving related emergency with a history of COVID-19 were also eligible.

All subjects gave their written, informed consent. The majority of divers received an in-person evaluation either in clinic or in the emergency department; divers who were physically unable to attend dive clinic due to geographical constraints were evaluated via telemedicine.

A history of the patient's COVID-19 illness and sequelae, as well as a diving history, were obtained from the patient and from available medical records in the electronic medical record or supplied by the patients. Testing was obtained at our institution if possible, but we also reviewed results of outside records when necessary if testing was done elsewhere. We reviewed the original records of spirometry and imaging (not just interpretations) when able. If available, the patient's spirometry and imaging results were compared to prior values. Based on this history, divers were categorised using the previously published guidelines.[4,5] Divers enrolled prior to February 2022 were evaluated according to the original guidelines and divers enrolled after were evaluated according to the revised guidelines. Testing was ordered and completed according to guideline recommendations (see Tables 1 and 2). Return to dive guidance was based on these testing results and any additional pertinent information. These evaluations were performed by four faculty physicians who oversee the diving medicine clinic, with assistance from rotating fellows, residents and medical students.

Patients were followed up via subsequent clinic visits as required by their employer and/or by additional chart review or telephone calls.

Results

One-hundred and twelve divers were enrolled: 59 commercial, 30 scientific, 20 recreational, two unknown (not documented in chart), one military. Demographics: 56 male and 56 female, age range 19–68, mean age of 38 and a median age of 35. One-hundred divers (89.3%) were cleared to return to diving without restrictions, four (3.6%) were unable to return to diving, four (3.6%) were able to return to diving with restrictions, and four (3.6%) did not complete recommended testing. Most divers had isolated cases with seven having two or more episodes of COVID-19. We categorised all but one according to our previously published guidelines (original n = 23 and revised n = 89) (detailed in Case Vignette 2). The distribution of the divers' classification and outcomes is shown in Table 3.

SPIROMETRY AND IMAGING

Spirometry was obtained in 66 divers. Spirometry was considered abnormal if FEV_1, FVC, or PEF were below the lower limit of normal (NHANES III reference values), or $FEV_1/FVC < 0.75$.[6] Diffusion capacity of the lungs for carbon monoxide (DLCO) was not measured in all divers (due to varying location of testing). Seven divers initially had abnormal spirometry: one received a return to diving with restrictions designation, two were unable to return to diving, and four were cleared to return to diving (see Table 4). Abnormalities showed both obstructive and restrictive patterns. Imaging was obtained on 67 divers. Six had initially abnormal imaging: two received return to diving with restriction designation, one was unable to return to diving, two were cleared to return to diving, and one remained uncategorised based on failure to follow-up (see Table 4). Abnormal imaging findings varied, including persistent ground glass opacities to interstitial lung abnormalities, consistent with findings of sequelae of COVID-19.[7]

Divers with abnormal imaging and/or spirometry fell into the following categories: five divers with isolated abnormal spirometry (Category 1, n = 4, Category 0.5, n = 1); four divers with isolated abnormal imaging alone (Category 2, n = 1, Category 1, n = 2, Category 0.5, n = 1); two divers with abnormal imaging and abnormal spirometry (Category 3, n = 1, Category 2, n = 1) (See Table 5).

Although imaging and spirometry is not required for Category 0.5 divers, 36 of these divers received spirometry and/or imaging as part of yearly dive physical for either commercial or scientific diving. Of these 36 workups, three were abnormal. One had abnormal spirometry concurrent with a coexisting non-COVID upper respiratory infection, which resolved with repeat testing upon recovery. One had decreased FEV_1/FVC ratio but had normal FEV_1 and FVC. The final case had mild hyperinflation evident on chest X-ray but normal spirometry. All three were ultimately cleared to return to diving.

A NOTE ON SPECIFIC CASES

Of those divers designated unable to return to diving, one did not receive any workup due to a history of syncope which merited the 'unable' designation. The other three divers were classified as unable to return to diving based on persistent hypoxia with home oxygen use, persistent shortness of breath, and abnormal spirometry with concurrent asthma history not controlled on medication. One diver (Category 1) was initially diagnosed with long COVID

Table 1

Original classification of divers and work up recommendations based on severity of COVID-19 suspected illness; the categories of divers are based upon presenting symptoms and severity of disease which guides their subsequent work up recommendations. Noted factors include oxygen requirement, imaging, need for and level of hospitalisation, and cardiac involvement. If results are unknown or unavailable, recommendations are for more extensive cardiac and pulmonary evaluations. A diver should be placed in the highest category where they meet any (not all) of the criteria. If there is doubt that the diver's self-reported exercise level meets appropriate criteria, or concern it would not reveal underlying cardiac or pulmonary disease, further testing is warranted. BIPAP – bilevel positive airway pressure support; BNP – brain natriuretic peptide; CK-MB – creatine kinase MB fraction; CPAP – continuous positive airway pressure support; CT – computed tomography; DVT – deep venous thrombosis; ECG – electrocardiogram; ICU – intensive care unit; PA – posterior-anterior; RSTC – Recreational Scuba Training Council

Category 0 ***NO*** *history of COVID-19-suspected illness*	**Category 1** ***MILD*** *COVID-19-suspected illness*	**Category 2** ***MODERATE*** *COVID-19-suspected illness*	**Category 3** ***SEVERE*** *COVID-19-suspected illness*
Definition: Divers who have no history of COVID-19 suspected illness should proceed with normal evaluations. Additionally, we would use these criteria in those who may have had a positive screening PCR or antibody test, but without any history of illness or symptoms consistent with COVID-19.	**Definition:** • Did not seek health care or received outpatient treatment only without evidence of hypoxaemia. • Did not require supplemental oxygen. • Imaging was normal or not required.	**Definition:** • Required supplemental oxygen or was hypoxic. • Had abnormal chest imaging (chest radiograph or CT scan). • Admitted to the hospital but did NOT require mechanical (intubation) or assisted ventilation (BIPAP, CPAP) or ICU level of care. • If admitted, had documentation of a normal cardiac work up including normal ECG and cardiac biomarkers e.g., troponin or CK-MB and BNP.	**Definition:** • Required mechanical (intubation) or assisted ventilation (BIPAP, CPAP) or ICU level of care. • Cardiac involvement defined as abnormal ECG or echocardiogram, or elevated cardiac biomarkers e.g., troponin or CK-MB and BNP (or absence of documented work up). • Thromboembolic complications (such as pulmonary embolism, DVT, or other coagulopathy).
Work up recommendations: • Initial/periodic exam per professional group or RSTC guidelines. • Chest radiograph only if required per professional group or RSTC guidelines. No additional testing required.	**Work up recommendations:** • Initial/periodic exam per professional group or RSTC guidelines. • Spirometry. • Chest radiograph (PA and lateral); if abnormal, obtain chest CT. If unknown (or unsatisfactory) exercise tolerance*, perform exercise tolerance test with oxygen saturation.	**Work up recommendations:** • Initial/periodic exam per professional group or RSTC guidelines. • Spirometry. • Chest radiograph (PA and lateral); if abnormal, obtain chest CT. • ECG. • Echocardiogram (if no work up was done as an inpatient. Can forgo if had negative work up). • If unknown (or unsatisfactory) exercise tolerance*, perform exercise tolerance test with oxygen saturation. • Investigation and management of any other complications or symptoms per provider and professional group or RSTC guidelines.	**Work up recommendations:** • Initial/periodic exam per professional group or RSTC guidelines. • Spirometry • Chest radiograph (PA and lateral); if abnormal, obtain chest CT. • ECG. • Repeat cardiac troponin or CK-MB and BNP to ensure normalisation. • Echocardiogram. • Exercise Echocardiogram with oxygen saturation. • Investigation and management of any other complications or symptoms per provider and professional group or RSTC guidelines.

Table 2

Revised classification of divers and work up recommendations based on severity of COVID-19 suspected illness; the categories of divers are based upon presenting symptoms and severity of disease which guides their subsequent work up recommendations. Noted changes are the addition of Category 0.5 for those with very mild disease and primarily upper respiratory symptoms. Otherwise, interpretations and acronym definitions are as noted in Table 1. * for example, cough that is productive, prevents from sleeping, or requires medication, ultimately defined at the discretion of the evaluating physician

Category 0 ***NO*** *history of COVID-19-suspected illness*	**Category 0.5** ***VERY MILD*** *COVID-19-suspected illness*	**Category 1** ***MILD*** *COVID-19-suspected illness*	**Category 2** ***MODERATE*** *COVID-19-suspected illness*	**Category 3** ***SEVERE*** *COVID-19-suspected illness*
Definition: No history of COVID-19 or asymptomatic positive screening test.	**Definition:** • Isolated upper respiratory or systemic symptoms (rhinorrhea/ congestion/ pharyngitis/ loss of taste or smell), fevers, fatigue, or myalgias but WITHOUT lower respiratory or cardiac symptoms. Returned to baseline exercise tolerance.	**Definition:** • Symptomatic COVID-19 including any of the following: Any lower respiratory or cardiac symptoms, including chest pain, palpitations, significant* cough, shortness of breath with exertion or at rest. • Outpatient treatment only without evidence of hypoxemia. • Did not require supplemental oxygen • Imaging was normal or not required • Returned to baseline exercise tolerance.	**Definition**: • Required supplemental oxygen or was hypoxic. • Had abnormal chest imaging (chest radiograph or CT scan). • Admitted to the hospital but did NOT require mechanical (intubation) or assisted ventilation (BIPAP, CPAP) or ICU level of care. • If admitted, had documentation of a normal cardiac work up including normal ECG and cardiac biomarkers e.g., troponin or CK-MB and BNP. • Returned to baseline exercise tolerance.	**Definition:** • Required mechanical (intubation) or assisted ventilation (BIPAP, CPAP) or ICU level of care. • Cardiac involvement defined as abnormal ECG or echocardiogram, or elevated cardiac biomarkers e.g., troponin or CK-MB and BNP (or absence of documented work up). • Thromboembolic complications (such as pulmonary embolism, DVT, or other coagulopathy). • Returned to baseline exercise tolerance.
Work up recommendations: • Initial/periodic exam per professional group or RSTC guidelines. • Chest radiograph only if required per professional group or RSTC guidelines. No additional testing required.	**Work up recommendations:** • Initial/periodic exam per professional group or RSTC guidelines. • Chest radiograph only if required per professional group or RSTC guidelines. No additional testing required.	**Work up recommendations:** • Initial/periodic exam per professional group or RSTC guidelines. • Spirometry. • Chest radiograph (PA and lateral); if abnormal, obtain chest CT. If unknown (or unsatisfactory) exercise tolerance*, perform exercise tolerance test with oxygen saturation.	**Work up recommendations:** • Initial/periodic exam per professional group or RSTC guidelines. • Spirometry. • Chest radiograph (PA and lateral); if abnormal, obtain chest CT. • ECG. • Echocardiogram (if no work up was done as an inpatient. Can forgo if had negative work up). • If unknown (or unsatisfactory) exercise tolerance*, perform exercise tolerance test with oxygen saturation. • Investigation and management of any other complications or symptoms per provider and professional group or RSTC guidelines.	**Work up recommendations:** • Initial/periodic exam per professional group or RSTC guidelines. • Spirometry • Chest radiograph (PA and lateral); if abnormal, obtain chest CT. • ECG. • Repeat cardiac troponin or CK-MB and BNP to ensure normalisation. • Echocardiogram. • Exercise Echocardiogram with oxygen saturation. • Investigation and management of any other complications or symptoms per provider and professional group or RSTC guidelines.

Table 3

Diver COVID-19 classification and return to dive status; RTD – cleared to return to diving; RTDWR – cleared to return to diving with restrictions; URTD – unable to return to diving; Unknown – those who did not complete recommended testing or failed to return for follow-up

Status	Category 0	Category 0.5	Category 1	Category 2	Category 3	Uncategorised
RTD	6	61	33	–	–	–
RTDWR	–	2	–	2	–	
URTD	–	–	2	–	1	1
Unknown	–	1	3	–	–	
Total	6	64	38	2	1	1

Table 4

Diver spirometry/imaging results and return to dive status; RTD – cleared to return to diving, RTDWR – cleared to return to diving with restrictions, URTD – unable to return to diving; Unknown – those who did not complete recommended testing or failed to return for follow-up

Parameter	RTD	RTDWR	URTD	Unknown
Abnormal spirometry	4	1	2	–
Abnormal imaging	2	2	1	1
Totals	6	3	3	1

Table 5

Diver spirometry/imaging results and COVID-19 categorisation; number of divers by category with abnormal spirometry, imaging, or both

Category	Spirometry abnormal only	Imaging abnormal only	Both abnormal
0.5	1	1	0
1	4	2	0
2	0	1	1
3	0	0	1
Totals	5	4	2

syndrome but was cleared for return to diving based on complete resolution of symptoms and otherwise normal workup. Regarding diving related complications, it was noted that one diver had an episode of immersion pulmonary oedema one year after having COVID-19 and one diver presented with decompression sickness and tested positive for COVID-19 (though asymptomatic).

Below are two case vignettes that highlight challenges in evaluating and counseling divers after COVID-19 infection.

Case 1

A 55-year-old male recreational diver without significant past medical history presented for clearance to return to dive after COVID-19 infection. The visit was three months after initial infection. During his initial illness, he presented to the emergency department after developing fever (39.4°C, 103°F) and cough. He had a positive COVID-19 test in the emergency department, abnormal chest X-ray with bilateral consolidations, and a peripheral oxygen saturation of 85%. He had not received a COVID-19 vaccination. He was admitted to the hospital and treated with supplemental oxygen for eight days. After discharge, he observed decreased exercise tolerance, shortness of breath and hypoxia and tachycardia with exertion. This was documented using a home pulse oximeter showing oxygen saturations in mid-80s and heart rates in 130s when walking between rooms in the house. Over time, this resolved and at time of presentation he felt he was back to aerobic exercise baseline and noted oxygen saturation of 95–97% with exercise. He was also discharged with incentive spirometer and reported an increase in vital capacity from 1 L to 4 L. Physical examination in clinic was unremarkable. He had previously completed approximately 180 dives; he was a certified divemaster with plans to become instructor.

Based on his symptoms and history, the patient was designated Category 2 (moderate disease) and work up recommendations included electrocardiogram (ECG), two-view chest X-ray or chest computed tomography (CT), echocardiogram and stress echocardiogram, exercise tolerance test with oxygen saturation, and spirometry.

The ECG showed sinus rhythm with sinus arrhythmia, nonspecific ST-T changes. The two-view chest X-ray four months post infection showed near resolution of prior extensive bilateral consolidations. A repeat chest X-ray three months later was unchanged, with the radiologist's report stating that this "*may represent residual scarring*". A chest CT (eight months post infection) showed "*chronic peripheral interstitial disease and scarring with subtle residual ground glass disease with mild reticular component. Areas of peripheral scarring and subpleural banding*

are present…likely the chronic sequelae of COVID-19." These findings would later be considered typical for interstitial lung abnormalities after COVID-19.[7] An exercise echocardiogram (seven months post infection) showed no inducible ischaemia and fair exercise tolerance (7.0 MET). Spirometry and DLCO (five months post infection) were both normal. A resting echocardiogram and exercise tolerance test with oxygen saturation were not obtained for this patient.

Recommendations: The main residual abnormality noted was scarring on the CT chest, presumably from infection. We advised the patient that the implication in diving from these images is unknown but may theoretically increase the risk of barotrauma. Patient acknowledged the risks but wished to continue diving. We discussed risk mitigation techniques, including very slow ascents and prolonged safety stops. We also discussed doing a trial dive in a pool or in very shallow, controlled conditions to make sure he can tolerate it. He agreed with this plan and, in follow up, was found to have made approximately 50 subsequent dives without complications.

Case 2

A 57-year-old male recreational diver without significant past medical history presented for clearance to return to dive after COVID-19 infection. His initial illness with COVID-19 was 11 months prior. He had a prolonged hospitalisation (127 days) requiring intensive care admission and support with high flow nasal cannulae and home oxygen at discharge. Work up in the hospital included chest X-rays and chest CTs that were notable for pneumothorax that resolved without intervention and bilateral ground glass opacities. An echocardiogram was normal. He had not received a COVID-19 vaccination.

Since returning home, the patient reported his exercise tolerance had continued to improve, but he still required oxygen supplementation. For example, his oxygen saturation dropped to the mid-80s walking upstairs. He was very motivated to return to diving and had started diving already, despite not having clearance from a physician. He reported that he had been using nitrox (32%) tanks at home and had dived in a lake without issues using 33% nitrox. He was also using albuterol and budesonide inhalers and nebulisers at home twice daily. Physical examination in the clinic was notable for mild dyspnoea at rest. The guidelines were not appropriate to apply to this patient, as he had clearly not returned to his exercise capacity baseline, despite initially claiming to be asymptomatic. However, we still used the same principles of guidance for those who had suffered severe COVID-19 infection (Category 3) to evaluate this patient, including spirometry, chest CT, and echocardiogram.

Spirometry obtained six months after infection showed FVC 1.69 L (33% predicted value), FEV_1 1.51 L (38%), FEV_1/FVC 89%, DLCO 11.12 ml·min·mmHg^{-1} (33%), TLC 2.93 L (40%), consistent with severe restrictive disease. These values were unchanged post bronchodilator challenge. Chest CT obtained 10 months after the initial illness showed patchy air space disease, ground glass opacities, and traction bronchiectasis that were stable when compared to a CT done three months prior. An echocardiogram was obtained and was normal. An ECG was not immediately available. Exercise tolerance test with oxygen saturation was not ordered because the patient was still requiring home oxygen.

Recommendations: In addition to a history of spontaneous pneumothorax and pneumomediastinum (in the setting of viral illness but not on positive pressure ventilation), this patient had significant residual structural pulmonary abnormalities, resulting in abnormal gas diffusion and pulmonary function testing, as well as an ongoing oxygen requirement. Due to a high risk of multiple complications, including barotrauma and hypoxia, we recommended the patient stop diving.

Although uncommon, the above cases are representative of some of the residual changes noted in divers after COVID-19. Case 1 represents an example of return to diving with restrictions: although there were persistent changes noted on the CT scan, the implication of these findings with regards to risk in diving is still unknown. The subject was already certified and very much wished to return to diving and so was given counseling on the implications of potential risk and risk mitigation. It should be noted that this was a recreational diver and advice may have been different in a commercial or scientific diver.

Discussion

To our knowledge, this study represents the first and largest prospective study to date of divers post-COVID-19 infection. Reassuringly, the vast majority of our divers were able to return to diving safely after evaluation. However, we believe that our study highlights the importance of divers receiving, at minimum, an evaluation by a physician after infection and focused testing based on initial severity of symptoms.

In general, the initial severity of disease correlated well with abnormal findings/outcomes, though we did not necessarily find a strong relationship between abnormal imaging and abnormal spirometry, which underscores the need to obtain both and not rely solely on a single result. This finding is similar to those published by others. A retrospective study of 143 French military divers with COVID-19 found that 20 had persistent abnormal spirometry and 24 had abnormal chest CTs, but only three had both.[8] Another study found a higher rate of abnormal chest CTs in military divers post COVID-19 infection, but it should be noted that all subjects had chest CTs done, which likely have a higher sensitivity for abnormal findings, though the clinical significance is still unknown. The severity of disease was variable amongst these divers with a trend towards those who were hospitalised being more likely to have abnormal imaging.[9]

As the virus itself continued to evolve and particularly after the omicron variant became widespread, we noted that many of our divers presented with much milder, primarily upper respiratory infections, thus prompting the guideline revision and development of Category 0.5. Although this study was not designed to formally validate these categories, we do note that all of our category 0.5 divers (except one who failed to follow up) were able to return to dive. We suspect that the small percentage of abnormal findings in this category were either related to non-covid illness or likely the divers' baseline. These findings are similar to a study aiming to validate the South Pacific Underwater Medicine Society criteria for very mild disease (essentially identical to our own). They found that amongst 57 occupational divers who met very mild criteria, all had spirometry unchanged from their baseline and were able to return to diving without incident.[10] Another study of military hyperbaric personnel with asymptomatic or 'subclinical' COVID-19 (not hypoxic or hospitalised) found no significant difference in spirometry compared with non COVID-19 infected peers.[11]

Regarding the feasibility and ease of use of the guidelines, the authors and their colleagues found them easy to use in evaluation and guidance for recommended testing. It should be noted that the evaluation of these divers for this study was performed by a variety of physicians at the clinic (see acknowledgments), thus reinforcing that these guidelines are straightforward to interpret and implement by practitioners. Overall, the testing required was not significantly onerous and able to be accomplished in almost all subjects.

As evidenced by the case vignettes presented, the results of this testing and subsequent counseling of the divers may be nuanced. We ultimately labeled some divers as "*return to dive with restrictions*", but recognise that this is a broad categorisation and dependent on many factors. For example, restrictions or disqualifications for commercial or military divers are often much more clear-cut than for recreational divers. Divemasters or instructors may also have additional tasks or requirements that would make it impossible to modify their diving practices in that role. Additionally, any advice given to recreational divers to potentially mitigate risk is based on expert opinion, but lacks published evidence to support it. It is the authors' opinion and practice that these must be evaluated on a case-by-case basis, taking into account the type of diving and risk tolerance of the individual diver.

We acknowledge that the guidelines do have limitations. We did not specify a mandatory waiting time after infection to be evaluated, though functionally with the mandatory quarantine times, this was a minimum of 10–14 days. We suspect that if we had waited longer, we would have seen fewer initial abnormalities in spirometry. We do not necessarily recommend implementing a longer mandatory time to evaluation but note that it seems more likely to have transient abnormal values of spirometry immediately after infection, as with other non-covid viral respiratory infections.

We also did not specify time intervals for testing or retesting, nor specific types of exercise testing. This was done intentionally to allow flexibility for both divers and practitioners, but we acknowledge that it can make results hard to compare between divers. We also note that our guidelines only addressed cardiac and pulmonary symptoms, but that sequelae of COVID-19 can have a much broader impact. Future studies may need to address other organ system manifestations that could potentially affect divers such as neurologic and systemic (muscle fatigue, decreased exercise tolerance, other cardiac manifestations, postural orthostatic tachycardia syndrome, long COVID).

We did not find a significant incidence or correlation with diving accidents such as decompression sickness, barotrauma, or immersion pulmonary oedema, but given the relatively low incidence of these disease states, we would expect to need a larger sample size and longer follow up period to evaluate such an association.

Conclusions

In conclusion, using the previously published guidelines to evaluate divers after COVID-19, we found that the vast majority of divers were able to return to diving safely, particularly those with relatively mild infections (categories 0.5 and 1).[4,5]

The persistent abnormal findings observed are still of unknown clinical significance to divers and further work will need to be done to better understand their physiologic implications and consequences. These guidelines were relatively easy to adopt and implement by multiple practitioners. It is our stance that work up and evaluation of divers after COVID-19 is still indicated, even if the divers are asymptomatic. At this time, these authors do not have intentions to modify the guidelines or change evaluation protocols for divers who have had COVID-19, though we are open to modifications if indicated in the future.

References

1 Hartig F. Target organ lung - diving after Covid 19 disease? Wetnotes [Internet]. 2020 Apr. [cited 2024 Feb 25]. Available from: https://duikgeneeskunde.nl/wp-content/uploads/2020/04/diving-after-Covid-19.pdf.

2 Spagnolo P, Balestro E, Aliberti S, Cocconcelli E, Biondini D, Casa GD, et al. Pulmonary fibrosis secondary to COVID-19: a call to arms? Lancet Respir Med. 2020;8:750–2. doi: 10.1016/S2213-2600(20)30222-8. PMID: 32422177. PMCID: PMC7228737.

3 Akhmerov A, Marbán E. COVID-19 and the heart. Circ Res. 2020;126:1443–55. doi: 10.1161/CIRCRESAHA.120.317055. PMID: 32252591.

4 Sadler C, Alvarez-Villela M, Van Hoesen K, Grover I, Lang M, Neuman T, et al. Diving after COVID-19: an update to fitness

to dive assessment and medical guidance. Diving Hyperb Med. 2022;52:66–7. doi: 10.28920/dhm52.1.66-67. PMID: 35313377. PMCID: PMC9016139.

5 Sadler C, Alvarez Villela M, Van Hoesen K, Grover I, Lang M, Neuman T, et al. Diving after SARS-CoV-2 (COVID-19) infection: fitness to dive assessment and medical guidance. Diving Hyperb Med. 2020;50:278–87. doi: 10.28920/dhm50.3.278-287. PMID: 32957131. PMCID: PMC7755459.

6 Hankinson JL, Odencrantz JR, Fedan KB. Spirometric reference values from a sample of the general U.S. population. Am J Respir Crit Care Med. 1999;159:179–87. doi: 10.1164/ajrccm.159.1.9712108. PMID: 9872837.

7 Han X, Chen L, Fan Y, Alwalid O, Jia X, Zheng Y, et al. Longitudinal assessment of chest CT findings and pulmonary function after COVID-19 infection. Radiology. 2023;307(2):e222888. doi: 10.1148/radiol.222888. PMID: 36786698. PMCID: PMC9969419.

8 Morin J, Vallée N, Dufresne PL, Rives S, Lehot H, Daubresse L, et al. Symptomatic or asymptomatic SAR-CoV-2 positive divers should be medically evaluated before returning to scuba diving. Front Physiol. 2022;13:1022370. doi: 10.3389/fphys.2022.1022370. PMID: 36439242. PMCID: PMC9691879.

9 Mirasoglu B, Yetis G, Erelel M, Toklu AS. Post COVID-19 fitness to dive assessment findings in occupational and recreational divers. Diving Hyperb Med. 2022;52:35–43. doi: 10.28920/dhm52.1.35-43. PMID: 35313371. PMCID: PMC9177431.

10 Smart D. Validation of very mild COVID-19 illness criteria to guide successful return to occupational diving. Diving Hyperb Med. 2022;52:222–3. doi: 10.28920/dhm52.3.222-223. PMID: 36100936. PMCID: PMC9722341.

11 Schaap JP, Zuluaga Fernandez ME, Houtkooper A, Endert EL, van Ooij PAM. How fit are military hyperbaric personnel after an asymptomatic or mild symptomatic COVID-19 infection? A retrospective study. Diving Hyperb Med. 2023;53:120–8. doi: 10.28920/dhm53.2.120-128. PMID: 37365129. PMCID: PMC10584392.

Acknowledgments

The authors would like to thank and acknowledge Tom Neuman MD, Miguel Alvarez Villela MD, and Michael Lang PhD for their contributions in developing these clinical guidelines. The authors also wish to thank Tiffany Castellano MD, Ian Kirby MD, Craig Kutz MD, Evan Laveman MD, Paulina Pantcheva MD, Casey Smith MD, and William Toppen MD for their assistance in data acquisition.

Conflicts of interest and funding

No conflicts of interest were declared. Funding for this project was provided by the Academy of Clinician Scholars (AOCS), the Gurnee endowed chair at the University of California, San Diego (La Jolla, CA USA), and the Padi Foundation (Beverly Hills, CA USA). The authors would also like to acknowledge Divers Alert Network (DAN) (Durham, NC USA) for their ongoing financial support of the Undersea and Hyperbaric Medicine Fellowship program at the University of California, San Diego.

Submitted: 25 February 2024
Accepted after revision: 6 July 2024

Retrospective analysis of challenging cases for medical examiners of diving

Inge Reus[1], Erik van de Sande[2,3], Rienk Rienks[2,4], Thijs Wingelaar[1,2]

[1] *Royal Netherlands Navy Diving and Submarine Medical Centre, Den Helder, the Netherlands*
[2] *Dutch Society of Diving and Hyperbaric Medicine (DSDHM), Bilthoven, the Netherlands*
[3] *Dutch Society for Sports Medicine (VSG), Bilthoven, the Netherlands*
[4] *CardioExpert Clinic for Sports and Occupational Cardiology, Amsterdam, the Netherlands*

Corresponding author: *Inge Reus, Royal Netherlands Navy Diving and Submarine Medical Centre, Den Helder, the Netherlands*
ORCiD: *0009-0005-7416-1469*
i.reus@mindef.nl

Keywords
Education; Fitness to dive; Medicals-diving; Recreational diving; Training

Abstract
(Reus I, van de Sande E, Rienks R, Wingelaar T. Retrospective analysis of challenging cases for medical examiners of diving. Diving and Hyperbaric Medicine. 2024 30 September;54(3):184–187. doi: 10.28920/dhm54.3.184-187. PMID:39288922.)
Introduction: Assessing a diver's fitness to dive enhances diving safety, with medical examiners of diving (MED) being entrusted with this responsibility. However, the effectiveness of MED training in preparing physicians for this task remains underexplored. In the Netherlands, where any physician can pursue MED qualification, challenging cases can be presented to a board of experts.
Methods: This retrospective analysis included all cases presented to a board of experts in the period 2013–2023. Aside from baseline information, cases were coded using the International Classification of Diseases 11th Revision (ICD-11). Additionally, the type of advice given by the board was also recorded.
Results: A total of 291 cases could be included, 62.5% were male divers with a median age of 47 years old (interquartile range 29–55). Circulatory (20.9%), respiratory (16.2%), neurologic (14.4%), psychiatric (9.6%) and endocrine (6.5%) disease comprised more than two-thirds of all presented cases. Problems for the MED included multimorbidity, knowledge of guidelines and interpretation of diagnostic data.
Conclusions: These results could be used to improve MED courses or serve as a topic for continuing medical education for MEDs, however, further research into generalisability is required.

Introduction

Although a recreational diver's medical condition may not directly cause a diving accident, it can predispose the individual to risk or impede their ability to respond effectively to emergencies.[1,2] Therefore, assessing fitness to dive is fundamental to ensuring diving safety, a responsibility often entrusted to medical examiners of diving (MED), who are physicians trained in this specific domain. Such standards governing this training are outlined in the Educational and Training Standards for Diving and Hyperbaric Medicine of the European Committee for Hyperbaric Medicine and the European Diving Technical Committee (ECHM- EDTC).[3]

Medical examiner training covers a broad spectrum of topics ranging from general diving protocols to specific dive-related medical conditions. In the Netherlands, any physician can pursue MED qualification by completing courses offered by two accredited providers adhering to the stipulated standards. Although most candidates are general practitioners or sports physicians, diverse medical specialists also participate, reflecting variations in prior knowledge and experience that may influence educational requirements and efficacy.

Despite the relevance of acquired knowledge from MED training and on-going medical education, the evaluation of course effectiveness remains largely unexplored. Evaluating the curriculum poses challenges, as recent MED graduates have limited experience in determining the course content's appropriateness. Traditional research methods, such as surveys or prospective cohort studies, are hindered by potential recollection bias and resource constraints.[4,5]

BOARD OF EXPERTS

In the Netherlands, a 'board of experts' comprising 15 physicians (more details below) and acting as a Board of the Dutch Society of Diving and Hyperbaric Medicine and the Netherlands Association of Sports Medicine, offers a valuable backup resource for MED practitioners. Examiners can freely present dive-related medical queries concerning recreational divers to this board, using their collective

expertise to seek guidance and reflection on challenging cases. This platform, operational for over a decade, provides real-world insights into the issues encountered by MEDs, which may contribute to improvements in the training curriculum and continuing medical education initiatives.

Criteria for a physician to participate in this board of experts were: being currently registered as a physician in the Netherlands, board certification of the applicable specialty, valid MED certification or equivalent, regular examination of patients with respect to diving-related questions of pathology and regular interaction with other board members about the questions posed by the MEDs. The board comprises two cardiologists, two neurologists, an ENT specialist, a pulmonologist, a gynaecologist, a psychiatrist, an internist, an allergologist, with the remainder being sport physicians and a general practitioner with extensive experience in, and knowledge of, scuba and free diving.

WHY DO MEDS NEED HELP?

This study aimed to determine what type of cases MEDs encounter in daily practice. Given the predominantly male and middle-aged diving population, it was hypothesised that cardiovascular-related inquiries constitute a significant portion of cases presented to MEDs.[6,7]

Methods

This retrospective analysis encompassed all 291 cases brought before the board of experts from 1 January 2013, to 31 March 2023. According to national law, retrospective analyses are exempt from evaluation by a medical ethics committee. Given that all cases presented to the board were anonymised, this study adhered to European General Data Protection Regulation (GDPR) and national privacy legislation. The protocols for managing medical information adhered to both national and European legislation, as well as the guidelines set forth by the Association of Universities in the Netherlands.

CONTEXT

In the Netherlands, recreational divers are not obligated to undergo dive medical assessments. Nonetheless, many diving associations recommend a fitness-to-dive evaluation by a MED, particularly for diving instructors. Notably, the board of experts does not handle cases concerning occupational and military divers, as the fitness-to-dive assessments for these groups are legally the purview of occupational physicians in the Netherlands. While a clear guideline for freedivers is absent in the Netherlands, the medical and science committee of the International Association for the Development of Apnea (AIDA) suggests medical assessments should be performed.[8]

To assist MEDs in conducting fitness-to-dive assessments, the Dutch Society of Diving and Hyperbaric Medicine (DSDHM) has published various guidelines, such as those pertaining to diving with psychotropic medication and cardiovascular disease, in both Dutch and international peer-reviewed journals.[9,10]

ANALYSIS

All 291 cases were entered into a separate database, with diagnoses being coded based on the International Classification of Diseases 11th Revision (ICD-11). Cases without an answer from the board of experts were excluded from the analysis. In instances involving multimorbidity two authors (IR and TW) deliberated, selecting the most pertinent diagnosis for inclusion in the database. The nature of advice provided by the board of experts was also coded. Given that this study entailed solely descriptive data, no statistical analyses were undertaken.

Results

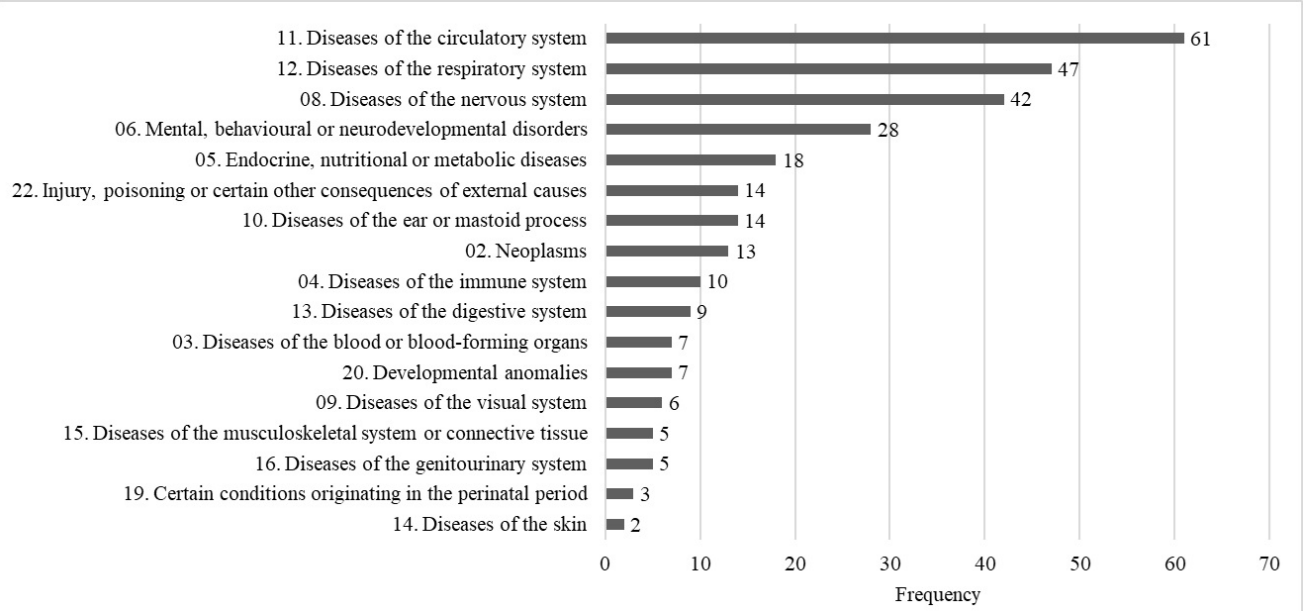
Over the span of 10 years, the board of experts reviewed a total of 291 cases, all of which were included in this study. Among these cases, 62.5% involved male divers (4.5% lacked sex information), with a median age of 47 years (interquartile range 29–55; 18.2% had missing age data). The majority of cases involved recreational scuba divers, with a smaller percentage comprising scuba diving instructors and one freediver, accounting for 92.8%, 6.9%, and 0.3% respectively.

PREVALENCE OF MEDICAL CONDITIONS

The most prevalent diseases involved the circulatory (20.9%), respiratory (16.2%), and nervous (14.4%) systems. Combined with, mental, behavioural, or neurodevelopmental disorders (9.6%) and endocrine diseases (6.5%, predominantly diabetes mellitus), this accounted for over two-thirds of all cases. Notably, specific diving-related illnesses were the focus of 14 (4.8%) cases presented to the board, as illustrated in Figure 1.

Different types of inquiries emerged for each of the top three medical categories. In cardiovascular disease, concerns primarily revolved around arrhythmia (18% of cases in this category), ischaemic heart disease (13%), and pulmonary embolism (11%), often driven by considerations of multimorbidity prompting consultation with the board. The board's recommendations typically involved referral to existing guidelines, additional diagnostic investigations, or case-specific considerations. Similarly, respiratory disease inquiries focused on spirometry (25%), asthma (21%), and pneumothorax (15%), with the board often advising referral to guidelines or further diagnostic assessments. Neurological disease inquiries predominantly involved diving with epilepsy (21%), post-cerebrovascular events (21%), and multiple sclerosis (12%), usually related to medication side effects or diving resumption timelines, with the board frequently referring to guidelines on diving with these conditions.

Figure 1
Overview of ICD-11 classification of presented cases; data are number of cases. The number shown before the categories is the corresponding ICD-11 group



RECOMMENDATIONS GIVEN BY THE BOARD OF EXPERTS

Of all cases reviewed, the board recommended resumption of diving activities in 68 cases (23.4%), with approximately half involving restrictions (such as depth limitation or avoiding drift diving). A 'temporary unfit' recommendation, typically pending additional medical investigations, was advised in almost half (47.8%) of cases of which 108 cases (37.1%) required additional investigations, 24 (8.2%) required a longer complaint-free interval and 7 (2.4%) required treatment. About one-fifth (18%) were deemed 'unfit to dive'. Consensus within the board of experts on the case could not be reached in 3.8% of cases, and in the remaining 5.8%, 'other' recommendations were provided, including changes in medication or advice on diving gases.

Discussion

To the best of our knowledge, this study represents the first systematic analysis of the cases that MEDs perceive as challenging when assessing recreational divers. The data presented in this study could be used to determine whether the MED course equips physicians adequately for conducting fitness-to-dive assessments. Analysis of inquiries to a board of experts revealed that MEDs most frequently encounter challenges with circulatory, respiratory, and neurological diseases. The nature of inquiries varies across medical categories, with multimorbidity, interpretation of diagnostic data, or unfamiliarity with existing guidelines being common reasons for seeking board consultation. Notably, the board was able to provide clear advice to MEDs in the majority of cases, with consensus unachievable in only 3.8%.

Given the absence of similar analyses, contextualising these findings is challenging. However, it is important to note that our study delineated the national context in the introduction and methods sections, with subsequent sections reflecting solely the authors' interpretation. We believe that a board of experts can offer significant support in complex cases involving multimorbidity or diseases lacking clear guidelines. Nevertheless, some inquiries could be readily addressed by referring to existing guidelines or aiding in the interpretation of diagnostic data, such as electrocardiograms (ECGs) or spirometry. Aside from the Dutch Society of Diving and Hyperbaric Medicine evaluation as to whether guidelines are sufficiently accessible for MEDs, addressing guidelines and issues such as clinical interpretation of spirometry and ECGs within the MED course or continuing medical education may be of value.

A further observation from our data is that cardiovascular, pulmonary, neurological, psychiatric, and endocrine diseases collectively account for more than two-thirds of presented cases posing challenges to MEDs. While guidelines exist for fitness-to-dive assessments in many of these diseases, we advocate for the development of improved guidelines, preferably published in international peer-reviewed journals, to address this gap comprehensively. Additionally, we recommend that MED course organisers should consider allocating more attention to these five categories to better equip MEDs for fitness-to-dive assessments.

Lastly, we think that our study highlights the potential practical utility of a board of experts, as described herein, in offering advice to MEDs in the majority of cases. While this assertion is not directly supported by our data, we believe that such a board could positively influence the quality of

fitness to dive assessments, particularly for MEDs lacking easy access to diving medical colleagues for case discussion or reflection.

STRENGTHS AND WEAKNESSES ANALYSIS

With nearly 300 cases spanning a decade, this dataset provides valuable insights into the challenges MEDs encounter in fitness-to-dive assessments. However, several limitations warrant consideration.

Firstly, the context in which MEDs operate is influenced by local, national, and international legislation, as well as the characteristics of the diving population. Thus, the generalisability of our results may be limited. Nonetheless, it is reasonable to infer that cardiovascular, respiratory, and neurological diseases, especially in cases of multimorbidity and complex medications, present a challenge for MEDs. We encourage our international colleagues to conduct similar studies to elucidate the educational needs of MEDs and optimise MED courses accordingly.

Secondly, the absence of a centralised registry makes it challenging to ascertain the total number of MEDs and fitness-to-dive assessments performed in the Netherlands, i.e., the denominator is missing. Variations may exist in the types of cases encountered by general practitioners or sports physicians compared to specialists, as well as in the nature of inquiries posed to the board of experts. Additionally, the organisational setting of MEDs, whether working collaboratively or independently, remains undocumented and could have impacted our findings.

Lastly, while the ICD-11 classification provides a standardised method for case description, it has its limitations. Cases may encompass elements from multiple medical categories, complicating classification. Although we opted to present the 'main problem' for clarity, an alternative approach involving the presentation of all problems could have provided deeper insight into diseases within the diving community and cases MEDs encounter. The 'all problems' approach did not alter the identification of the top five categories responsible for the majority of cases, therefore we chose to present the current data. However, we would like to underscore that while these five categories are responsible for the majority of the presented cases, other categories could very well be a challenge for MEDs and deserve the proper attention in MED courses and continuing education.

Conclusions

This retrospective analysis of nearly 300 fitness-to-dive assessments conducted by MEDs in the Netherlands demonstrates that circulatory, respiratory, neurological, psychiatric, and endocrine diseases collectively constitute a significant proportion of cases presenting challenges. While some cases are complex, involving multimorbidity, others entail less intricate queries regarding test data interpretation. We feel these data can be of value to improve MED courses or suggest topics for continuing medical education for MEDs. However, further research is necessary to confirm whether these findings can be generalised before adapting the MED course framework as described by the ECHM-EDTC.

References

1 Lock G. Under pressure: Diving deeper with human factors. 1st ed. Wiltshire: Human in the System Consulting; 2019.

2 Turner BL, van Ooij PA, Wingelaar TT, van Hulst RA, Endert EL, Clarijs P, Hoencamp R. Chain of events analysis in diving accidents treated by the Royal Netherlands Navy 1966-2023. Diving Hyperb Med. 2024;54:39–46. doi: 10.28920/dhm54.1.39-46. PMID: 38507908. PMCID: PMC11227959.

3 Joint Educational Subcommittee of the European Committee for Hyperbaric Medicine (ECHM) and the European Diving Technical Committee (EDTC). Educational and Training Standards for Physicians in Diving and Hyperbaric Medicine. 2011. [cited 2024 April 9]. Available from: http://www.echm.org/documents/ECHM-EDTC%20Educational%20and%20Training%20Standards%20(2011).pdf.

4 Simpson G, Roomes D. Scuba diving medical examinations in practice: a postal survey. Med J Aust. 1999;171(11-12):595–8. doi: 10.5694/j.1326-5377.1999.tb123812.x. PMID: 10721340.

5 Sames C, Gorman D, Mitchell S. Postal survey of fitness-to-dive opinions of diving doctors and general practitioners. Diving Hyperb Med. 2012;42:24–9. PMID: 22437972. [cited 2024 April 9]. Available from: https://dhmjournal.com/images/IndividArticles/42March/Sames_dhm.42.1.24-29.pdf.

6 Strauss MB, Busch JA, Miller SS. Scuba in older-aged divers. Undersea Hyperb Med. 2017;44:45–55. doi: 10.22462/1.2.2017.8. PMID: 28768085.

7 Lippmann J, Taylor D McD, Stevenson C, Mitchell S. The demographics and diving behaviour of DAN Asia-Pacific members with and without pre-existing medical conditions. Diving Hyperb Med. 2016;46:200–6. PMID: 27966201. [cited 2024 April 9]. Available from: https://dhmjournal.com/images/IndividArticles/46Dec/Lippmann_dhm.46.4.200-206.pdf.

8 Medical and Science Committee of the International Association for the Development of Apnea (AIDA). Medical recommendations for organizers of competitions under auspices of AIDA International. 2019. [cited 2024 April 9]. Available from: https://drive.google.com/file/d/1J8dFwY9S3x4el66sWghRSF-Yhbb0u46k/view.

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

10 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.

Conflicts of interest and funding: nil

Submitted: 31 May 2024
Accepted after revision: 29 July 2024

Arterial dissection in scuba divers: a potential adverse manifestation of the physiological effects of immersion

Neal W Pollock[1,2], John Lippmann[3,4], John Pearn[5], John Hayman[6,7]

[1] *Department of Kinesiology, Université Laval, Quebec, QC, Canada*
[2] *CISSS Chaudière-Appalaches (CHAU-Hôtel-Dieu de Lévis), Hyperbaric Medicine Unit, Emergency Department, Lévis, QC, Canada*
[3] *Australasian Diving Safety Foundation, VIC, Australia*
[4] *Department of Public Health and Preventive Medicine, Monash University, Clayton, VIC, Australia*
[5] *Queensland Children's Hospital, South Brisbane QLD, Australia*
[6] *Department of Clinical Pathology, The University of Melbourne, VIC, Australia*
[7] *Menzies School of Health Research, Casuarina, NT, Australia*

Corresponding author: *Associate Professor Neal W Pollock, Université Laval, Quebec, QC, Canada*
neal.pollock@kin.ulaval.ca

Keywords
Dissecting aneurysm; Dissecting aortic aneurysm; Dissecting coronary artery; Diving; Immersion; Mammalian dive response; Osteogenesis imperfecta

Abstract
(Pollock NW, Lippmann J, Pearn J, Hayman J. Arterial dissection in scuba divers: a potential adverse manifestation of the physiological effects of immersion. Diving and Hyperbaric Medicine. 2024 30 September;54(3):188–195. doi: 10.28920/dhm54.3.188-195. PMID: 39288923.)
Introduction: Aortic dissections and dissections of cervical, cerebral, and coronary arteries have been previously reported in scuba divers. These incidents may be the consequence of a variety of physiological effects. We review the reported cases of arterial dissection in scuba divers and discuss potential contributing factors related to immersion and diving.
Methods: Medline, CINAHL Plus, and SPORTDiscus were searched for published reports of arterial dissection and the Australasian Diving Safety Foundation fatality database was searched for additional cases from Australia. Identified cases were recorded and scrutinised for possible contributing factors.
Results: Nineteen cases of arterial dissection, both fatal and non-fatal, were identified. These included cervical or intracranial artery dissection (n = 14), aortic dissection (n = 4), and coronary artery dissection (n = 1). There were 14 male and five female victims; mean age 44 years (SD 14, range 18–65). Contributing factors may include a combination of vasoconstriction and blood redistribution, untreated hypertension, increased pulse pressure, abnormal neck movement or positioning, constrictive and burdensome equipment, exercise, increased gas density and circuit resistance with concomitant elevated work of breathing, atheroma, and possibly the mammalian dive response.
Conclusions: Dissecting aneurysms of the aorta or cervical, cerebral, and coronary arteries should be considered as a potential complication of scuba diving. The development of aneurysms associated with scuba diving is likely multifactorial in pathogenesis. Detailed reporting is important in the evaluation of cases. The potential role of the mammalian dive response as a contributing factor requires further evaluation.

Introduction

Underwater diving is used worldwide for a range of recreational, commercial, scientific, and military operations. Although most diving is to depths less than 40 metres of seawater (msw), divers may descend to much greater depths and stay underwater for extended periods. Scuba apparatus has evolved substantially in recent decades, but its use still requires appropriate selection, training, maintenance, and implementation. Although such equipment enables a diver to breathe underwater, usually in relative comfort, it introduces certain potential complications that need to be managed.

Incidents, including fatalities, do occur in diving. Many deaths result from human factors such as inexperience, impaired health and fitness, and failure to adhere to safety guidelines.[1–3] However, more than one-quarter of scuba fatalities have reportedly been associated with cardiac causes, often reflecting health issues such as obesity, poor physical fitness, and an aging diver cohort.[1–4] It is recognised that clinically silent, intermittent cardiac conduction abnormalities can be triggered during diving.[5] Significant ischaemic heart disease and other pre-existing cardiac conditions may also precipitate dysrhythmias while diving.[6] Dissecting arterial and aortic aneurysms are also possibilities. Although rare, cases have been reported in sufficient number for this to be accepted as a diving hazard.[7–25]

Arterial dissection involves tearing of the intima, splitting of the media, with blood tracking through the divided vessel wall, which can lead to malperfusion of end organs, and rarely, arterial rupture.[26] Neck artery dissections may occur through a variety of physically violent events and blunt trauma, but may also occur after less violent events such as exercise stretching or chiropractic neck manipulations.[27,28] Aortic dissection has also been reported in conjunction with weightlifting.[29]

There are several classification systems for aortic dissection, with the Stanford classification the most commonly used. The Stanford classification includes two types: in type A the entry tear originates in the ascending aorta but may continue into the descending and abdominal aorta; and in type B the entry tear originates in the descending aorta but can extend in a retrograde fashion through the arch and ascending aorta.[30] Of the two, type A is the more lethal. Both types can be temporally classified as acute, sub-acute, or chronic in nature.

The risk of arterial dissection in scuba divers is likely increased by a host of factors, including the physiological effects of immersion, notably centralisation of blood, in addition to other burdens associated with diving, such as equipment worn, workload pre-, during, and post-dive, and general or other environmental stressors. It is an open question as to whether there may be a contribution of the mammalian dive response.

The dive response is a complex physiological reflex, well developed in diving mammals. The effects seen during breath-hold include bradycardia, increased systolic blood pressure, constriction of cutaneous, muscular, and splanchnic vessels, and preservation of the blood flow to the brain and heart.[31] The overall effect is to preserve oxygen for these two vital organs while reducing overall oxygen consumption in tissues more tolerant of hypoxia. The practical cardiovascular effect is an increase in systolic and pulse pressures, primarily confined to cervical, cerebral, and coronary arterial vessels and the aorta (Figure 1). The pressure increase is augmented by an increase in venous return to the right heart as a result of peripheral vascular constriction.

The dive response is present in all human neonates, diminishing in intensity by 12 months of age, but remaining as a substantial response in most adults.[32] In humans the response involves the afferent arc of the trigeminal (fifth) cranial nerve; the area of response activation includes the forehead which is innervated by ophthalmic branch of that nerve.[33]

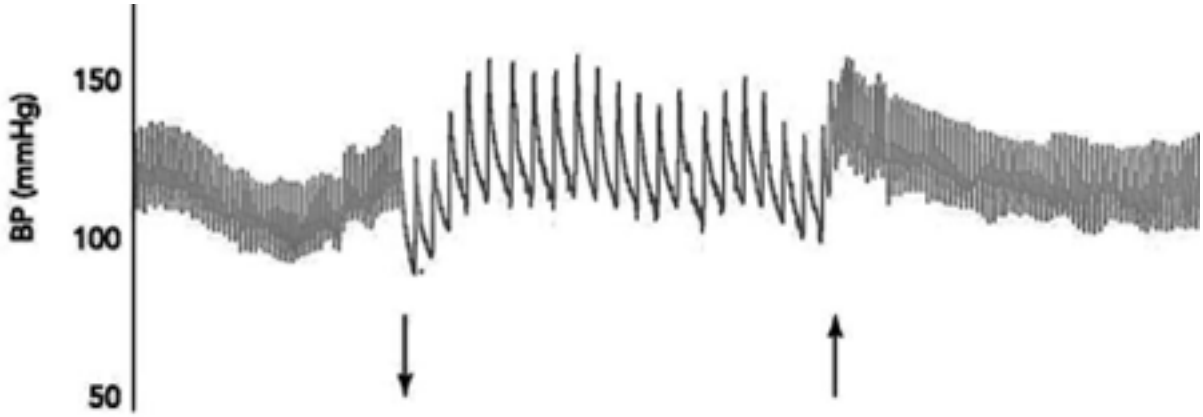
The classic evidence for dive response in humans is seen with breath-hold and facial immersion, particularly in cool or cold water. The effect would be blunted by the continued breathing associated with scuba diving, and likely also by the mask and often a hood covering a substantial portion of the face, but it is possible that the dive response pathway could augment a blood pressure spike if activated. The aim of this report is to discuss potential contributing factors to arterial dissection in scuba divers including the possible contribution of the dive response.

Methods

A literature search was conducted using Medline, CINAHL Plus and SPORTDiscus using the search terms "*arterial dissection*" or "*artery dissection*" or "*coronary artery dissection*" or "*aortic dissection*" or "*dissecting aneurysm*" AND "*scuba*". In addition, a search of the Australasian Diving Safety Foundation (ADSF) diving fatality database

Figure 1

Pulse and blood pressure recording from a rat trained to swim underwater through a tunnel; down arrow – start of dive; up arrow – end of dive. There is a marked bradycardia beginning immediately at the commencement and ceasing directly at the end of the dive. The blood pressure rise is slightly delayed and persists a short time after the dive. The pulse pressure increases from 20 mmHg to 40 mmHg during submersion. From WM Panneton, with kind permission



(adsf.org.au) was conducted to find any further documented fatalities attributed to aortic and arterial dissection.

As the dive response pressure changes also affect the coronary arteries a search was made for coronary artery dissection associated with scuba, using the search terms "*scuba*" AND "*coronary artery dissection*."

Results

The available case data are summarised in Table 1. The results are divided into three sections based on the location of the dissecting aneurysm – aorta, cervical and/or intracranial arteries, and coronary arteries. Descriptions of the dives, potential contributing factors, and the timing of problem development relative to diving were included as available.

AORTIC DISSECTING ANEURYSMS

One of us (author JH) reviewed and reported an early case of aortic dissection in a scuba diver and was aware of a second.[7,8] Two additional cases were more recently recorded.[9,10] All victims were male, and all dissections were type A, with onset evident during or within minutes of surfacing. Documentation of predisposing disease was limited. There was no evidence of contributing factors in the youngest victim (case 1 – 20 years of age), no information for one (case 2), and evidence of ischaemic heart disease and hypertension in cases 3 and 4. Three of the four cases were fatal, each of these reported in Australian waters. The most recent case had the only non-fatal outcome.[10]

CERVICAL AND INTRACRANIAL DISSECTING ARTERIAL ANEURYSMS

Four articles presented case reports, each listing cases of previous cervical artery dissections associated with scuba diving.[15,16,18,19] Five additional cases were identified in the search. A total of 14 cases were identified with age ranges from 18 to 60 years; four were female (Table 1). Unlike the aortic dissections there were no fatalities and the diagnoses were made based on clinical and radiological features. The onset of symptoms relative to diving was variable, including two cases during dives, six cases within minutes of surfacing, three cases within hours of surfacing, two cases within two days of diving, and one case with an unknown point of onset.

Fourteen cases involved the cervical or intracranial arteries (Table 1). Nine involved only the carotid arteries (cases 5, 8, 9, 10, 12, 13, 16, 17, 18), two only the right vertebral arteries (cases 7 and 14), and one involved both carotid and both vertebral arteries (case 6). The nine carotid artery-only cases included six involving only the left internal carotid artery (cases 9, 10, 12, 13, 17, 18), one involving only the right internal carotid artery (case 8), one involving both internal carotid arteries (case 5), and one involving both common carotid arteries (case 16). Seven of the 14 cases exhibited signs of Horner's syndrome (9, 10, 12, 13, 16, 17, 18).

Most internal carotid artery dissections occurred at the base of the skull, proximal to the entry of the artery into the carotid foramen (Figure 2). The one exception involved a 35-year-old woman with multiple dissections (case 6). In this instance, the internal carotid dissections occurred at the origin of these vessels from the common carotid arteries. The two vertebral artery cases occurred before the artery entered a cervical vertebral transverse foramen.

The two intracranial cases (11 and 15) involved the left posterior cerebellar artery, originating from the left vertebral artery, and the left anterior cerebral artery, a terminal branch of the left internal carotid artery.

One patient (case 6), with multiple cervical artery dissections, had evidence of osteogenesis imperfecta, a collagen disorder reported as being associated with dissecting aneurysm.[34] This person had only minimal clinical evidence of the disorder but developed the dissections within minutes of her first dive experience. Two patients were known to be smokers (cases 12 and 17, the latter also diagnosed with stable multiple sclerosis), and one was known to have treated hypertension (case 13). None of the remaining patients had reported conditions predisposing to arterial dissection.

CORONARY ARTERY DISSECTION

Only one case of coronary artery dissection has been reported in association with scuba diving.[25] This was a 65-year-old female (case 19) with no known risk factors. Chest pain developed during descent on her ninth dive in five days. She developed a myocardial infarction as a result of the dissection but there was resolution of the arterial lesion with anti-platelet therapy.

We are unaware of any reports of arterial dissection of the splanchnic and peripheral arteries in scuba divers.

Discussion

Head and neck artery dissections have been reported associated with a wide range of sporting activities as well as from neck manipulations and trauma.[22,27,28] Some involved abnormal neck movements, such as chiropractic manipulation, others blunt trauma, such as in karate, and some seemingly innocuous activities such as jogging. Not infrequently there are no obvious potentially precipitating events.

All four cases of aortic dissection (type A) affected the initial segment of the aorta, which includes the areas directly or indirectly connected to the origins of the cervical arteries. It is logical to infer that the factors leading to aortic dissection are the same as those responsible for the dissections in other arteries. If aortic dissections are included, the evidence for a connection between vessel dissection and scuba diving is substantial.

Table 1

Sex, age, anatomical locations of dissecting arterial aneurysms, and possible contributing factors reported for divers. Depth – reported maximum depth of dive in meters. Timing of symptom onset: 1 – during dive; 2 – within minutes of surfacing; 3 – within hours of surfacing; 4 – within two days of surfacing. ACA – anterior cerebral artery; CCA – common carotid artery; Dash (–) – no information; ICA – internal carotid artery; L – left; NF – non-fatal; PICA – posterior-inferior cerebellar artery; PLVA – posterior left ventricular (coronary) artery, R – right; VA – vertebral artery; y – years

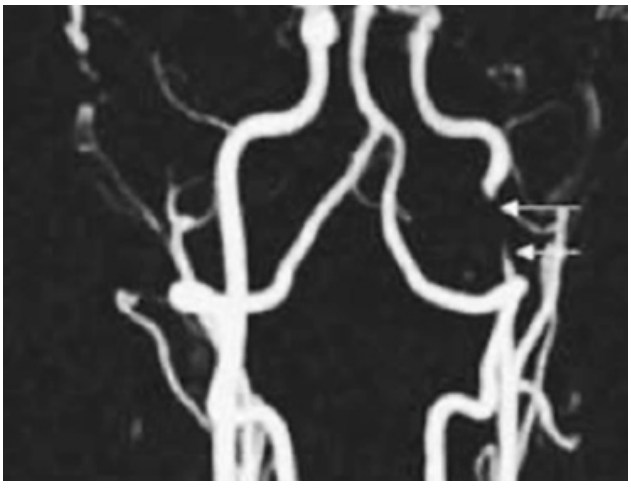
Case	Sex	Age (y)	Site	Dive description	Potentially relevant associations	Timing of symptom onset	Initial presentation of problem	Outcome
Aorta								
1[7,8]	M	20	Aorta	3 dives completed (36, 12, and 42 m) in one day; 15°C water	nil	2	Chest tightness noticed after second dive; chest pain when unsuiting after third dive; clinical check unremarkable; cardiac arrest during the night	Fatal
2[8]	M	63	Aorta	Resort dive (likely shallow and short)	–	2	Collapsed in dive boat post-dive	Fatal
3[9]	M	54	Aorta	3 min initial descent/surface	Ischaemic heart disease	1	Diver surfaces shortly after brief initial descent, vomiting with rapid loss of consciousness	Fatal
4[10]	M	60	Aorta	Developed chest pain ascending from 27 m dive	Hypertension, Dyslipidemia	1	Diver complains of chest pain starting during ascent and persisting post-dive; helicopter transport to hospital; patient survived emergency surgery	NF
Cervical or intracranial								
5[11]	M	52	ICA (R&L)	30 m for 25 min (mostly between 18–24 m); 3rd day of 1 dive/day pattern	nil	2	Headache "*within minutes of surfacing*" and dysphasia the next morning	NF
6[12]	F	35	VA (R&L), ICA (R.?L)	First lifetime dive	Osteogenesis imperfecta	2	Developed neck pain after first dive; evolving 2 days later	NF
7[13]	M	18	VA (R)	–	nil	4	R occipital headache, neck pain, and visual disturbance developed 2 days post-dive	NF
8[14]	F	38	ICA (R)	37 m max with "*several hours of diving*"	nil	1	Loss of consciousness at 2-3 msw at the end of unplanned rapid ascent; headache, L weakness, R anterolateral cervical neck discomfort, and mild aphasia 4 h later (during hospital assessment)	NF
9[15]	M	48	ICA (L)	8 m for 1.5 h	nil	2	Dizziness and stiff neck for 10 min immediately post-dive, neck pain and dysgeusia 2 days post-dive	NF
10[16]	M	51	ICA (L)	20 m (15 min near max) for 45 min total dive time; uneventful	nil	3	Developed slurred speech and difficulty finding words 3 hours post-dive (lasting 15 min); difficulties in swallowing and expressing words 2 days later; miosis and L ptosis	NF
11[17]	M	32	PICA (L)	–	nil	3	Persistent drowsiness experienced post-dive (reported 3 days later)	NF
12[18]	F	37	ICA (L)	Uneventful holiday dive; 22°C water	Smoking, multiple sclerosis	2	L facial paraesthia, L cervical pain, and L ear tinnitus starting within minutes post-dive; L nightly tension-neck pain noted 3 days later	NF
13[19]	M	52	ICA (L)	"*Scuba diving in cold water*"	Treated hypertension	4	Considerable and persistent neck pain radiating to left retroocular region starting the day after diving; L side facial edema, heaache, ptosis, miosis and neck pain worsened over days	NF
14[20]	M	27	VA (R)	"*Cold water*"	nil	3	Vertigo and gait imbalance developed 2 hours post-dive	NF
15[21]	M	51	ACA (L)	20 m for "*1 h*"	nil	1	Sudden R hemiparesis developed during dive (diver assisted to surface by instructor)	NF
16[22]	M	46	CCA (R&L)	–	–	–	Headache	NF
17[23]	M	32	ICA (L)	–	Smoking	2	L cervical pain and feeling of pressure in L ear starting within minutes post-dive; ipsilateral miosis and ptosis evident upon exam	NF
18[24]	F	60	ICA (L)	12 m max; uneventful	nil	2	Epistaxis and L frontal headache extending to L ear upon surfacing; L partial ptosis developed the next day; persistent ptosis and headache for 3 weeks	NF
Coronary artery								
19[25]	F	65	PLVA	30 m for 30 min (aborted from max depth); 9th dive in 5 days	nil	1	Sudden oppressive chest pain developed at 8 m during initial descent; cardiac signs and symptoms seen over following week	NF

The risk of dissection rises with increasing arterial pressure, such as in the case of individuals with untreated or poorly managed hypertension. Immersion and cooling stress increase arterial pressure through centralisation of blood volume. The increased partial pressure of oxygen produces an additional vasoconstrictive drive with concomitant increase in arterial pressure. An increase in arterial pressure can also be seen with constrictive suits or equipment, and also with breathing circuit resistance, more so as depth and respired gas density increases. It is unknown if the dive response may play a role in further augmenting arterial pressure in a spontaneously respiring diver, but it is at least theoretically possible. The reported cases of dissection in scuba divers all occurred in vessels where the dive response has been shown in animal studies to produce increased pulse pressure.[31] There are no reports of dissection involving peripheral or splanchnic arteries where the dive response is associated with vasoconstriction, but not with increased pulse pressure.

The cervical artery dissections identified in this review occurred at anatomical sites within those arteries that are distinct from the locations typically associated with neck manipulations or trauma. This finding suggests the involvement of different factors in their pathogenesis. In an earlier review of 24 patients with carotid artery dissection, none reportedly associated with scuba, one case involved the common carotid and the remainder the proximal internal carotid, the most mobile part of that vessel.[35] None involved the subcranial portion of the vessel, the region where the scuba-associated dissections occurred. The suggestion that neck hyperextension or other positioning or movements with diving play a key role in precipitating dissection in divers seems unlikely, but it is possible that restrictive equipment could create an additional burden of strain.

Figure 2

Magnetic resonance angiography from case 11; arrows show the dissection site in a sub cranial location where there is a 2 cm long with near total occlusion of the left internal carotid artery. From Prof M. Brodmann with kind permission



Cold stress has been demonstrated to induce the dive response[36] and/or to increase arterial pressures.[37] Exposure to cold water leads directly to increased parasympathetic stimulation and peripheral vasoconstriction, regulating body temperature homeostasis by increasing venous return to the heart. While the mask, continuous breathing, and thermal protection worn by divers may attenuate or abolish the dive response, activation is possible. Cold stress typically increases as a function of depth as neoprene insulation is compressed to become less effective and as divers cross thermoclines into colder water. It is possible that a quick chilling could initiate some degree of dive response.

Cervical artery dissections were generally seen to occur proximal to the entry points of the vessels into narrow bony spaces, these being the carotid canal for the internal carotid artery and the transverse foramina of the cervical vertebrae for the vertebral arteries. An enhanced pulse wave would be restricted at these points resulting in increased intraluminal arterial pressure. Instead of affecting the entire vessel, it is the specific region of arterial distension caused by the pulse wave that undergoes further stretching in accordance with the law of Laplace.[38] This increased distension could lead to elevated vessel wall shear stress with the potential for intimal tearing.

Pinnipeds (seals, sea lions and walruses) possess an elastic and bulbous ascending aorta,[39] an adaption that serves to reduce pulse pressure. It may be the lack of such an anatomical modification that promotes arterial dissection in human diving.

The dive response may also be activated in surface swimmers where there is total facial water exposure, yet reports of dissecting aneurysms in this group are relatively rare.[21] Other factors, such as muscular exertion, necessitating increased blood flow to the limbs, and regular breathing may serve to wholly or partially overcome the diver response. Surface swimmers also do not experience the strain of breathing circuit resistance and the depth-related increased gas density that divers must manage.

Three of the four reported cases of aortic dissection occurred in Australian waters, a region where only a small proportion of world diving occurs. One case occurred in a sinkhole diver in cold water, but the other two were in northern Australian waters that are relatively warm. It is unclear if water temperature may have played a contributing factor given the potential confounding of protective garments. Australia is noted for a robust system of dive fatality and dive incident investigation and reporting; it is possible that further cases have occurred elsewhere but remain unrecorded.

The only non-fatal case of aortic dissection was associated with quick recognition of a serious problem and helicopter evacuation to a medical centre, followed by transfer to

another hospital where emergency surgery was conducted. None of the cases of aortic dissection include detailed timelines to determine if the speed of transportation played a role in patient survival.

Coronary artery dissection is a rare cause of heart attacks and sudden cardiac death. It may be difficult to diagnose both clinically and radiologically. Even at post-mortem examination dissection may be confused with haemorrhage into an atheromatous plaque. The one case report of dissecting coronary artery dissection in a scuba diver could be an solitary event coincident with diving but it too may represent a failure of reporting or the failure of diagnosis of similar cardiac events.

IMPLICATIONS

Dissecting aneurysms should be considered among other conditions potentially affecting the collapsed diver, particularly when there has been no untoward event during the dive and in cases where there has been no response to recompression. Cardiopulmonary resuscitation will generally not be beneficial to a patient with aortic or arterial dissection.

Dissecting aneurysms of cervical vessels may present with neck or head pain, neurological disturbance, and Horner's syndrome. Horner's syndrome was observed in six of the eight patients with dissections confined to the internal carotid arteries. Horner's syndrome consists of a constellation of signs, including miosis (contraction of the pupil), ptosis (drooping of the upper eyelid), and anhidrosis (absent or reduced sweating on the affected side of the face). The syndrome is due to interruption of the sympathetic nerve supply to the eye and face.[40] This sympathetic supply reaches these areas through a network closely applied to the external layer (adventitia) of the carotid artery. Physical distortion of this network from arterial distension can result in the interruption of sympathetic transmission. Miosis is the most readily clinically recognised Horner's syndrome feature. Neck pain or tenderness, unequal pulses and disparity of pupil size should be looked for as signs of arterial dissection. Even with symptoms such as headache and confusion clinical signs may be absent and angiography necessary to establish a diagnosis.

With every dive incident, detailed dive history and profile should be recorded, together with dive conditions, including the water temperature, all equipment and thermal protection worn, the state of equipment and garments at the point of compromise, and a comprehensive timeline of all events. Prior medical disorders should be known before scuba diving. In fatal cases a meticulous autopsy is required, and an accurate cause of death established.

We speculate on the potential contribution of the dive response but cannot offer any estimate on the possible magnitude of the effect. Dive response effects are most commonly associated with breath-hold, which is not a normal situation for compressed gas divers. Further work is needed to determine whether any contribution goes beyond the theoretical to be practically important.

LIMITATIONS

The number of reported vascular dissection cases is small and inconsistently reported. There may be considerable under-reporting of scuba associated dissecting aneurysms both due to failure of diagnosis and failure of recording. Dissection of vertebral arteries may not be detected due to limited autopsy technique and aneurysm of a coronary artery may be misdiagnosed as haemorrhage into an atheromatous plaque. Single case reports are also not encouraged by many journals.

The available reports are inconsistent in descriptions of dive profiles (e.g., depth, duration, decompression obligations), water conditions (e.g., temperature, thermoclines, sea state, and entry/exit requirements), equipment worn by divers (e.g., scuba device, total weight, thermal protection), and event timelines through to completion. Incomplete and inconsistent autopsy records are also a problem. In fatal cases the vertebral arteries may not have been examined and the coronary arteries not specifically studied with serial transverse sectioning.

The impact of the diving activity in the two cases in which symptoms reportedly developed either on the next day or two days after diving (cases 7 and 4, respectively) is difficult to assess. It is possible that these were co-incidental and not causal events. Indeed, even in cases more proximally associated with diving a causal relationship cannot be confidently assumed.

Conclusions

Dissecting aneurysms of aorta, cervical, cranial and perhaps coronary arteries should be considered as a potential complication of scuba diving. The development of aortic and arterial aneurysms associated with scuba diving is likely multifactorial in pathogenesis. Detailed reporting is important in the evaluation of cases. The potential role of the diving response as a contributing factor in the pathogenesis of dissections requires further evaluation.

References

1 Lippmann J, Taylor D McD. Scuba diving fatalities in Australia 2001 to 2013: chain of events. Diving Hyperb Med. 2020;50:220–9. doi: 10.28920/dhm50.3.220-229. PMID: 32957123. PMCID: PMC7819731.

2 Tillmans F, editor. DAN annual diving report: 2020 edition: A report on 2018 diving fatalities, injuries and incidents. Durham (NC): Divers Alert Network; 2020. PMID: 35944087.

3 Lippmann J, Lawrence C, Davis M. Scuba diving-related

fatalities in New Zealand, 2007 to 2016. Diving Hyperb Med. 2021;51:345–54. doi: 10.28920/dhm51.4.345-354. PMID: 34897599. PMCID: PMC8920894.
4 Denoble PJ, Pollock NW, Vaithiyanathan P, Caruso JL, Dovenbarger JA, Vann RD. Scuba injury death rate among insured DAN members. Diving Hyperb Med. 2008;38:182–8. PMID: 22692749. [cited 2024 Aug 3]. Available from: https://dhmjournal.com/images/IndividArticles/38Dec/Denoble_dhm.38.4.182-188.pdf.
5 Tso JV, Powers JM, Kim JH. Cardiovascular considerations for scuba divers. Heart. 2022;108:1084–9. doi: 10.1136/heartjnl-2021-319601.
6 Lippmann J, Taylor D McD. Medical conditions in scuba diving fatality victims in Australia, 2001 to 2013. Diving Hyperb Med. 2020;50:98–104. doi: 10.28920/dhm50.2.98-104. PMID: 32557410. PMCID: PMC7481113.
7 James R, Hayman JA. Fatal dissecting aneurysm of the aorta in a diver. Pathology. 1986;18:345–7. doi: 10.3109/00313028609059488. PMID: 3785985.
8 Walker D. Report on Australian diving deaths 1972–1993. Ashburton, Australia: J.L. Publications; 1998.
9 Lippmann J, Lawrence C, Fock A. Compressed gas diving fatalities in Australian waters 2014 to 2018. Diving Hyperb Med. 2023;53:76–84. doi: 10.28920/dhm53.2.76-84. PMID: 37365124. PMCID: PMC10584389.
10 Yanagawa Y, Ohsaka H, Yatsu S, Suwa S. Acute aortic dissection during scuba diving. Undersea Hyperb Med. 2024;51:185–7. PMID: 38985154.
11 Nelson EE. Internal carotid artery dissection associated with scuba diving. Ann Emerg Med. 1995;25:103–6. doi: 10.1016/s0196-0644(95)70363-2. PMID: 7802358.
12 Mayer SA, Rubin BS, Starman BJ, Byers PH. Spontaneous multivessel cervical artery dissection in a patient with a substitution of alanine for glycine (G13A) in the alpha 1 (I) chain of type I collagen. Neurology. 1996;47:552–6. doi: 10.1212/wnl.47.2.552. PMID: 8757037.
13 Konno K, Kurita H, Ito N, Shiokawa Y, Saito I. Extracranial vertebral artery dissection caused by scuba diving. J Neurol. 2001;248:816–7. doi: 10.1007/s004150170102. PMID: 11596791.
14 Gibbs JW 3rd, Piantadosi CA, Massey EW. Internal carotid artery dissection in stroke from SCUBA diving: a case report. Undersea Hyperb Med. 2002;29:167–71. PMID: 12670119.
15 Skurnik YD, Sthoeger Z. Carotid artery dissection after scuba diving. Isr Med Assoc J. 2005;7:406–7. PMID: 15984390.
16 Bartsch T, Palaschewski M, Thilo B, Koch AE, Stingele R, Volkman J, et al. Internal carotid artery dissection and stroke after SCUBA diving: a case report and review of the literature. J Neurol. 2009;256:1916–9. doi: 10.1007/s00415-009-5221-4. PMID: 19557495.
17 Koçyiğit A, Çinar C, Kitis Ö, Çalli C, Oran İ. Isolated PICA dissection: an unusual complication of scuba diving: case report and review of the literature. Clin Neuroradiol. 2010;20:171–3. doi: 10.1007/s00062-010-0002-0. PMID: 20798911.
18 Hafner F, Gary T, Harald F, Pilger E, Groell R, Brodmann M. Dissection of the internal carotid artery after SCUBA-diving: a case report and review of the literature. Neurologist. 2011;17:79–82. doi: 10.1097/NRL.0b013e3181e6a416. PMID: 21364358.
19 Brajkovic S, Riboldi G, Govoni A, Corti S, Bresolin N, Comi GP. Growing evidence about the relationship between vessel dissection and scuba diving. Case Rep Neurol. 2013;5:155–61. doi: 10.1159/000354979. PMID: 24163671. PMCID: PMC3806682.
20 Chojdak-Łukasiewicz J, Dziadkowiak E, Bladowska J, Paradowski B. Vertebral artery dissection and stroke after scuba diving. Neurol India. 2014;62:711. doi: 10.4103/0028-3886.149455. PMID: 25591706.
21 Fukuoka T, Kato Y, Ohe Y, Deguchi I, Maruyama H, Hayashi T, et al. A case of anterior cerebral artery dissection caused by scuba diving. J Stroke Cerebrovasc Dis. 2014;23:1982–4. doi: 10.1016/j.jstrokecerebrovasdis.2014.02.016. PMID: 24784014.
22 Fragoso YD, Adoni T, do Amaral LL, Braga FT, Brooks JBB, Campos CS, et al. Cerebrum-cervical arterial dissection in adults during sports and recreation. Arq Neuropsiquiatr. 2016;74:275–9. doi: 10.1590/0004-282X20150150. PMID: 26445125.
23 Alonso Formento JE, Fernández Reyes JL, Envid Lázaro BM, Fernández Letamendi T, Yeste Martín R, Jódar Morente FJ. Horner's syndrome due to a spontaneous internal carotid artery dissection after deep sea scuba diving. Case Rep Neurol Med. 2016;2016:5162869. doi: 10.1155/2016/5162869. PMID: 27525139. PMCID: PMC4971302.
24 Wasik M, Stewart C, Norris JH. Delayed recognition of Horner syndrome secondary to internal carotid artery dissection after scuba diving. Clin Exp Ophthalmol. 2017;45:551–3. doi: 10.1111/ceo.12931. PMID: 28186387.
25 Mahendiran T, Desgraz B, Antiochos P, Rubimbura V. Case report: a first case of spontaneous coronary artery dissection potentially associated with scuba diving. Front Cardiovasc Med. 2022;9:855449. doi: 10.3389/fcvm.2022.855449. PMID:35497983. PMCID: PMC9046929.
26 Criado FJ. Aortic dissection: a 250-year perspective. Tex Heart Inst J. 2011;38:694–700. PMID: 22199439. PMCID: PMC3233335.
27 Rubinstein SM, Peerdeman SM, van Tulder MW, Riphagen I, Haldeman S. A systematic review of the risk factors for cervical artery dissection. Stroke. 2005;36:1575–80. doi: 10.1161/01.STR.0000169919.73219.30. PMID: 15933263.
28 Engelter ST, Traenka C, Grond-Ginsbach C, Brandt T, Hakimi M, Worrall BB, et al. Cervical artery dissection and sports. Front Neurol. 2021;12:663830. doi: 10.3389/fneur.2021.663830. PMID: 34135851. PMCID: PMC8200565.
29 Hatzaras I, Tranquilli M, Coady M, Barrett PM, Bible J, Elefteriades JA. Weight lifting and aortic dissection: more evidence for a connection. Cardiology. 2007;107:103–6. doi: 10.1159/000094530. PMID: 16847387.
30 Daily PO, Trueblood HW, Stinson EB, Wuerflein RD, Shumway NE. Management of acute aortic dissections. Ann Thorac Surg. 1970;10:237–47. doi: 10.1016/s0003-4975(10)65594-4. PMID: 5458238.
31 Panneton WM. The mammalian diving response: an enigmatic reflex to preserve life? Physiology (Bethesda). 2013;28:284–97. doi: 10.1152/physiol.00020.2013. PMID: 23997188. PMCID: PMC3768097.
32 Pedroso FS, Riesgo RS, Gatiboni T, Rotta NT. The diving reflex in healthy infants in the first year of life. J Child Neurol. 2012;27:168–71. doi: 10.1177/0883073811415269.
33 Scuitema K, Holm B. The role of different facial areas in eliciting human diving bradycardia. Acta Physiol Scand. 1988;132:119–20. doi: 10.1111/j.1748-1716.1988.tb08306.x. PMID: 3223302.
34 Balasubramanian M, Verschueren A, Kleevens S, Luyckx I, Perik M, Schirwani S, et al. Aortic aneurysm/dissection

and osteogenesis imperfecta: four new families and review of the literature. Bone. 2019;121:191–5. doi: 10.1016/j.bone.2019.01.022. PMID: 30684648.

35 Treiman GS, Treiman RL, Foran RF, Levin PM, Cohen JL, Wagner WH, et al. Spontaneous dissection of the internal carotid artery: a nineteen-year clinical experience. J Vasc Surg. 1996:597–607. doi: 10.1016/s0741-5214(96)70075-7. PMID: 8911408.

36 Speck DF, Bruce DS. Effects of varying thermal and apneic conditions on the human diving reflex. Undersea Biomed Res. 1978;5:9–14. PMID: 636078.

37 Wester TE, Cherry AD, Pollock NW, Freiberger JJ, Natoli MJ, Schinazi EA, et al. Effects of head and body cooling on hemodynamics during immersed prone exercise at 1 ATA. J Appl Physiol. 2009;106:691–700. doi: 10.1152/japplphysiol.91237.2008. PMID: 19023017.

38 Basford JR. The law of Laplace and its relevance to contemporary medicine and rehabilitation. Arch Phys Med Rehabil. 2002;83:1165–70. doi: 10.1053/apmr.2002.33985. PMID: 12161841.

39 Blix AS, Kuttner S, Messelt EB. Ascending aorta of hooded seals with particular emphasis on its vasa vasorum. Am J Physiol Regul Integr Comp Physiol. 2016;311(1):R144–9. doi: 10.1152/ajpregu.00070.2016. PMID: 27122367.

40 Shankar Kikkeri N, Nagarajan E, Sakuru RC, Bollu PC. Horner syndrome due to spontaneous internal carotid artery dissection. Cureus. 2018;10:e3382. doi: 10.7759/cureus.3382. PMID: 30519521. PMCID: PMC6263518.

Acknowledgments

The work of the late Douglas Walker in maintaining a record of fatal diving accidents in Australian waters over several decades (‘Operation Stickybeak’) provided the foundation for this work. Thanks also to Michael Panneton, James Caruso, and F Michael Davis for their feedback on various drafts of the manuscript.

Conflicts of interest and funding

Associate Professor Neal Pollock is a member of the editorial board of *Diving and Hyperbaric Medicine* but was not involved in the peer review or publication decision-making process for this article.

Submitted: 12 December 2023
Accepted after revision: 28 July 2024

Role of tympanocentesis in the prevention of middle ear barotrauma induced by fast buoyant ascent escape from 200 m underwater

Xu Liu[1,2]*, Hengrong Yuan[3]*, Jieying Peng[1]*, Guanghao Zhu[1], Nan Wang[3], Yukun Wen[3], Hongliang Zheng[1], Yiqun Fang[3], Wei Wang[1]

[1] *Department of Otolaryngology – Head and Neck Surgery, Changhai Hospital, Naval Medical University, Shanghai, China*
[2] *Department of Otorhinolaryngology and Head and Neck Surgery, No. 905 Hospital of PLA Navy, Shanghai, China*
[3] *Department of Diving and Hyperbaric Medicine, Naval Medical Center, Naval Medical University, Shanghai, China*
** Xu Liu, Hengrong Yuan and Jieying Peng contributed equally to this work*

Corresponding authors: *Professor Yiqun Fang, 880 Xiangyin Road, Yangpu District, Shanghai, China*
1287225836@qq.com
Dr Wei Wang, 168 Changhai Road, Yangpu District, Shanghai, China 200433
ORCiD: *0000-0001-9991-9356*
wangw0503@163.com

Keywords
Hearing loss; Submarine; Military diving; Deep diving; Rescue

Abstract
(Liu X, Yuan H, Peng J, Zhu G, Wang N, Wen Y, Zheng H, Fang Y, Wang W. Role of tympanocentesis in the prevention of middle ear barotrauma induced by fast buoyant ascent escape from 200 m underwater. Diving and Hyperbaric Medicine. 2024 30 September;54(3):196–203. doi: 10.28920/dhm54.3.196-203. PMID: 39288924.)
Introduction: We aimed to study middle ear barotrauma caused by fast compression followed by buoyant ascent escape from 200 m underwater and its effect on the auditory system, and to validate the preventive effect of tympanocentesis on middle ear barotrauma.
Methods: Twenty Sprague Dawley rats were divided into two groups: rats in group A underwent a simulated fast buoyant ascent escape from a depth of 200 m, while those in group B underwent tympanocentesis before the procedure described for group A. Ear endoscopy, acoustic conductance, and auditory brainstem response (ABR) tests were conducted before and after the procedure to evaluate the severity of middle ear barotrauma and auditory function in both groups. Additionally, histopathological examination of the middle ear in both groups was conducted to evaluate the severity of middle ear barotrauma by observing submucosal haemorrhage.
Results: None of the ears in either group showed any abnormalities before the experiment. In group A, middle ear barotrauma was universally observed after the simulation procedure. The tympanograms of all ears were initially type A and became type B after the procedure. Further, after the simulation, the hearing thresholds at different frequencies (4, 8, 16, 24, and 32 kHz) assessed by ABR significantly increased compared to those before the procedure. In group B, no middle ear barotrauma was observed, and the hearing threshold at each frequency did not change significantly compared with post-puncturing. After dissecting the middle ear, gross pathological observations were consistent with the above results. Microscopically, blood accumulation and submucosal haemorrhage in the middle ear cavity were observed in group A but not in group B.
Conclusions: Fast buoyant ascent from 200 m underwater can cause middle ear barotrauma, resulting in hearing loss. Tympanic membrane puncture can effectively prevent middle ear barotrauma caused by the rapid buoyant ascent escape procedure.

Introduction

Submarines play an important role in the combat power of naval forces. Owing to the special sailing environment during submarine missions, shipwrecks may occur during war or peacetime. Submarine accidents have been reported in America, the former Soviet Union, Russia, and China. Therefore, countries that operate submarines value lifesaving technologies for submariners. Fast buoyant ascent escape is an internationally recognised advanced measure for submarine self-rescue. The process can be briefly described in three steps: rapid pressurisation, short stay, and fast ascent. The theoretical maximal depth for fast buoyant ascent escape ranges from 180 m to 240 m, and the greater the depth, the greater the risk. Currently, China's research on fast-ascent escape technology is at the global forefront. According to published news reports, the maximum depth of human testing in China has reached 194.6 m which to our knowledge is the deepest achieved to date.

Middle ear barotrauma (MEBt), the most common diving-related injury, occurring in > 50% of experienced divers.[1] It

is also a common injury in the fields of hyperbaric oxygen treatment and aviation.[2,3] Preventative measures have been studied. For example, one study reported that MEBt can be avoided by reducing the rate of pressure change in patients receiving hyperbaric oxygen treatment.[4] This is not an option in fast buoyant ascent escape because it increases the exposure time to high pressure and the risk of fatal decompression sickness.

Some other studies suggested that in aviation and hyperbaric treatment, the use of devices such as Cirrus ear plugs, Otovent, Ear Popper®, and pressurised masks could help open the Eustachian tube, balancing the pressure on both sides of the eardrum, and therefore reducing the incidence of MEBt.[5–7] However, these devices are not suitable for fast buoyant ascent escape because of the extremely rapid pressure change and need for additional operations.

As a simpler procedure with a favorable prognosis, tympanocentesis has been proven effective in preventing MEBt in aviation and hyperbaric oxygen treatment.[7,8] This is the most feasible method for preventing MEBt caused by fast buoyant ascent escape. However, compared to hyperbaric oxygen treatment and aviation environments, in submarine escape the degree and rate of pressure change is much greater. Thus, this study aimed to ascertain the preventive effect of tympanic membrane puncture on MEBt in this extreme-pressure-change environment.

Methods

ETHICAL APPROVAL

Ethical approval for this research project was obtained from the Ethics Committee of the PLA Naval Medical Center under the ethics number AF-HEC-041.

ANIMALS

Twenty male Sprague-Dawley rats, weighing 180–300 g, were provided by Vital River Laboratory Animal Technology Co., Ltd. Before the experiment, all rats were kept at the animal centre of the Naval Medical Centre for more than one week. The rats were evenly and randomly divided into two groups (A and B). To obtain the pathological morphology of the normal middle ear, two other rats, which did not undergo any experimental intervention, had their middle ears dissected and isolated for pathological examination, which served only as normal controls.

For group A, auditory endoscopy, acoustic impedance, and auditory brainstem response (ABR) tests were performed before and after the rats experienced simulated fast buoyant ascent escape from 200 m underwater. The effects of this procedure were evaluated by comparing the two sets of results.

In group B, tympanic membrane puncture was performed using the needle of a 1 mL syringe on each rat before the simulated fast buoyant ascent escape from 200 m underwater. Auditory endoscopy and ABR tests were performed pre-puncture, post-puncture, and after the simulation procedure. The results of the three tests were compared to evaluate any preventive effect of tympanic membrane puncture on MEBt.

LABORATORY INSTRUMENTS AND EQUIPMENT

We used an animal simulation chamber for fast buoyant ascent escape (Yantai Hongyuan Oxygen Industry Co. Ltd, China). An OTOflex 100 middle ear analyser (Denmark Otometrics, Shanghai Sonbett Instrument Co. Ltd, China), a high-definition electronic ear endoscope (2.7 mm diameter, Shanghai Fiveboats Electronic Technology Co. Ltd, China) and a TDT auditory electrophysiological system (Tucker Davis Technologies, USA) were used to evaluate the middle ear.

SIMULATED FAST BUOYANT ASCENT ESCAPE FROM 200 M UNDERWATER

The rats were placed in a cabin that was quickly pressurised to 2.1 MPa (equivalent to 200 metres of seawater [msw]) using a $2^{t/4}$ exponential scheme. The rats remained at the target pressure for 4 s and were then decompressed to 0 msw at a speed of 3 $m \cdot s^{-1}$. The total decompression time was 67 s. The pressure adjustment process is illustrated in Figure 1.

OUTCOME MEASURES

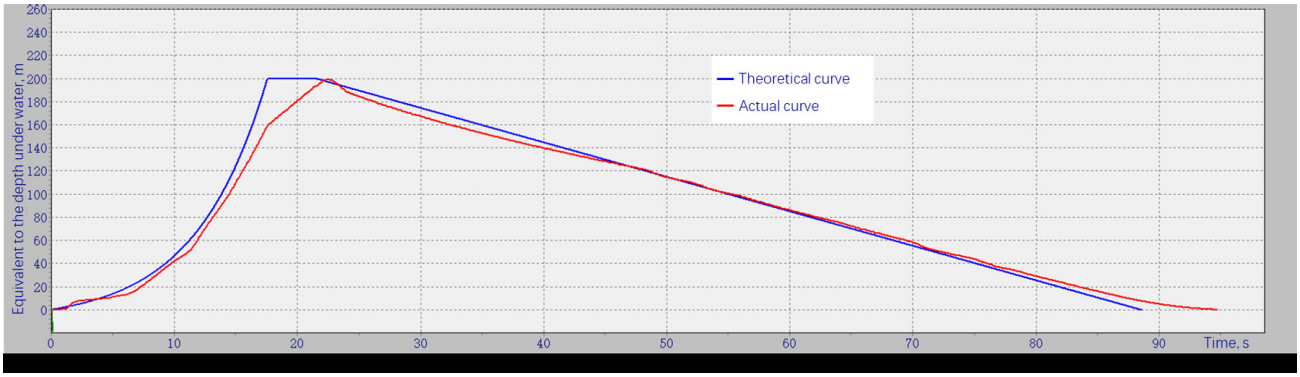
Tympanic membrane

The morphology of the tympanic membrane was observed using ear endoscopy at various stages of the experimental process, and the images were saved for subsequent analysis. The degree of MEBt was graded according to the O'Neill Injury Classification as follows. Grade 0 – no otologic signs of barotrauma on auricular endoscopy. Grade I – increased redness of the tympanic membrane compared with baseline, serous or slightly serosanguinous fluid, and/or trapped air behind the tympanic membrane. Grade II – bleeding at any location, perforation of the tympanic membrane, and/or free haemorrhage in the tympanic cavity.[3,9]

Tympanogram

An OTOflex 100 middle ear analyser was used to acquire the tympanogram of each ear before and after the simulation procedure to evaluate the conduction function of the middle ear. The middle ear analyser stimulation tone frequency was 1,000 Hz, and the pressure ranged from -400 daPa to 200 daPa at a speed of 400 $daPa \cdot s^{-1}$.

Figure 1
Curves showing pressure changes; the theoretical and actual pressure curves over time are shown in blue and red, respectively



ABR threshold

Auditory function was evaluated using the ABR test. The TDT auditory electrophysiological system (Tucker Davis Technologies, Alachua, FL, USA) was used to check the auditory threshold of each ear at different frequencies (4, 8, 16, 24, and 32 kHz). A custom-made soundproof chamber was used for testing. The effects of fast buoyant ascent escape from 200 m on the ears of rats were estimated by comparing the difference in ABR thresholds pre- and post-escape. The recording electrode was placed subcutaneously on the top of the head, the reference electrode was placed subcutaneously on the side of the mastoid process, and the ground electrode was placed subcutaneously on the contralateral mastoid process. The sound pressure level (SPL) decreased to a gradient of 10 dB from 90 dB. When a regular waveform could not be elicited, the SPL increased by 10 dB and then decreased in increments of 5 dB. The SPL, where wave II can be repeatedly elicited, was taken as the ABR threshold.[10]

Histopathological examination

After the examination, the rats were euthanised, and their middle ears were dissected and isolated. Hematoxylin and eosin staining was performed on the removed middle ears after decalcification. Tympanic haematocele and submucosal haemorrhage were observed under a microscope to provide evidence of MEBt severity.

STATISTICAL ANALYSIS

The auditory threshold was described by the mean and standard deviation (SD). A paired-sample *t*-test was used to analyse the difference between the two results in group A. For group B, analysis of variance for repeated measurements was applied to analyse the difference among the three results, while the least significant difference *t*-test was used as a post hoc test to make paired comparisons among different results. Fisher's exact test was used to analyse the difference in the incidence of MEBt between the two groups. Statistical significance was set at $P < 0.05$.

Results

ANIMALS

Three rats in Group A and two in Group B died during the experiment. The cause of death was considered an overdose of anesthetic drugs. Therefore, the number of samples were 14 ears from seven rats in group A and 16 ears from eight rats in group B.

TYMPANIC MEMBRANE

There was no abnormality of the tympanic membrane or effusion of the tympanic cavity observed by the otic endoscope in either group before the experiment. In contrast, MEBt was detected in all ears in group A after the simulated fast buoyant ascent escape from 200 m underwater. According to the O'Neill injury classification, grade I injury occurred in two ears and grade II injury in 12 ears, among which two ears had tympanic membrane perforation (Figure 2). In group B, no obvious MEBt was observed after the procedure, and the tympanocentesis perforation was not enlarged (Figure 3). There was a statistically significant difference in the incidence of MEBt between the two groups (Table 1, $P < 0.05$).

TYMPANOGRAM

In group A, the tympanograms of all ears showed a type A curve with a pronounced peak before the simulation procedure. After the simulation procedure, the tympanograms of all the ears changed to type B (Figure 4), showing that the conduction function of the middle ear was impaired. The rats in group B were not subjected to this test because they underwent tympanic membrane puncture, which changed the tympanogram to type B.

Figure 2
Photos of the varying severities of MEBt in tympanic membranes in group A; A – normal tympanic membrane; B – Grade I MEBt with hyperaemia and mild tympanic effusion; C – Grade II MEBt with haematotympanum; D – Grade II MEBt with tympanic membrane perforation

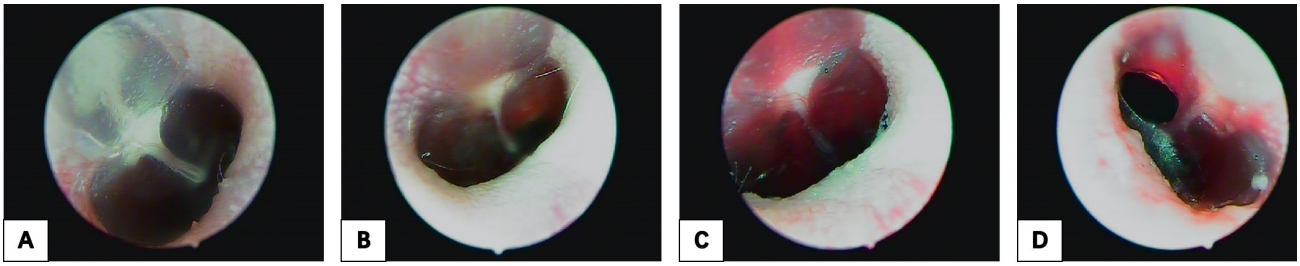


Figure 3
Photos of the tympanic membrane at different experimental stages in group B where no MEBt was observed; A – normal tympanic membrane before puncturing; B – after puncturing, the yellow arrow indicates the location of the perforation; C – after the emergency ascent simulation procedure

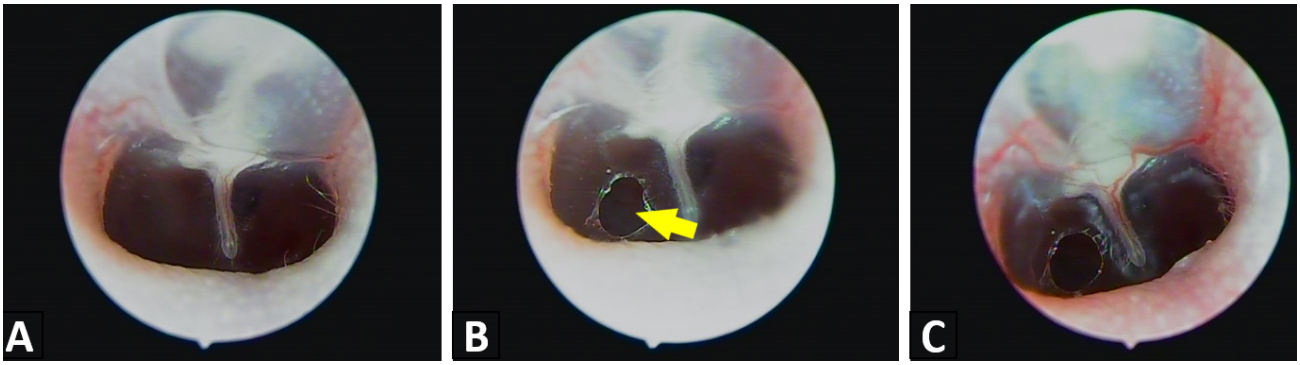


Table 1
Incidence of MEBt in two groups after simulated fast buoyant ascent escape from 200 m underwater; there was a significant difference in incidence between the two groups ($P < 0.05$)

Outcome by group	With MEBt	Without MEBt
Group B	0	16
Group A	14	0

ABR THRESHOLD

In group A, the average values of the ABR thresholds at frequencies of 4, 8, 16, 24, and 32 kHz after the simulation procedure were significantly higher than those before it, and the average ABR threshold values at each frequency before and after the procedure are shown in Table 2. A paired sample *t*-test was used to detect the difference in the ABR threshold before and after the simulation procedure, showing a statistically significant difference ($P < 0.05$). These results suggest that a simulated fast buoyant ascent escape from 200 m underwater can cause hearing loss in rats at all frequencies.

In group B, the average values of the ABR thresholds at each frequency were significantly increased after puncturing compared to pre-puncturing ($P < 0.05$), whereas there was no significant difference between post-puncturing and post-simulation procedure ($P > 0.05$). The average ABR threshold values at each frequency in Group B are shown in Table 3. These results indicate that tympanic membrane puncture causes hearing loss, while a simulated fast buoyant ascent escape from 200 m underwater after tympanocentesis does not cause further hearing loss.

HISTOPATHOLOGICAL EXAMINATION

In gross observation, as is shown in Figure 5, the middle ear samples were dark red in group A, indicating middle ear bleeding. However, similar to the normal controls, the samples in group B were white, suggesting the absence of bleeding in the middle ear cavity. The results described above were confirmed by observation at low magnification (Figure 6). Furthermore, under a high-magnification microscope, intratympanic and submucosal hemorrhages were observed in all ears in group A, regardless of perforation. No obvious submucosal bleeding was observed in group B or the standard control group. Interestingly, in group A, intratympanic haemorrhage was less severe, but submucosal haemorrhage was more severe in ears with

Figure 4

Tympanograms of experimental rats in group A; A – Type A curve before the simulation procedure; B – Type B curve after the simulation procedure

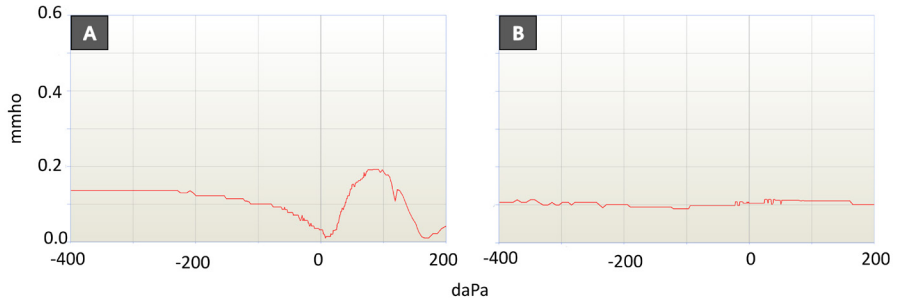


Table 2

Results of pre- and post-simulation ABR thresholds of rats in group A; data are mean (SD) dB; there was a significant difference pre- and post-simulation (fast buoyant ascent escape from 200 m underwater) at every frequency ($P < 0.05$)

Parameter	4 kHz	8 kHz	16 kHz	24 kHz	32 kHz
Pre-simulation	31.1 (5.3)	30.4 (6.0)	29.6 (6.0)	32.5 (4.3)	36.8 (4.2)
Post-simulation	71.1 (8.1)	63.2 (9.3)	68.6 (6.9)	71.8 (7.2)	72.9 (4.7)

Table 3

Results of pre- and post-simulation ABR thresholds of rats in group B; data are mean (SD) dB; * – statistically significant difference between pre-puncturing and post-puncturing at every frequency ($P < 0.05$); # – no statistically significant difference between post-puncturing and post-simulation (fast buoyant ascent escape from 200 m underwater) at every frequency ($P > 0.05$)

Parameter	4 kHz	8 kHz	16 kHz	24 kHz	32 kHz
Pre-puncturing*	31.9 (4.4)	31.6 (4.7)	28.8 (4.7)	31.9 (6.3)	33.4 (4.0)
Post-puncturing*#	50.9 (5.8)	52.2 (6.0)	50.3 (9.7)	47.5 (9.5)	47.5 (9.5)
Post-simulation#	49.7 (5.0)	53.4 (4.7)	49.4 (7.0)	49.4 (7.9)	50.3 (9.4)

perforation than in those without perforation (Figure 7). The most likely reason for this is that blood in the tympanic chamber flows out through the perforation, although perforation indicates a more serious injury.

Discussion

In this study, we assessed MEBt caused by fast buoyant ascent escape from 200 m underwater and its effect on the auditory system and evaluated the preventive effect of tympanocentesis on MEBt. The results showed that the incidence of MEBt in rats was 100% in group A, and most of them were seriously injured. However, prior tympanocentesis effectively protected rats from MEBt in group B.

The Eustachian tube connects the tympanic cavity to the external environment. Owing to the physiological characteristics of the 'unidirectional valve' of the eustachian tube,[11] it is necessary to actively open the pharyngeal orifices of the tube to balance the pressure inside and outside the tympanic cavity when environmental pressure changes rapidly. When the pressure difference reaches a level equivalent to 3 m of seawater depth (approximately 30 kPa), the eustachian tube closes completely. Once the eustachian tube is closed completely, it seldom reopens with the usual equalisation techniques, such as the Valsalva manoeuvre.[3] As the ambient pressure continues to increase, the pressure difference between the inside and outside of the tympanic cavity becomes more pronounced, resulting in MEBt.

Studies have shown a positive correlation between the duration of exposure to high pressure and the incidence of cardiopulmonary decompression sickness, which may lead to death in severe cases.[12,13] Therefore, it is necessary to shorten the exposure time to high pressures as much as

Figure 5

Gross specimen of middle ears in different groups; A – MEBt in group A without tympanic membrane perforation; B – MEBt in group A with tympanic membrane perforation; C – No MEBt in group B; D – Middle ear of normal control

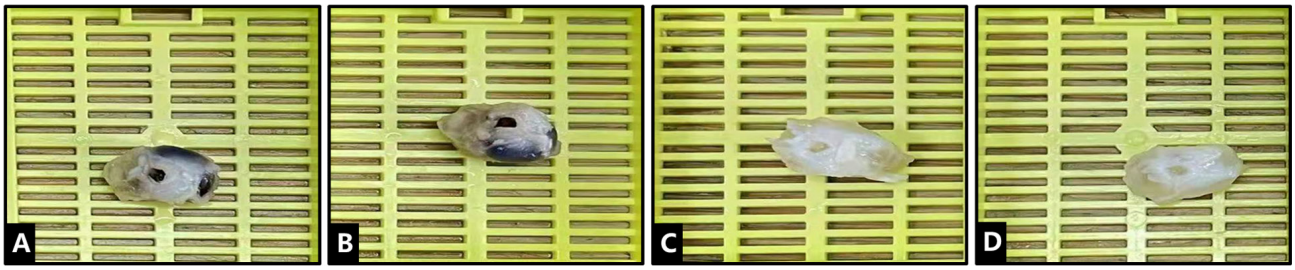


Figure 6

Hematoxylin and eosin staining of the mucosa of the tympanic cavity in different groups observed at low magnification (20×); A – moderate submucosal haemorrhage detected in group A without tympanic membrane perforation; B – obvious submucosal haemorrhage detected in group A with tympanic membrane perforation; C – no submucosal haemorrhage detected in group B without barotrauma; D – no submucosal haemorrhage detected in the middle ear of normal control

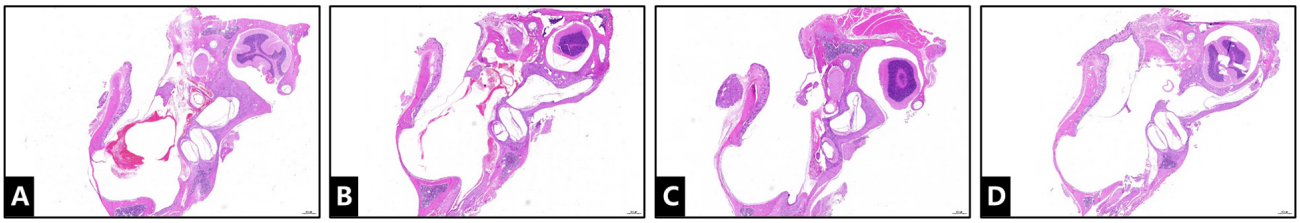
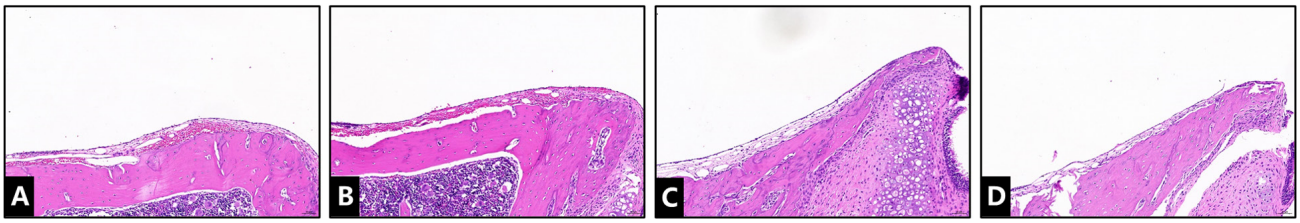


Figure 7

Hematoxylin and eosin staining of the mucosa of the tympanic cavity in different groups observed at high magnification (200×); A – moderate submucosal haemorrhage detected in group A without tympanic membrane perforation; B – obvious submucosal haemorrhage detected in group A with tympanic membrane perforation; C – no submucosal haemorrhage detected in group B without barotrauma; D – no submucosal haemorrhage detected in the middle ear of normal control



possible, which means that escaping submariners need to undergo pressurisation and initiate decompression extremely quickly. Studies have suggested that the severity of MEBt positively correlates with the rate of pressure change. Thus, during the procedure of fast buoyant ascent escape, if someone cannot open the Eustachian tube quickly, whether due to tube dysfunction or poor technique, MEBt would occur accompanied with symptoms such as pain, tinnitus, vertigo, and hearing loss.[3,5,14] Under these conditions, the safe operation of the escape equipment is affected, and the escape process may be compromised, perhaps catastrophically so.

The morphology, anatomy, and physiology of a rat's middle ear and Eustachian tube do not significantly differ from those of humans; therefore, rats are a suitable model for otological studies.[15] In clinical practice, 226 Hz tympanometry is the most commonly used method for adults. However, the anatomical structures of the ears in rats are similar to those of human babies.[16] Due to the characteristics of a small volume of the tympanic cavity and soft external auditory canal with good compliance, in our preliminary experiment, stable and repeatable tympanograms were not obtained from rats using 226 Hz tympanometry, consistent with the findings of others.[17]

It is generally accepted that 226 Hz tympanometry is not reliable for assessing the middle ear functional status of infants under seven months.[18,19] Several studies have shown that 1,000 Hz tympanometry is more accurate

than 226 Hz tympanometry for middle ear dysfunction in newborn babies.[20–22] The diagnostic consistency of 1,000 Hz tympanometry and temporal bone CT examinations in infants has been confirmed to be significantly higher than that of 226 Hz tympanometry.[23] Similarly, the diagnostic consistency of 1,000 Hz tympanometry and temporal bone magnetic resonance imaging in infants is significantly higher than that of 226 Hz tympanometry.[24] Therefore, in this study, 1,000 Hz tympanometry was used to evaluate the functional status of the middle ear to obtain stable and reliable test results.

For group A, our findings revealed that after undergoing a simulation of fast buoyant ascent escape from 200 m underwater, all experimental SD rats suffered obvious MEBt with symptoms of tympanic membrane hyperaemia or haemorrhage, haematotympanum, or tympanic membrane perforation. After the simulation procedure, the tympanograms of all ears changed from type A to type B, suggesting that the conduction function of the middle ear was impaired. Additionally, the ABR threshold in different frequencies increased significantly after the procedure, indicating that hearing loss occurred in experimental rats after simulated fast buoyant ascent escape from 200 m underwater.

In group B, tympanic membrane puncture effectively prevented MEBt caused by the simulation procedure. However, the hearing abilities of the rats decreased significantly after the needle puncture (approximately 0.4 mm in diameter). Based on clinical experience, the commonly used puncture needle for tympanocentesis is about 0.7–0.9 mm in diameter, and such perforation does not cause significant hearing loss in patients. The results of animal experiments are inconsistent with clinical experience. Studies have shown that the degree of hearing loss caused by tympanic membrane perforation is positively correlated with the size of tympanic membrane perforation and negatively correlated with the volume of the mastoid air chamber in the middle ear.[25,26] Some scholars have also pointed out that the degree of hearing loss caused by tympanic membrane perforation positively correlates with the percentage of perforated areas in the total tympanic membrane.[27] ImageJ software was used to measure the size of the tympanic membrane and perforation. The percentage of the perforated area was then calculated. In our study, the measurements and calculations showed that the perforated area accounted for approximately 10% of the tympanic membrane area. As is known, the area of the tympanic membrane of the human ear is about 55 mm^2, and the diameter of the tympanic membrane puncture needle usually used in clinical practice is about 0.7–0.9 mm. The percentage of the tympanic membrane perforation area caused by the needle in clinical practice is approximately 0.7–1.2%. The percentage of the tympanic membrane area of rats accounted for by tympanic membrane perforation in this study was much larger than that of human eardrum perforation caused by tympanic membrane puncture in clinical work, which may explain the significant hearing loss in rats caused by puncture (tympanocentesis) in this study.

There was no significant difference in the ABR thresholds of rats in group B after the simulated fast buoyant ascent escape from 200 m underwater compared to that after puncturing, indicating that after the tympanic puncture, the simulated fast buoyant ascent escape procedure did not lead to further hearing loss in rats. In other words, tympanic puncture had a protective effect against hearing loss caused by the simulation procedure.

Histopathological examination showed intratympanic and submucosal haemorrhages in all ears with MEBt in group A. However, the intratympanic haemorrhage was less severe, but submucosal haemorrhage was more severe in ears where barotraumatic perforation occurred than in those without. Eardrum perforation indicates more severe barotrauma; however, blood in the tympanic chamber flows out through the perforation, whereas submucosal blood does not. Therefore, the extent of submucosal tympanic haemorrhage can be used as an objective reference index to evaluate the severity of MEBt.

An invasive operation such as a tympanic membrane puncture should have the smallest diameter while ensuring safety and minimising injury. Therefore, the critical minimum diameter value should be determined in future studies. Second, there may be an incidence of harm associated with myringotomy by casual operators in a subsunk situation. Therefore, it is necessary to explore how to perform tympanic membrane puncture handily, promptly and safely in emergency cases. The above problems are the focus of our further research.

Conclusions

Fast buoyant ascent escape from deep depths can lead to MEBt and hearing loss. Barotrauma occurred in all animals not submitted to tympanocentesis, whereas tympanocentesis prevented ear injury.

References

1 Covington D, Pitkin A. Underwater nasal decongestant use: a novel approach to middle ear equalization. Undersea Hyperb Med. 2018;45:679–82. PMID: 31158935.

2 Varughese L, O'Neill O, Marker J, Smykowski E, Dayya D. The effect of compression rate and slope on the incidence of symptomatic Eustachian tube dysfunction leading to middle ear barotrauma: a Phase I prospective study. Undersea Hyperb Med. 2019;46:95–100. PMID: 31051053.

3 O'Neill OJ, Brett K, Frank AJ. Middle ear barotrauma. Treasure Island (FL): StatPearls Publishing LLC. 2021. PMID: 29763026.

4 Hwang L, Song M, Lee Y, Shin T. Methods for preventing middle ear barotrauma in computer-controlled pressurization

of monoplace hyperbaric chambers. Undersea Hyperb Med. 2019;46:107–16. PMID: 31051055.
5 Mirza S, Richardson H. Otic barotrauma from air travel. J Laryngol Otol. 2005;119:366–70. doi: 10.1258/0022215053945723. PMID: 15949100.
6 O'Neill O, Smykowski E, Marker J, Perez L, Gurash S, Sullivan J. Proof of concept study using a modified Politzer inflation device as a rescue modality for treating Eustachian tube dysfunction during hyperbaric oxygen treatment in a multiplace (Class A) chamber. Undersea Hyperb Med. 2019;46:55–61. PMID: 31154685.
7 Wu J, Zheng Z, Wang X, Mao H, Cheng P. The prevention of the ear barotrauma by positive pressure on nasopharynx. Lin Chuang Er Bi Yan Hou Ke Za Zhi. 2006;20:878–9. PMID: 17168113. Chinese.
8 Jiang D, Zhang X, Sun J, Liu Y. Experimental investigation of tympanostomy for preventing barotrauma. Chin J Aerospace Med. 2009;20:283–6. doi: 10.3760/cma.j.issn.1007-6239.2009.04.013.
9 Hamilton-Farrell M, Bhattacharyya A. Barotrauma. Injury. 2004;35:359–70. doi: 10.1016/j.injury.2003.08.020. PMID: 15037370.
10 Gedik Ö, Doğan R, Babademez M, Karataş E, Aydın M, Koçyiğit A, et al. Therapeutic effects of metformin for noise induced hearing loss. Am J Otolaryngol. 2020;41:102328. doi: 10.1016/j.amjoto.2019.102328. PMID: 31732304.
11 Yule C. The mechanism of opening and closing the Eustachian Tube. J Anat Physiol. 1873;8:127–32.1. PMID: 17230993. PMCID: PMC1319011.
12 Yiqun F, Pu Y, Haitao W, Xiaochen B, Jun M, Shi Z, et al. Metabonomic potential plasma biomarkers in abnormal fast buoyancy ascent escape-induced decompression sickness model and the protective effects of pyrrolidine dithiocarbamic acid. Undersea Hyperb Med. 2017;44:109–19. doi: 10.22462/3.4.2017.4. PMID: 28777901.
13 Klapa S, Meyne J, Kähler W, Tillmans F, Werr H, Binder A, et al. Decompression illness with hypovolemic shock and neurological failure symptoms after two risky dives: a case report. Physiol Rep. 2017;5:e13094. doi: 10.14814/phy2.13094. PMID: 28325788. PMCID: PMC5371546.
14 Lynch JH, Deaton TG. Barotrauma with extreme pressures in sport: from scuba to skydiving. Curr Sports Med Rep. 2014;13:107–12. doi: 10.1249/jsr.0000000000000039. PMID: 24614424.
15 Van Heerbeek N, Tonnaer ELGM, Ingels KJAO, Curfs JHAJ, Cremers CWRJ. Effect of exogenous surfactant on ventilatory and clearance function of the rat's eustachian tube. Otol Neurotol. 2003;24:6–10. doi: 10.1097/00129492-200301000-00003. PMID: 12544020.
16 Daniel H, Fulghum R, Brinn J, Barrett K. Comparative anatomy of eustachian tube and middle ear cavity in animal models for otitis media. Ann Otol Rhinol Laryngol. 1982;91:82–9. doi: 10.1177/000348948209100118. PMID: 7073182.
17 Zheng H, Li X, Yu L. Study of 1000 Hz probe tone tympanometry on examination of fluid accumulation in tympanic cavity. Chin Arch Otolaryngol Head Neck Surg. 2008;15:79–81. doi: 10.3969/j.issn.1672-7002.2008.02.006.
18 Holte L, Margolis R, Cavanaugh R. Developmental changes in multifrequency tympanograms. Audiology. 1991;30:1–24. doi: 10.3109/00206099109072866. PMID: 2059166.
19 McKinley A, Grose J, Roush J. Multifrequency tympanometry and evoked otoacoustic emissions in neonates during the first 24 hours of life. J Am Acad Audiol. 1997;8:218–23. PMID: 9188078.
20 Meyer S, Jardine C, Deverson W. Developmental changes in tympanometry: A case study. Br J Audiol. 1997;31:189–95. doi: 10.3109/03005364000000021. PMID: 9276101.
21 Kei J, Allison-Levick J, Dockray J, Harrys R, Kirkegard C, Wong J, et al. High-frequency (1000 Hz) tympanometry in normal neonates. J Am Acad Audiol. 2003;14:20–8. doi: 10.3766/jaaa.14.1.4. PMID: 12833925.
22 Margolis R, Bass-Ringdahl S, Hanks W, Holte L, Zapala D. Tympanometry in newborn infants – 1 kHz norms. J Am Acad Audiol. 2003;14:383–92. PMID: 14620612.
23 Zhiqi L, Kun Y, Zhiwu H. Tympanometry in infants with middle ear effusion having been identified using spiral computerized tomography. Am J Otolaryngol. 2010;31:96–103. doi: 10.1016/j.amjoto.2008.11.008. PMID: 20015724.
24 Yang K, Liu Z. Comparison of 1000 Hz-, 226 Hz-probe tone tympanometry and magnetic resonance imaging in evaluating the function of middle ear in infants. Int J Pediatr Otorhinolaryngol. 2020;136:110135. doi: 10.1016/j.ijporl.2020.110135. PMID: 32544643.
25 Aslıer M, Özay H, Gürkan S, Kırkım G, Güneri E. The effect of tympanic membrane perforation site, size and middle ear volume on hearing loss. Turk Arch Otorhinolaryngol. 2019;57:86–90. doi: 10.5152/tao.2019.4015. PMID: 31360926. PMCID: PMC6640663.
26 Mehta R, Rosowski J, Voss S, O'Neil E, Merchant S. Determinants of hearing loss in perforations of the tympanic membrane. Otol Neurotol. 2006;27:136–43. doi: 10.1097/01.mao.0000176177.17636.53. PMID: 16436981. PMCID: PMC2918411.
27 Choffor-Nchinda E, Djomou F, Meva'a Biouele R, Mindja D, Bola A, Kewe I, et al. Determinants of hearing loss severity in tympanic membrane perforations in a sub-Saharan African setting. J Laryngol Otol. 2018;132:1013–7. doi: 10.1017/s0022215118001962. PMID: 30409241.

Acknowledgements

The work was supported by the Major applied basic research projects of PLA army (AWS16J033), the National Natural Science Foundation of China (81600791), the PLA Navy logistics key project (BHJ06J023), the Military Medical Science and Technology Youth Cultivation Program of PLA (14QNP070), the Changhai Hospital Basic Special Research Foundation (2021JCMS14), and the Special Project for Incubation of Civil-Military Integration Achievements (2020-RP07).

Conflicts of interest and funding: nil

Submitted: 12 May 2024
Accepted after revision: 4 August 2024

Technical reports

Acoustic emission, an innovative diagnosis tool for therapeutic hyperbaric chambers: or how to requalify safely using pneumatic pressure test

Johann Catty[1], Olivier Seguin[2], Jean-Laurent Juillie[2], Daniel Mathieu[2], Erika Parmentier-Decrucq[2]

[1] *CETIM, F-60304 Senlis Cedex, France*
[2] *Intensive Care Unit and Hyperbaric Center, Lille University Hospital, F-59037 Lille Cedex, France*

Corresponding author: *Johann Catty, CETIM, Pôle MCO, 52 Avenue Félix Louat, F-60304 Senlis Cedex, France*
ORCiD: *0009-0006-4853-2825*
johann.catty@cetim.fr

Keywords
Bioengineering; Equipment; Hyperbaric facilities; Hyperbaric oxygen treatment; Therapeutic hyperbaric chamber; Requalification

Abstract

(Catty J, Seguin O, Juillie J-L, Mathieu D, Parmentier-Decrucq E. Acoustic emission, an innovative diagnosis tool for therapeutic hyperbaric chambers: or how to requalify safely using pneumatic pressure test. Diving and Hyperbaric Medicine. 2024 30 September;54(3):204–211. doi: 10.28920/dhm54.3.204-211. PMID: 39288925.)
Therapeutic hyperbaric chambers require continuous monitoring and maintenance, including periodic requalification. The primary aim is to verify the suitability for continued safe service. Maintenance is regulated in Europe, and in France requalification is mandatory where a hyperbaric chamber operates above pressures equal to or greater than 4 bar gauge. French requalification requires a hydraulic (hydrostatic) pressure test to determine the absence of deformation and leaks during the test. However, in such cases, it is often necessary to move the chamber if the combined mass of the chamber and water may exceed the allowable floor loading strength. In 2009, an innovative alternative to a hydraulic pressure testing was authorised in France. It consists of carrying out a pneumatic pressure test simultaneously with a non-destructive monitoring technique called 'acoustic emission'. This can be compared to a microseismology technique, where sensors are applied to the pressure retaining boundary of the hyperbaric chamber, and signals emitted by the vessel under load are captured. These signals are analysed, prioritised, and classified, to determine the physical position of any sources (artifacts) through triangulation calculations. This technique makes it possible to assess the behaviour of the vessel very accurately in real time and, *a posteriori*, to assess its fitness for continued service. This technique reduces the unavailability time of the chamber to two days, compared to potentially several weeks when a hydraulic test is performed. Over and above financial considerations and availability of facilities, this technique provides a baseline of the integrity of pressure vessels and allows monitoring over time of any potential deterioration.

Introduction

All pressure vessels, and especially those intended for human occupancy, require continuous inspection and periodic maintenance. Depending on local regulations, inspection and testing of the integrity of the pressure boundary may be legally mandated. In some European countries, and particularly in France, this periodic requalification is mandatory every 10 years for pressure vessels operating at a pressure equal to or greater than 4 Bar gauge (Barg).[1–4] In France, this requalification is carried out by means of a hydraulic (hydrostatic) test, usually conducted using water. The test may require, for reasons of insufficient allowable floor loads, moving the hyperbaric chamber to a more secure location. This allows for it to be filled with water and then pressurised to a pre-determined test pressure value (referred to as a hydrostatic test). The chamber is then checked for the absence of deformation and leaks. This entails a complex operation as internal equipment is usually required to be dismantled and removed. External equipment needs to be isolated from exposure to both water and elevated pressure. This is expensive, results in the hyperbaric chamber being out of service for a significant period of time, and can even cause damage (hydrostatic test pressure is more than the chamber's design pressure). Many centres do not perform this ten-year requalification, deeming it too costly or too restrictive. The hyperbaric chamber may then be downgraded and lose its ability to be compressed to equal to or greater than 4 Barg. Note that no therapeutic hyperbaric chamber limited to 4 Barg can perform HBOT at 4 Barg. In fact, the

chambers are equipped with safety valves which must open at the authorised pressure limit. This limit cannot therefore be the pressure prescribed for treatment.

Given our clinical activity, it was deemed essential to maintain the possibility of higher pressure exposures for several reasons. The first is to be able to test our equipment.[5] Any non-CE approved hyperbaric equipment must be pressurised to our maximum, normal working pressure of 3 Barg for certain indications (air embolism, decompression accidents)[6] and may even require testing beyond this working depth. Our validation procedures require that we test applicable patient care equipment to a higher pressure than the intended maximum working pressure. Therefore, without the capability of being pressurised to at least 4 Barg, beds, glucometers or other equipment essential to the proper care of patients would not be authorised for use in our hyperbaric chamber. Testing of equipment above 4 Barg is financially very challenging.[7] We also offer an educational service to diving clubs through a visit to our centre, where we provide an awareness of narcosis through psychometric tests during a dry dive to 4 or 5 Barg. Finally, diploma training and assessing aptitude for work in a hyperbaric environment requires training where students undertake dry dives at 5 Barg in order to obtain certification as a medical hyperbaric worker.[8] There are no longer any strong indications to use therapeutic exposures at more than 3 Barg.

Hyperbaric chambers are therapeutic devices often installed in areas with reduced visual access. Most of them are in high demand. Indeed, each treats numerous patients, some of whom have urgent and vital indications such as gas embolism,[9] decompression sickness,[10] carbon monoxide poisoning[11] or gas gangrene.[12] Closing a hyperbaric chamber, even temporarily, can compromise a healthcare system.[13] Requalification, although necessary, must close the hyperbaric chamber for as short a time as possible. Finding an alternative to the hydraulic test was therefore desirable. However, a pneumatic test on its own is not allowed in France. An acoustic emission test carried out during a pneumatic pressure test was the only solution, allowed under French legislation since 2009.[3] This innovative technique goes beyond the simple possibility of replacing the hydraulic test, as it allows a more precise and reliable diagnosis of the structural integrity of the vessel. Our hyperbaric centre considered this alternative, evaluated its feasibility performed the testing, and is publishing its conclusions so that other centres may potentially benefit from it.

Methods

Acoustic emission testing is performed by installing passive piezoelectric sensors (resonant frequencies between 100 and 500 kHz) on a pressure vessel. Each position and distance to neighbouring sensors is defined by:

- regulatory rules, codes and guides such as the ASME Boiler and Pressure Vessels Code,[14] European Standards (EN14584)[15] and the French technical guide for good practices for acoustic emission control of pressure equipment;[3]
- a desired location accuracy objective (basic location by area or precise planar location); and
- the experience of the qualified person in charge of the tests, who will be able to adapt the mesh (position of the sensors on the structure) to the specificities of the vessel, such as geometric discontinuities.[16]

Once the sensors are in place, a data acquisition system records the acoustic signals generated by the vessel when pressurised. This system, coupled with a computer, provides data in real time.

REQUIREMENTS RELATED TO TEST PRESSURE

The pressurisation cycle is made up of phases of pressure rise, in increments, and successive depressurisations. In order to respect code- or standards-defined constraints on one hand and technical constraints on the other, the maximum test pressure corresponds to the greater of the following two values:

- the value linked to the acoustic emission technique, which requires applying pressure at least 10% in excess of the actual maximum pressure applied in service in the preceding six months (‘110% actual maximum pressure’); or
- the applicable regulation rules, which may require a test pressure of between 90% and 110% of the maximum allowable pressure.

CONFIGURATION OF INSTRUMENTATION USED

The configuration recommended for carrying out such an examination control is a mesh of sensors sufficiently dense to allow planar localisation. Thus, any source of acoustic emission at least as energetic as the reference source (called Hsu-Nielsen source) can be located by triangulation, which is based on the same principles for locating the epicentres of earthquakes in the field of seismology.

INSTALLATION AND VERIFICATION OF SENSITIVITIES OF THE SENSORS

The sensors used for this type of application are 150 kHz piezoelectric resonant (ultrasonic) sensors. This frequency in the ultrasonic range is most suitable for detecting damage phenomena in metallic materials (such as cracking, microcracking, etc.). In addition, being far from the audible frequency domain, acoustic emission can be used in a wide range of industrial environments.

Once the sensors are installed and connected to the data acquisition system via a preamplifier, the sensitivity level is checked using a reference source. This phase is essential to ensure the quality of the coupling between the sensor and the pressure vessel, and therefore ensuring good transmission of the ultrasonic waves. These installation, verification and

thereafter dismantling phases can be carried out during normal operation of the chamber.

ANALYSIS AND INTERPRETATION OF DATA

Acoustic emission is an extremely simple technique in principle, but demanding in terms of analysis. Often considered as a special, even exotic technique in the field of non-destructive testing, due to difficulties in understanding data processing, it needs to comply with the relevant codes and standards for all phases of application, including rigorous analysis of signals.[3,14,15,17–19]

The analysis of a test takes place in two stages:

1. Analysis in real time, i.e., when pressure is applied to the vessel. This analysis integrates shutdown criteria, making it possible to secure the test under pneumatic pressure, and anticipates potential premature failure of the equipment.
2. A delayed-time analysis which leads to precise conclusions on the strongest emitting areas of the vessel. This emissivity is quantified by classification into categories (from category 1: notable/non-critical acoustic activity to category 3: intense acoustic activity, requiring additional investigation).

At the end of a test, the qualified person in charge of acoustic emission examination can immediately provide initial indications on the state of health of the vessel.

Whether in real or delayed time, several types of analysis, from the simplest to the most complex, make it possible to assess the integrity of the vessel.

Analysis of the background noise level of each sensor

This is carried out by measurement of the effective voltage of the signal (root mean square voltage value). This makes it possible to detect continuous phenomena, such as a leak, with great sensitivity.

Zonal analysis

Zonal analysis focuses on impulse signals (called bursts) whose origin can be the progression of microcracks or fracture of fragile corrosion layers (all discrete phenomena by nature). It consists of allocating a series of bursts resulting from the same physical phenomenon to the first sensor reached. We can therefore simply localise the source, although without significant accuracy. This analysis has the advantage of being simple, defined in the codes and standards, and results in the classification (into three categories) of each area covered by a sensor using numerous analysis criteria.

Planar localisation-based analysis

This is the most complex processing involving calculating the coordinates of the epicentre of the acoustic emission sources by carrying out triangulation calculations based on the arrival times of the signals. The accuracy of the result depends on several factors:

- the quality of the mesh of sensors used (number, distance between sensors, placement accuracy, etc.);
- the wave propagation speed; and
- the precision of determining the signals arrival times (which requires precisions of less than a microsecond).

This analysis results in a map of the acoustic activity of the monitored vessel. The most emissive regions of the vessel are automatically identified using the concept of 'clusters' (spatial grouping of several events). Used in the field of acoustic emission for nearly 30 years, whether in real time (during the test) or in delayed time, this methodology makes it possible to identify the most active regions very quickly and precisely. However, it is important to emphasise that the reliable use of planar localisation requires:

- a denser network of sensors than for simple zonal localisation;
- optimal listening conditions requiring the total cessation of sessions in the tested hyperbaric chambers; and
- mastery of all calculation parameters by the engineers in charge of these tests.

Today, the most advanced codes and standards in the field of acoustic emission, applied to the evaluation of pressure equipment, strongly recommend the systematic application of planar localisation. Consequently, this applies to the instrumentation conditions to achieve this, by proposing a methodology which makes it possible to quantify the expected performance of a sensor network (ASME, BPVC 2023, Section V, Article 12 – Mandatory Annex n°3).[14]

REQUIREMENTS RELATED TO PRESSURE

Our hyperbaric chamber is made up of two compartments ('Poseidon' and 'Scylla') and can reach a pressure of 6 Barg. Therefore, the maximum test pressure was set at 6.6 Barg. Figure 1 illustrates the pressurisation cycle defined in the test procedure and which must be applied in the case of the Poseidon hyperbaric chamber.

INSTRUMENTATION USED

Hyperbaric chambers often have complex shapes and are made up of several elements. The rules of the acoustic emission technique had to be adapted to these specificities. We have thus taken advantage of the localisation capabilities of this technique. In doing so, it becomes possible to detect and locate any defects potentially harmful to the vessel with a precision of a few centimetres for a hyperbaric chamber having a total volume of more than 30 m^3.

A precise mesh of sensors adapted to the geometries of the Poseidon chamber and the Scylla airlock was defined based on acoustic wave attenuation measurements (Figure 2). The mesh of sensors also takes into account the geometric

Figure 1
Theoretical pressurisation cycle (the Poseidon hyperbaric chamber and its airlock); Barg – pressure in Bar (gauge)

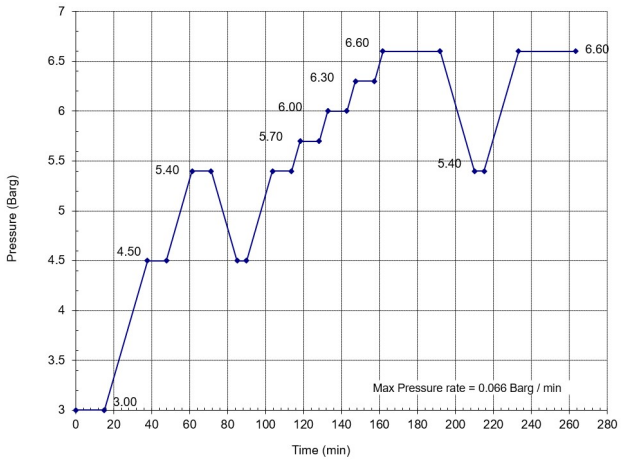


Figure 2
Typical attenuation curve taken into account (hyperbaric chamber - direction perpendicular to the reinforcements); dB_{ae} – acoustic emission signal amplitude unit

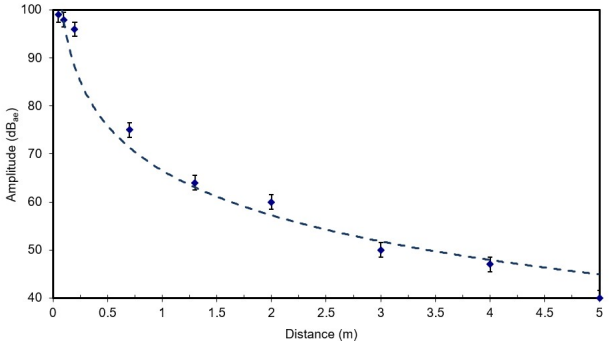


Figure 3
General view of the sensors mesh for Poseidon chamber (the position of the sensors is symbolised by yellow circles)

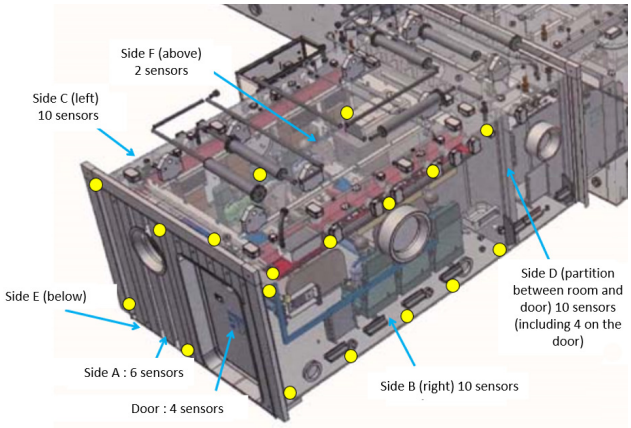
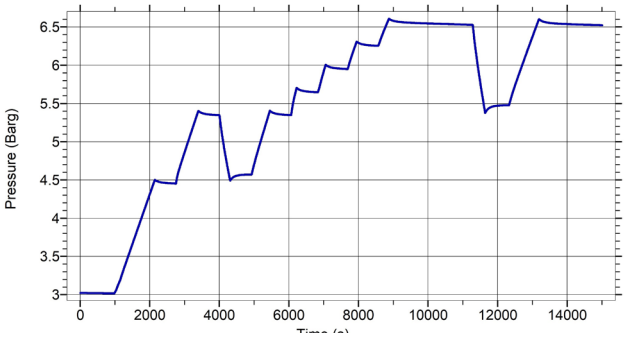


Figure 4
Actual pressurisation cycle (Scylla airlock); Barg – pressure in Bar (gauge)



particularities of these vessels such as the square chamber shell, the presence of numerous reinforcements (which cause propagation asymmetry), discontinuities such as doors (which disrupt the propagation of waves), and areas sometimes inaccessible. Thus, beyond compliance with codes, standards and application guides, it is appropriate to have a sufficient level of expertise to define a network of sensors (43 sensors were installed for Poseidon and 28 for Scylla) to ensure complete coverage of each vessel (Figure 3).

OPERATING PROCEDURE AND PREPARATION AHEAD OF THE REQUALIFICATION OPERATION

During the months preceding the deadline we had to collect technical acoustic emission data, in order to define and configure the means of pneumatic pressurisation, and prepare the necessary documents so that the authorised body could give us a favourable notice on the file.

PRESSURE TESTING AND ITS REAL–TIME MONITORING

Compliance with the pressurisation cycle defined in the test procedure is essential. Also, the engineers in charge of the hyperbaric chambers at the Lille University Hospital configured the means of pressurisation to allow, on the one hand, the recommended cycle to be respected (Figure 4), but on the other hand, to make pressurisation of the vessel as quiet as possible (so as not to disturb the sensors).

In this case, it made it possible to carry out these tests highly accurately and effectively, and thus respect the planning constraints, namely test completion before noon, oxygen therapy sessions resumed as planned at the beginning of the afternoon.

Results

ANALYSIS AND INTERPRETATION OF DATA

Analysis of the background noise level

In the case of the pressure test of the Scylla airlock, this analysis made it possible to highlight a minor leak which was not detectable from a pressure measurement but nevertheless present. Figure 5 shows the root mean square voltage values for each sensor. The most affected sensor in this case being sensor c2 (channel 2), which revealed a minor leak in the area of the door between Poseidon and Scylla.

Zonal analysis

Figure 6 illustrates a summary of the zonal analysis of Scylla. In particular, we were able to observe that the area covered by sensor c3 was the most emissive (events vs channel graphs, at the right).

Planar localisation-based analysis

Figure 7 illustrates the acoustic activity map for part of the Scylla airlock.

SYNTHESIS FROM THE TESTS ON THE POSEIDON CHAMBER AND THE SCYLLA AIRLOCK

The acoustic emission controls of the two compartments were carried out successfully and resulted in requalification of the complete chamber. Only two half-days of unavailability were necessary to carry out these operations, with many of the preparatory phases being able to be carried out while normal treatments were underway. The acoustic broadcast highlighted:

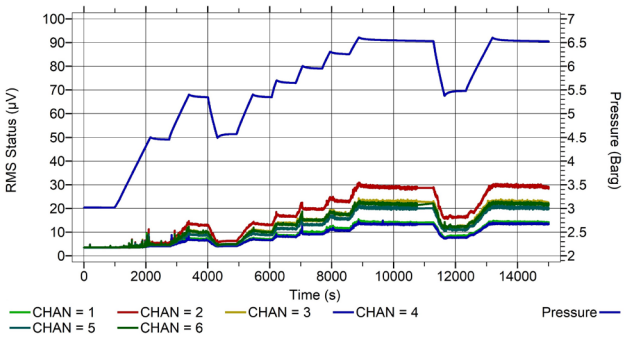
- For Poseidon, four regions classified in category two, corresponding to particular areas (weld portions, viewport flange welding connection portion).
- For Scylla, a region corresponding to part of the Scylla-Poseidon manway junction.
- Additional examinations were carried out on these areas (magnetic particle and ultrasonic inspections) which determined that the vessels were still compliant and safe for continued service.

Discussion

During the acoustic emission test, we detected small anomalies such as the detection of a leak in the area of the door between Poseidon and Scylla. The high sensitivity of the acoustic emission technique to turbulent flow enabled the detection of a small leak that would otherwise be imperceptible. Thanks to a dense network of sensors, it was possible to localise this leak. Moreover, as the data are continuously recorded during the test, acoustic emission can characterise the level of pressure from which the leak has been detected. A simple pressure test without acoustic emission monitoring would have not highlighted such leak, as the pressure loss is not easily measurable, and would not have been able to give any information on its localisation.

Figure 5

Analysis of the Scylla airlock test showing root mean square (RMS) voltage values recorded for channels c1 to c6; CHAN – sensor channel



Acoustic emission testing revealed areas where activity was classified as Category 2, thus detecting regions of potential anomalies. Follow-up inspections using appropriate non-destructive evaluation techniques concluded that these indications were non-significant. Such anomalies would not be detected using a simple hydrostatic test. This illustrates the acoustic emission technique's advantages in being able to detect latent defects, providing precise location, and then allowing focused (or precise) additional investigative work to be done.

The overall finding is in no way negative, resulting in compliant requalification of the chambers. From a regulatory point of view, even if acoustic emission is very sensitive, it allows the requalification of a hyperbaric chamber without imposing any additional constraints compared to a traditional requalification.

Although the active areas have been considered as non-significant during this pressure test, information on their localisation and characteristics (including pressure threshold) will serve as a baseline for comparison at the next pressure tests (or next requalification). Since the acoustic emission technique gives an image of the behaviour of a structure under load, it is able to reveal the appearance of anomalies during the life of the structure.

Hyperbaric chambers are considered 'safety-critical' installations because they can, in the event of an incident, cause major, even catastrophic damage. Given that these facilities accommodate patients and caregivers, it is important to obtain accurate assessment of their fitness for service. Regulations may differ from one country to another but all require vessel integrity to be checked periodically. In Europe, when rated to operate at pressures greater than 3 Barg, this type of equipment is subject to periodic visual inspections (every 48 months) and requalification every

Figure 6

Zonal location analysis of the Scylla airlock test; Barg – pressure in Bar (gauge)

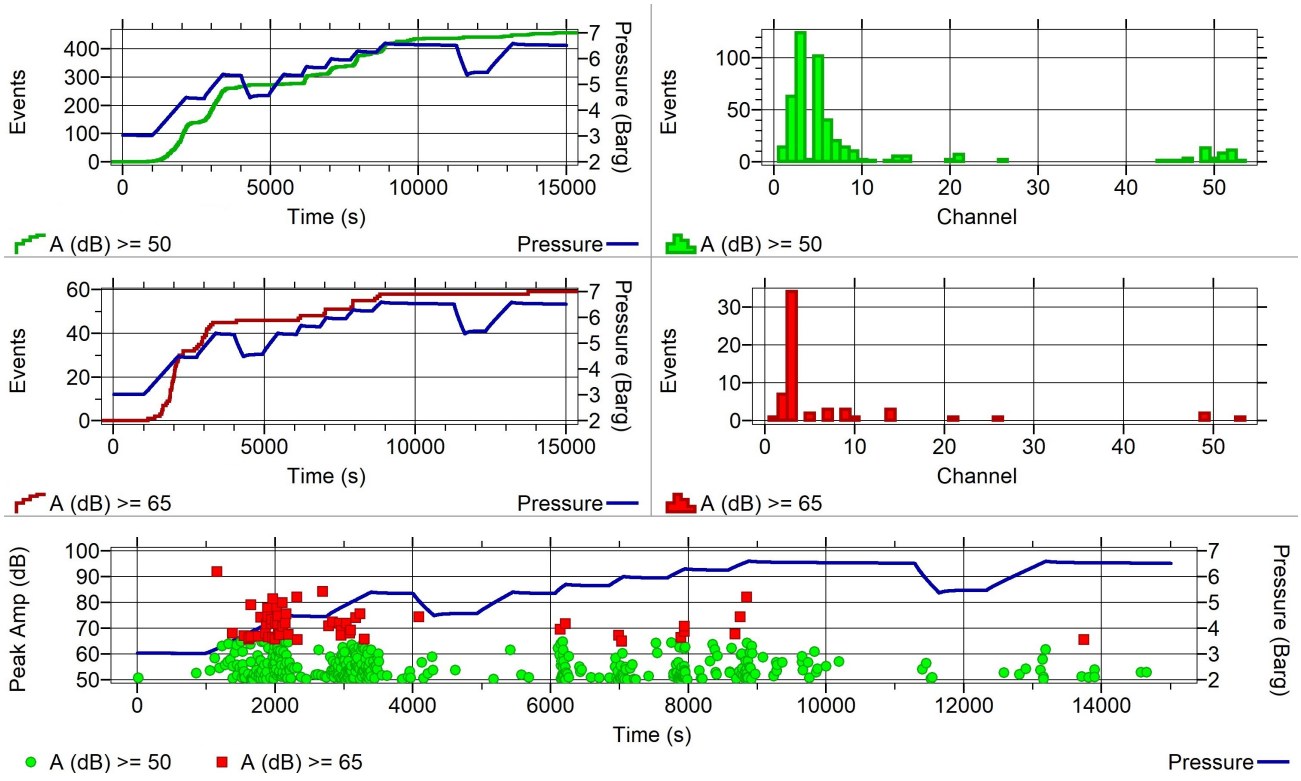
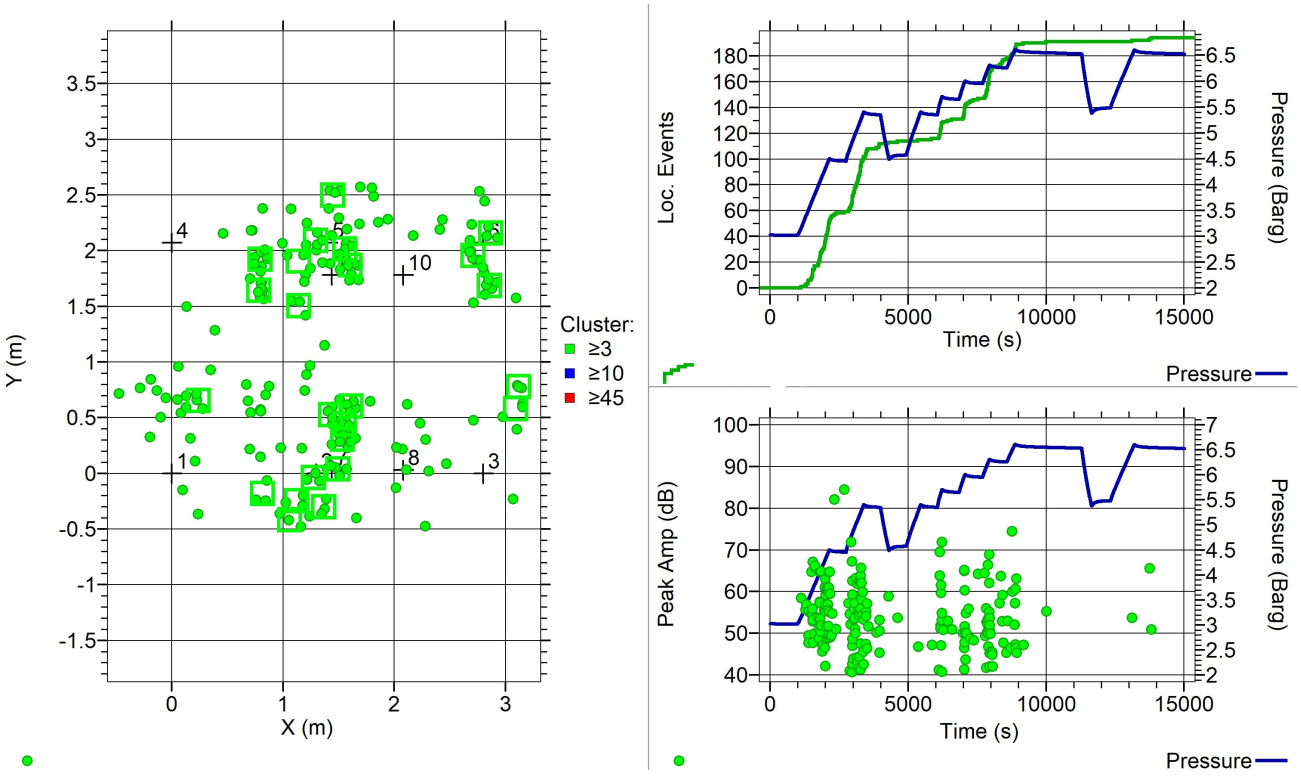


Figure 7

Planar location analysis of the Scylla airlock test; Barg – pressure in Bar (gauge) X & Y – coordinates of the located events



10 years. Traditionally, this requalification is based on a hydraulic test to ≥ 1.2 x maximum allowed pressure, requiring the following operations:

1. Complete dismantling of the chamber;
2. Inspection of the chamber by an authorised body;
3. Hydraulic testing which may involve moving the chamber to support the weight of the water;
4. Drying of the chamber then repainting;
5. Reassembly of all the elements of the chamber; and
6. Carrying out pressure (leak) tests before re-commissioning.

Beyond the fact that many of these operations can be tedious and time-consuming, involving substantial downtime (around three weeks), the hydraulic test does not allow a detailed diagnosis of the state of health of the vessel. Its diagnosis is binary; the equipment either resists or does not resist the pressure, it either leaks or it does not.

Acoustic emission is, today, the most suitable technique for evaluating the serviceability of vessels subjected to stress for several reasons:

1. It is by nature a monitoring technique; and
2. It is similar in many respects to seismology, it makes it possible to detect developments of potentially critical structural defects, and simultaneously, the appearance of even minor leaks.

Since the 1970s, this technique has been used to assess the fitness for service of pressure equipment, whether metallic or composite. The first code appeared in the 1980s in the United States,[14] and later in France and Europe. Today, this technique is recognised statutorily, and the necessary requalification can be based on the result of an acoustic emission test carried out during a pneumatic pressure test.

Several hyperbaric chambers have been able to benefit from the advantages of this technique such as Nice University Hospital and Angers University Hospital. To our knowledge, these chambers (Nice, Angers, Lille) are the only ones that have been requalified in this way. This innovative method has many advantages including:

1. Dismantling operations are no longer necessary;
2. The constraints linked to the hydraulic test are removed;
3. The operations of drying and reassembling accessories are also eliminated;
4. The diagnosis is more precise (mapping the emissivity of the vessel); and
5. Downtime is reduced drastically.

The acoustic emission method makes it possible to obtain precise information on the dynamic behaviour of even complex vessels. It is a conservative method in that the results are sufficiently accurate to detect damage phenomena well before they pose a risk of failure. It allows engineers to have a baseline, ongoing history, and thus detect changes over time (acoustic emission is increasingly used, particularly in the field of condition monitoring). In addition to saving time, it saves floor load calculation work which needs to be carried out by civil engineering teams.

Acoustic emission was also successfully performed on the hyperbaric facility's eight air buffer tanks, each rated at 4,000 litres at 12 Barg, again reducing costs primarily as a result of reduced downtime.

Conclusions

The acoustic emission technique can be the most effective and least expensive tool in obtaining a very precise assessment of the state of health of a vessel under pressure while avoiding long unavailability of the hyperbaric installation. This provides a higher level of confidence in the integrity of all parts of the vessels, together with a baseline for comparison of future tests. We hope that the results of this study will allow other hyperbaric centres to consider this useful and effective alternative to hydraulic tests.

References

1 Arrêté du 20 novembre 2017 relatif au suivi en service des équipements sous pression et des récipients à pression simples. [cited 2024 Jan 7]. Available from: https://www.legifrance.gouv.fr/jorf/id/JORFTEXT000036128632.

2 Directive 97/23/CE du Parlement Européen et du Conseil du 29 mai 1997 relative au rapprochement des législations des États membres concernant les équipements sous pression. [cited 2024 Jan 7]. Available from: https://www.legifrance.gouv.fr/jorf/id/JORFTEXT000000521231.

3 Association française des ingénieurs en appareils à pression. Guide des bonnes pratiques pour le contrôle par émission acoustique des équipements sous pression. 3e éd. Courbevoie: Afiap; 2016. [cited 2024 Jan 7]. Available from: https://www.afiap.org/guides-afiap.

4 Article R557-14-1 - Code de l'environnement - Légifrance. [cited 2024 Jan 7]. Available from: https://www.legifrance.gouv.fr/codes/article_lc/LEGIARTI000033741441.

5 Cony P, Mathieu D, Houman R, Macchi J, Dubois P, Wisson F. Méthodologie d'intégration de dispositifs médicaux en milieu hyperbare. ITBM-RBM. 2005;26(5):363–70. doi: 10.1016/j.rbmret.2005.09.002.

6 Moon RE. Hyperbaric oxygen treatment for air or gas embolism. Undersea Hyperb Med. 2014;41:159–66. PMID: 24851554.

7 Bryson P, Millar I, Burman F. Emergency life support equipment for commercial diving operations: guidance note. 2016. [cited 2024 Jan 7]. Available from: https://www.dmac-diving.org/guidance/JIP-201602.pdf.

8 Section 4: Formation (Articles R4461-27 à R4461-36) - Légifrance. [cited 2024 Jan 7]. Available from: https://www.legifrance.gouv.fr/codes/section_lc/LEGITEXT000006072050/LEGISCTA000023414578/#LEGISCTA000023414578.

9 Blanc P, Boussuges A, Henriette K, Sainty JM, Deleflie M. Iatrogenic cerebral air embolism: importance of an early hyperbaric oxygenation. Intensive Care Med. 2002;28:559–63. doi: 10.1007/s00134-002-1255-0. PMID: 12029402.

10 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.

11 Weaver LK, Hopkins RO, Chan KJ, Churchill S, Elliott CG, Clemmer TP, et al. Hyperbaric oxygen for acute carbon monoxide poisoning. N Engl J Med. 2002;347(14):1057–67. doi: 10.1056/NEJMoa013121. PMID: 12362006.

12 Hedetoft M, Madsen MB, Hyldegaard O. Hyperbaric oxygen treatment in the management of necrotising soft-tissue infections: results from a Danish nationwide registry study. BMJ Open. 2023;13(2):e066117. doi: 10.1136/bmjopen-2022-066117. PMID: 36813488. PMCID: PMC9950903.

13 Mathieu D, Ratzenhofer-Komenda B, Kot J. Hyperbaric oxygen therapy for intensive care patients: position statement by the European Committee for Hyperbaric Medicine. Diving Hyperb Med. 2015;45:42–6. PMID: 25964038. [cited 2024 Jan 7]. Available from: https://dhmjournal.com/images/IndividArticles/45March/Mathieu_dhm.45.1.42-46.pdf.

14 BPVC Section V – Nondestructive Examination – ASME. [cited 2024 Jan 7]. Available from: https://www.asme.org/codes-standards/find-codes-standards/bpvc-v-bpvc-section-v-nondestructive-examination.

15 NF EN 14584. Afnor EDITIONS. [cited 2024 Jan 7]. Available from: https://www.boutique.afnor.org/fr-fr/norme/nf-en-14584/essais-non-destructifs-essais-demission-acoustique-controle-des-equipements/fa169914/41666.

16 Catty J. A new way to evaluate acoustic emission instrumentation: CATS methodology. J Acoust Emiss. 2016;33:1–16. [cited 2024 Jan 7]. Available from: http://www.aewg.org/jae/JAE-Vol_33-2016.pdf.

17 BSI British Standards. Non-destructive testing. Acoustic emission testing. Examination of metallic pressure equipment during proof testing. Planar location of AE sources. London: BSI British Standards; 2013. [cited 2024 Jan 7]. Available from: https://www.document-center.com/standards/show/BS-EN-14584.

18 BSI British Standards. Non-destructive testing. Acoustic emission. Examination of metallic pressure equipment during proof testing. Zone location of AE sources. London: BSI British Standards; 2007. [cited 2024 Jan 7]. Available from: https://www.boutique.afnor.org/en-gb/standard/bs-en-154952007/nondestructive-testing-acoustic-emission-examination-of-metallic-pressure-e/eu101145/203335.

19 NF EN 15495. Afnor EDITIONS. [cited 2024 Jan 7]. Available from: https://www.boutique.afnor.org/en-gb/standard/nf-en-15495/nondestructive-testing-acoustic-emission-examination-of-metallic-pressure-e/fa140736/30960.

Conflicts of interest and funding: nil

Submitted: 10 January 2024
Accepted after revision: 14 July 2024

Evaluation of a new hyperbaric oxygen ventilator during pressure-controlled ventilation

Cong Wang[1], Qiuhong Yu[1], Yaling Liu[1], Ziqi Ren[1], Ying Liu[1], Lianbi Xue[2]

[1] *Department of Hyperbaric Oxygen, Beijing Tiantan Hospital, Capital Medical University, A zone, No.199 Nansihuan West Road, Fengtai District, Beijing 100070, China*
[2] *Department of Hyperbaric Oxygen, Huazhong University of Science and Technology Union Shenzhen Hospital (Nanshan Hospital), No. 89 Taoyuan Road, Nanshan District, Shenzhen 518052, China*

Corresponding author: *Professor Lianbi Xue, Department of Hyperbaric Oxygen, Huazhong University of Science and Technology Union Shenzhen Hospital (Nanshan Hospital), No. 89 Taoyuan Road, Nanshan District, Shenzhen 518052, China*
xue40@vip.sina.com

Keywords
Airway resistance; Intensive care; Intermittent positive-pressure ventilation; Respiratory mechanics

Abstract
(Wang C, Yu Q, Liu Y, Ren Z, Liu Y, Xue L. Evaluation of a new hyperbaric oxygen ventilator during pressure-controlled ventilation. Diving and Hyperbaric Medicine. 2024 30 September;54(3):212–216. doi: 10.28920/dhm54.3.212-216. PMID: 39288926.)
Introduction: The stability of a new hyperbaric ventilator (Shangrila590, Beijing Aeonmed Company, Beijing, China) at different clinically relevant pressures in a hyperbaric chamber during pressure-controlled ventilation (PCV) was investigated.
Methods: The ventilator was connected to a test lung in the multiplace hyperbaric chamber. The inspiratory pressure (PI) of the ventilator was set to 1.0, 1.5, 2.0, 2.5 and 3.0 kPa (approximately 10, 15, 20, 25 and 30 cmH_2O). The compliance and resistance of the test lung were set to 200 $mL \cdot kPa^{-1}$ and 2 $kPa \cdot L^{-1} \cdot s^{-1}$, respectively. Experiments were conducted at 101, 203 and 284 kPa ambient pressure (1.0, 2.0 and 2.8 atmospheres absolute respectively). At each of the 5 PI values, the tidal volume (VT), peak inspiratory pressure (Ppeak) and peak inspiratory flow (Fpeak) displayed by the ventilator and the test lung were recorded for 20 cycles. Test lung data were considered the actual ventilation values. The ventilation data were compared among the three groups to evaluate the stability of the ventilator.
Results: At every PI, the Ppeak detected by the ventilator decreased slightly with increasing ambient pressure. The Fpeak values measured by the test lung decreased substantially as the ambient pressure increased. Nevertheless, the reduction in VT at 284 kPa and PI 30 cmH_2O (compared to performance at 101 kPa) was comparatively small (approximately 60 ml).
Conclusions: In PCV mode this ventilator provided relatively stable VT across clinically relevant PI values to ambient pressures as high as 284 kPa. However, because Fpeak decreases at higher ambient pressure, some user adjustment might be necessary for precise VT maintenance during clinical use at higher PIs and ambient pressures.

Introduction

Hyperbaric oxygen treatment (HBOT) is widely used for patients in the intensive care unit (ICU) for conditions such as acute carbon monoxide poisoning, decompression sickness, and arterial gas embolism.[1–3] Most life support technologies, such as haemofiltration, electrical defibrillation and extracorporeal membrane oxygenation, are incompatible with hyperbaric environments, but ventilators are a necessary exception.[4,5] However, standard ICU ventilators cannot maintain stable output during HBOT, especially when operating with volume-controlled ventilation (VCV). Tests have been carried out on ventilators in hyperbaric environments with basic ventilation modes, such as VCV and pressure-controlled ventilation (PCV).[6–10] Early use of standard ICU ventilators for HBOT required manual adjustment to compensate for predicted changes. According to the equation:[11] $\sqrt{\Delta P} \propto flow \times density$, if gas density doubles, maintaining a stable ventilator driving pressure (ΔP) will result in reducing flow to half; however, obtaining stable flow needs four times ΔP. Thus, PCV is preferentially used with better stability than VCV.[7,11]

In the last few years, hyperbaric ventilators have been developed, replacing the standard ICU ventilators in hyperbaric chambers. These incorporate automatic pressure compensation systems.[11] For example, the Siaretron 1000 IPER ventilator (Bologna, Italy) can maintain a constant tidal volume (VT) with VCV at various ambient pressures by adjusting the inspiratory valve opening within the operational range, but it does not meet the hyperbaric chamber safety requirements of China.[8,12,13] Recently, bench tests of a new hyperbaric ventilator made in China (Shangrila590, Beijing Aeonmed Company, China) were carried out in our hospital. The Shangrila590 ventilator was evaluated in VCV mode in a 101–284 kPa (1.0–2.8 atmospheres absolute [atm abs]) environment.[14] In the present study, the peak inspiratory pressure (Ppeak) and VT stability of the Shangrila590 were measured during PCV in a hyperbaric chamber.

Methods

VENTILATOR

The Shangrila590 ventilator is an electropneumatic ventilator manufactured by the Beijing Aeonmed Company in China. According to the safety regulations of medical hyperbaric chambers in China, the pneumatic part of the ventilator is situated within the chamber, and the electronic component is outside the chamber.[12,13] These two components were connected through a chamber wall penetrator. In the experiment, doctors operated the ventilator outside the hyperbaric chamber.

TEST LUNG

A Michigan Instruments PneuView® 3 System (Grand Rapids, MI, USA) was used to measure the ventilation parameters. The detection system included a test lung and PneuView® 3.3 software. The test lung data were collected and recorded by a computer with PneuView® 3.3 software.

CRITICAL CARE MULTIPLACE HYPERBARIC CHAMBER

The critical care hyperbaric chamber (GY3800-A [GY3800 M2-D], Yantai Hongyuan Oxygen Industrial Inc., Yantai, China) is a multiplace hyperbaric chamber that has the capacity for 24 seated people or eight gurneys. In addition to ventilators, electrocardiogram monitors, transcutaneous oxygen (O_2) and carbon dioxide (CO_2) tension monitors, syringe drivers, and infusion pumps were equipped to ensure the continuous treatment of ICU patients.

EXPERIMENTAL CONFIGURATION

The ventilator and the test lung were calibrated at atmospheric pressure before the experiments. The test lung was located inside the hyperbaric chamber and connected to the pneumatic part of the ventilator. The ventilation data were detected by the test lung and recorded by a computer. Moreover, the ventilation data were detected by the ventilator and displayed on the screen of the ventilator component. According to the parameters shown in the 'Calibration Specification for Ventilators in China', the parameters of the ventilator and test lung were set by the investigators.[15,16]

EXPERIMENTAL PROCEDURE

Tests were undertaken at three hyperbaric chamber pressures: 101, 203 and 284 kPa (1.0, 2.0 and 2.8 kPa (1.0, 2.0 and 2.8 atm abs). Under each ambient pressure, the ventilator was operated in PCV mode at five preset inspiratory pressure (PI) values being 1.0, 1.5, 2.0, 2.5 and 3.0 kPa (approximately 10, 15, 20, 25 and 30 cmH_2O). Since ventilation pressures in the clinical setting are commonly expressed in cmH_2O we use that metric in reporting VT results. Other PCV parameters were the respiratory rate (f) at 15 breaths per minute (BPM), inspiratory/expiratory ratio (I/E) at 1:2, positive end-expiratory pressure (PEEP) at 0 kPa, and fraction of inspired oxygen (FiO_2) 40%.[15,16] The resistance and compliance of the test lung were set at 200 $mL \cdot kPa^{-1}$ and 2 $kPa \cdot L^{-1} \cdot s^{-1}$, respectively.[15,16] The ventilator was considered at steady state two minutes after setting changes. The Ppeak and VT data measured by the ventilator and test lung were collected for 20 cycles in each setting ($n = 20$). Outcomes for these measures are expressed as the means and standard deviations of those 20 readings. Peak inspiratory flow (Fpeak) could only be detected by the test lung in each setting. The temperature in the hyperbaric chamber was maintained between 24°C and 26°C.

Results

EFFECTS OF DIFFERENT AMBIENT PRESSURES ON Ppeak AND Fpeak DURING PCV

When the ambient pressure increased, the Ppeak detected by the ventilator decreased slightly at every PI setting (Table 1). Compared with the Ppeak at 1.0 atm abs, the decrease in this value was less than 5% at 2.0 atm abs, and it was 8–10% at 2.8 atm abs. Inspiratory flow provided by

Table 1
Value of peak inspiratory pressure (Ppeak) during pressure-controlled ventilation (PCV) at different ambient pressures; data are mean (standard deviation)

Inspiratory pressure kPa (cmH_2O)	Peak pressure detected by the ventilator (kPa)		
	101 kPa (1 atm abs)	203 kPa (2 atm abs)	284 kPa (2.8 atm abs)
1.0 (10)	1.15 (0.03)	1.20 (0.05)	1.10 (0.03)
1.5 (15)	1.72 (0.03)	1.72 (0.03)	1.62 (0.04)
2.0 (20)	2.39 (0.09)	2.37 (0.07)	2.18 (0.10)
2.5 (25)	2.95 (0.06)	2.87 (0.07)	2.75 (0.45)
3.0 (30)	3.43 (0.06)	3.32 (0.08)	3.18 (0.05)

Figure 1
Mean peak inspiratory flow (Fpeak) measured by the test lung during pressure-controlled ventilation at different ambient pressures and at five levels of inspiratory pressure (PI); error bars are standard deviation; atm abs – atmospheres absolute

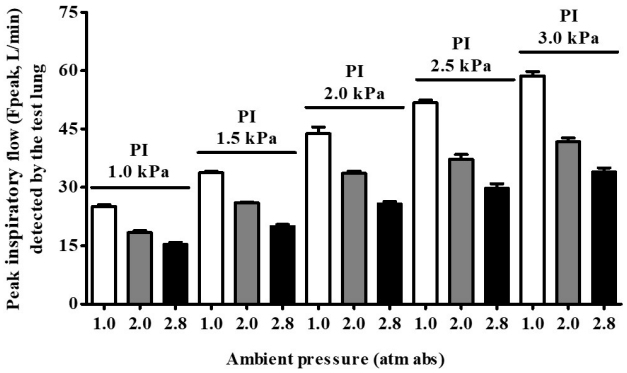
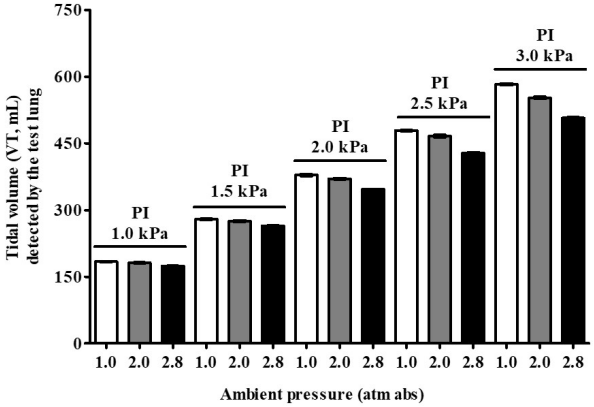


Figure 2
Mean tidal volume (VT) measured by the test lung during pressure-controlled ventilation at different ambient pressures and at five levels of inspiratory pressure (PI); error bars are standard deviation; atm abs – atmospheres absolute



the ventilator during PCV was evaluated by Fpeak, which was detected only by the test lung. Figure 1 shows that with a fixed respiratory system, Fpeak decreased significantly as the ambient pressure increased at every PI value. The Fpeak decreased by 26% at 2.0 atm abs and 41% at 2.8 atm abs compared with that at 1.0 atm abs.

EFFECTS OF DIFFERENT AMBIENT PRESSURES ON VT DURING PCV

When the ambient pressure increased, the VT detected by the test lung decreased slightly at every PI setting (Figure 2). Compared with the VT at 1.0 atm abs, at a low PI (1.0–2.0 kPa), the VT decrease was less than 2% at 2.0 atm abs and 8% at 2.8 atm abs; at a high PI (2.5–3.0 kPa), the VT decrease was 3–5% at 2.0 atm abs and 10–13% at 2.8 atm abs.

Discussion

Essentially, a ventilator is a generator of gas flow in a mechanical ventilation system.[11] Various ventilation modes have emerged which determine how a ventilator controls the gas flow to produce effective pulmonary ventilation. Regardless of the complexity of ventilation modes, they are different combinations of basic models, such as PCV, VCV and spontaneous (SPONT) ventilation.[7–9] In terms of flow generation and control, ventilation during HBOT introduces added complexity and has been investigated by many researchers.

Gas flow occurs between the supply pressure of the ventilator (Pv) and the internal pressure inside the respiratory system of the patient (Pp) which is approximately equal to the environmental pressure (Pe). The driving pressure (ΔP) of flow generation is defined as the difference between Pv and Pp (or Pe), and ΔP is the direct power of flow generation.[11,17] During HBOT, Pe is no longer a constant but rather a variable. Pe increases and causes a decrease in ΔP, leading to the unstable output of standard ICU ventilators.[7,8,11] The ΔP change induced by the ambient pressure is one factor that determines whether gas flow can be generated; the other factor is the flow type. Compared with laminar flow, achieving the same flow under turbulent conditions needs more ΔP; moreover, during HBOT, in the same respiratory system, the prevalence of turbulent flow increases because of the high gas density.[17] Theoretically, with increasing ambient pressure, obtaining stable pressure requires reducing gas flow; in contrast, obtaining stable gas flow requires increasing pressure. The improved algorithm of the hyperbaric ventilator automatically controls gas flow to compensate for these changes caused by increased ambient pressure while maintaining stable output.[6–11]

Pressure controlled ventilation aims to provide constant airway pressure during inhalation. Table 1 shows a roughly stable Ppeak with increasing ambient pressure. The Shangrila590 can maintain a stable Ppeak by decreasing Fpeak with increasing ambient pressure (Figure 1). Others have shown that the time to reach the preset PI decreases because of high airway resistance at high ambient pressure, although Ppeak can reach the preset PI value.[7]

Physiologically, VT may vary between breaths with PCV because of uncertain respiratory resistance, but it is independent of ambient pressure. However, if the respiratory system is stable and ambient pressure is fixed, VT will remain stable. With increasing ambient pressure, the airway resistance increases, and Fpeak decreases to obtain a stable Ppeak (Figure 1). If the inspiratory time (Ti) is sufficient, VT is stable; if Ti is insufficient, VT obviously decreases.[7] In our study, although Fpeak decreases, at a lower PIs (1.0–2.5 kPa [10–25 cmH_2O]), VT is roughly stable; at a high PI (2.5–3.0 kPa [25–30 cmH_2O]), VT decreases by 10–13% at 284 kPa (2.8 atm abs) (Figure 2). In practice, on the one hand, if the respiratory rate increases or Ti decreases,

a decrease in Fpeak induces a decrease in VT even in patients with stable conditions. On the other hand, if the increase in airway resistance is further induced by disease progression, the decrease in Fpeak is amplified, and VT eventually decreases. Therefore, during use of a ventilator in hyperbaric conditions we must carefully monitor VT, carry out percutaneous O_2 and CO_2 monitoring and properly regulate Ti to avoid incomplete inhalation during use of PCV mode.

LIMITATIONS

First, the test lung accuracy was not calibrated at high ambient pressures. In the clinic and in HBOT, a ventilator operates continuously as the setting parameters are regulated. The test lung and the ventilator were all calibrated at normal atmospheric pressures before the experiments. In the pre-experiment phase, a water tank was used as a simulated lung to determine the accuracy of the ventilator spirometer at high ambient pressures. The water tank could detect the TV of the ventilator at different ambient pressures according to the change in the water level roughly and continuously. The VT values of the ventilator and the water tank data were similar. However, this water tank could not be used in the experiments because of its low precision (50 mL).

Second, humidification was not used. To avoid the accumulation of a large amount of condensed water in the test lung, humidification was not used in the experimental system. In China, according to National Standards, humidifiers with high voltages (> 24 V) could not be used in hyperbaric chambers. Additionally, it is indeed a fact that humidification will add airway resistance.[19,20]

Conclusions

In summary, with increasing ambient pressure from 101–284 kPa (1.0–2.8 atm abs), the Shangrila590 ventilator provided an approximately stable Ppeak value during PCV with an PI ranging from 1.0 to 3.0 kPa (10–30 cmH_2O). However, a stable Ppeak was accompanied by a decrease in Fpeak as the ambient pressure increased, and incomplete inhalation could occur if inspiratory time is inadequate.

References

1 Tibbles PM, Edelsberg JS. Hyperbaric-oxygen therapy. N Engl J Med. 1996;334:1642–8. doi: 10.1056/NEJM199606203342506. PMID: 8628361.

2 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. Erratum in: Diving Hyperb Med. 2017;47:131–2. PMID: 28357821. PMCID: PMC6147240.

3 Weaver LK. Hyperbaric oxygen in the critically ill. Crit Care Med. 2011;39:1784–91. doi: 10.1097/CCM.0b013e31821858d1. PMID: 21460713.

4 Millar IL. Hyperbaric intensive care technology and equipment. Diving Hyperb Med. 2015;45:50–6. PMID: 25964040. [cited 2024 Mar 10]. Available from: https://dhmjournal.com/images/IndividArticles/45March/Millar_dhm.45.1.50-56.pdf.

5 Kot J. Medical devices and procedures in the hyperbaric chamber. Diving Hyperb Med. 2014;44:223–7. PMID: 25596835. [cited 2024 Mar 10]. Available from: https://dhmjournal.com/images/IndividArticles/44Dec/Kot_dhm.44.4.223-227.pdf.

6 Lewis RP, Szafranski J, Bradford RH, Smith HS, Crabbe GG. The use of the Penlon Nuffield 200 in a monoplace hyperbaric oxygen chamber. An evaluation of its use and a clinical report in two patients requiring ventilation for carbon monoxide poisoning. Anaesthesia. 1991;46:767–70. doi: 10.1111/j.1365-2044.1991.tb09775.x. PMID: 1928680.

7 Stahl W, Radermacher P, Calzia E. Functioning of ICU ventilators under hyperbaric conditions – comparison of volume- and pressure-controlled modes. Intensive Care Med. 2000;26:442–8. doi: 10.1007/s001340051179. PMID: 10872137.

8 Lefebvre JC, Lyazidi A, Parceiro M, Sferrazza Papa GF, Akoumianaki E, Pugin D, et al. Bench testing of a new hyperbaric chamber ventilator at different atmospheric pressures. Intensive Care Med. 2012;38:1400–4. doi: 10.1007/s00134- 012-2590-4. PMID: 22588650.

9 Stanga DF, Beck G, Chimiak JM. Evaluation of respiratory support devices for use in the hyperbaric chamber. Research Report NEDU TR 03-18. Panama City (FL): Navy Experimental Diving Unit; 2003. [cited 2022 Nov 1]. Available from: https://www.semanticscholar.org/paper/Evaluationof-Respiratory-Support-Devices-for-Use-Stanga-Beck/ a74afe5416b366ffb65726fbce4117f5ca9cd1de.

10 An LS, Ting LS, Chuan LC, Joang KS, Rick SC. Performance of the Oxylog® 1000 portable ventilator in a hyperbaric environment. Diving Hyperb Med. 2018;48:102–6. doi: 10.28920/dhm48.2.102-106. PMID: 29888382. PMCID: PMC6156829.

11 Kot J. Medical equipment for multiplace hyperbaric chambers. Part II: ventilators. Europ J Underwater Hyperbaric Med. 2006;7:9–12.

12 State administration for market regulation (PRC). Standardization administration (PRC). Hyperbaric oxygen chambers (GB/T 12130-2020). The People's Republic of China; 2020 Sep 9. [cited 2024 Mar 15]. Available from: https://openstd.samr.gov.cn/bzgk/gb/newGbInfo?hcno=5981FE6F33F0E847F5F90D6A788B171F.

13 General administration of quality supervision, inspection and quarantine of the People's Republic of China. Supervision regulation on safety technology for hyperbaric oxygen chambers (TSG 24-2015), PRC, 2015 Nov 20. [cited 2022 Nov 1]. Available from: https://www.samr.gov.cn/cms_files/filemanager/samr/www/samrnew/tzsbj/zcfg/aqjsgf/aqjsgf/201906/P020190621529724704158.pdf.

14 Wang C, Xue L, Yu Q, Liu Y, Ren Z, Liu Y. Evaluation of a new hyperbaric oxygen ventilator during volume-controlled ventilation. Diving Hyperb Med. 2023;53:129–37. doi: 10.28920/dhm53.2.129-137. PMID: 37365130. PMCID: PMC10584397.

15 General administration of quality supervision, inspection and quarantine of the People's Republic of China. Calibration specification for ventilators (JJF 1234-2018). PRC, 2018 Feb 27. [cited 2024 Mar 15]. Available from: http://jjg.spc.org.cn/resmea/standard/JJF%25201234-2018/?.

16 National health commission of the People's Republic of China. Safety management for lung ventilator (WS/T 655-2019). PRC, 2019 Oct 19. [cited 2024 Mar 15]. Available from: http://www.nhc.gov.cn/wjw/s9495/202003/b58664f1dcaf4f349caed80820800c08/files/af323f6cdf0e422d83d2ada4359e7359.pdf.
17 Welslau W. Physiologic effects of increased barometric pressure. In: Mathieu D, editor. Handbook on hyperbaric medicine. Dordrecht, The Netherlands: Springer-Verlag; 2006. p. 41–3.
18 Weaver LK, Greenway L, Elliot CG. Performance of the Seachrist 500A hyperbaric ventilator in a monoplace hyperbaric chamber. J Hyperbaric Med. 1988;3(4):215–25. [cited 2023 Aug 1]. Available from: https://www.uhms.org/images/Equipment-Articles/sechrist_vent_weaver.pdf.
19 Doolette DJ, Mitchell SJ. Hyperbaric conditions. Compr Physiol. 2011;1:163–201. doi: 10.1002/cphy.c091004. PMID: 23737169.
20 Arieli R, Daskalovic Y, Ertracht O, Arieli Y, Adir Y, Abramovich A, et al. Flow resistance, work of breathing of humidifiers, and endotracheal tubes in the hyperbaric chamber. Am J Emerg Med. 2011;29:725–30. doi: 10.1016/j.ajem.2010.02.003. PMID: 20825878.

Conflicts of interest and funding: nil

Submitted: 15 March 2024
Accepted after revision: 18 May 2024

The investigation of diving accidents and fatalities

John Lippmann[1,2,3], James Caruso[4]

[1] *Australasian Diving Safety Foundation, Canterbury, Victoria, Australia*
[2] *Department of Public Health and Preventive Medicine, Monash University, Victoria, Australia*
[3] *The Royal Lifesaving Society – Australia, Sydney, Australia*
[4] *Denver Office of the Medical Examiner, Denver, Colorado, United States of America*

Corresponding author: *Dr John Lippmann, Australasian Diving Safety Foundation, PO Box 478, Canterbury VIC 3126, Australia*
johnl@adsf.org.au

Keywords
Arterial gas embolism; Autopsy; Coroner; Decompression sickness; Diving deaths; Diving incidents; Investigations; Scuba

Abstract

(Lippmann J, Caruso J. The investigation of diving accidents and fatalities. Diving and Hyperbaric Medicine. 2024 30 September;54(3):217–224. doi: 10.28920/dhm54.3.217-224. PMID: 39288927.)
Diving accidents result from a variety of causes including human error, inadequate health and fitness, environmental hazards and equipment problems. They usually involve a cascade of events resulting in the diver being injured or deceased. The accuracy and usefulness of a diving accident investigation relies on well-targeted interviews, good field investigation, evidence collection and preservation, and appropriate equipment assessment. In the event of a fatality, a thorough and targeted autopsy is indicated. Investigators should have the appropriate knowledge, training, skills and support systems to perform the required tasks. Relevant investigations include the victim's medical and diving history, the dive circumstances and likely accident scenario, management of the accident including rescue and first aid, equipment inspection and testing and a thorough postmortem examination conducted by a forensic pathologist with an awareness of the special requirements of a diving autopsy and the knowledge to correctly interpret the findings. A chain of events analysis can determine the likely accident scenario, identify shortcomings and inform countermeasures.

Introduction

Diving is a popular recreation for many millions of people throughout the world who dive for recreation, exploration, seafood harvesting, photography and other reasons. The underwater world is also a workplace for a variety of occupational divers. No matter what the goal, diving is conducted in a potentially hostile environment and so involves various risks, although many of these may be mitigated in a healthy, experienced diver with an adequate and appropriate breathing gas supply and suitable, familiar, functional equipment.

Common contributors to a diving incident include human error, natural disease, underwater accidents, equipment problems or a combination of these. Safety can be influenced by a range of factors present before, during, and sometimes after the dive. Such factors may include health and fitness, organisation and planning, communication and supervision, equipment problems, decision making and various environmental factors.

In the earlier decades of recreational scuba diving, most participants were relatively young, training was less available, equipment generally less suitable and reliable and a higher proportion of deaths were due to primary drowning.[1] The age of recreational divers has increased[2,3] and with this there is a higher prevalence of co-existing disease.[4] Unsurprisingly, more recent fatality reports include an increased representation of older divers[5,6] with at least one quarter of the deaths being cardiac-related.[7,8]

Investigation of diving accidents

When an accident occurs, an investigation will commence and its effectiveness will be enhanced by good field investigation, including targeted witness interviews, evidence collection and preservation, appropriate equipment assessment, and, in the event of a fatality, a thorough and targeted autopsy.

It is advantageous to have a system in place to quickly activate various emergency services and expertise, such as diving medical advisors, diver evacuation specialists and dive search and rescue squads. This may be easier to establish in a locality where diving accidents are more common. With a fatality, such a system can ensure that the victim's body and equipment is secured with minimal inappropriate handling, witness interviews are timely, targeted and thorough, and that a postmortem computed

tomography (CT) scan is performed with minimal delay. In a non-fatal accident, the system should facilitate more efficient and appropriate onsite management and transfer of the patient to suitable medical care.

DEPTH AND FOCUS

The depth and focus of an investigation may vary greatly depending on a variety of factors, including:

- The outcome of the accident – fatal or non-fatal;
- The nature of the accident – recreational or occupational;
- The location of the accident – in some countries or jurisdictions, dive accidents are investigated relatively thoroughly. In others, such as in many developing countries, only relatively scant investigations are usually done, if any.
- The interest and experience of the investigators – in some places, investigations are conducted by experienced police divers, dive equipment technicians and diving-savvy forensic pathologists/medical examiners. In others, it is only a rudimentary overview by local police with no understanding of diving or diving pathology.
- The reason for the investigations – in the event of a suspected homicide, police will take a special interest. If the accident occurred in an occupational setting, a workplace authority will often be involved. If the diver or dive company were insured, the insurer may be involved. In some cases, a private investigator may be commissioned by the family or insurer.

INVESTIGATION STAGES

The investigation itself will include a variety of stages. The inclusion and thoroughness of some of these may depend on the severity of the accident, the purpose of the investigation and the skill of the investigators. For example, if the desire is to allocate or negate culpability to a person or operation, there will likely be a greater focus on duty of care and responsibility of others involved, rather than simply an explanation of what occurred. If the aim is to understand and improve shortcomings in an operation, there will be a greater focus on organisational processes and procedures. The stages of the investigation may include:

Interviewing witnesses

Witnesses may include dive buddies, dive supervisor, dive operator, vessel crew, rescuers, first aiders and bystanders. Information may be sought about the roles and responsibilities, qualifications and experience of some of these parties and their relationship to the victim, as appropriate.

It is important to seek relevant information about the diver's training, certification and experience; health, fitness, state of mind and preparedness before the accident; as well as pertinent events leading up to the dive. Information should also be sought about the known or likely accident scenario, as well as details of the rescue or recovery and any first aid or resuscitation provided.

Rescuers or recovery divers should be questioned about where the diver was found, their posture, any entanglements, what equipment was in place or obviously missing, what equipment was inflated, deflated, or ditched by the rescuer during the process and the timings and complications. If there has been a considerable submersion time and survival is impossible, it is useful for the recovery divers to photograph or video the victim's body as found for later consideration. Information sought from first aiders should include the time frames involved, any equipment used, complications and outcomes. Such information can help to assess any shortcomings in preparation or procedures which need to be addressed and to inform improvement measures. The time from unconsciousness to rescue, commencement of resuscitation, attachment of a defibrillator and the potential oxygen concentration delivered is often not recorded, so the opportunity to assess the effectiveness of these measures is often lost.[9]

If the accident was non-fatal, the diver themselves may provide valuable information and insights into the sequence of events. In fatal accidents, witnesses may be able to provide information about the accident depth, what the victim was last seen doing, any apparent signs of distress and any obvious causes of the event. Gaining a thorough history of the victim and the accident will provide a solid basis for the entire investigation. However, many accidents are unwitnessed, especially fatalities, and the likely or possible scenario will need to be constructed based on any available information and varying degrees of speculation.

Site inspection

Site inspection should include a physical description of the site including the topography, hazards and other features, the forecast and prevailing weather and sea conditions (e.g., swell, chop, surge, current, visibility, water temperature, tide) at the time of the accident and their possible influence.

Equipment collection, inspection and testing

The victim's equipment should be collected, photographed and secured for inspection and testing by an appropriately qualified, independent technician. The 'Chain of Custody' of all equipment and other evidence must be fully and carefully documented to facilitate preservation of evidence and minimise the chance of loss or manipulation.

At the site, the contents of the breathing gas cylinder(s) should be recorded, and the pressure gauge photographed before the cylinder valve is turned off, with the number of turns, or part thereof, required recorded. The lines should not be purged so any leaks might be evident. The level of inflation of the buoyancy compensator device (BCD) should

also be noted. All equipment needs to be clearly listed and a note made of any that is missing so that it is clear what the diver was wearing and what might have been ditched or become detached. The equipment then needs to be securely stored for thorough inspection and testing, which should be done as soon as possible as some components may deteriorate over time.

Inspection includes checking that the equipment had been assembled correctly, and carefully checking all components for faults, damage and wear and tear. The type, thickness and coverage of exposure suit should be recorded. Testing of regulators and demand valves involves assessing their function and breathing performance and comparing these to the manufacturers' specifications. Buoyancy control devices and drysuits should be checked for leaks and correct inflation and deflation performance. Pressure gauges and depth gauges should be checked for accuracy and dive data downloaded from the dive computer(s) for later analysis. Available weights should be recorded and whether it is likely that some are missing. If the technician is unfamiliar with a piece of the equipment (e.g., with a certain model of rebreather), they should enlist the help of an independent expert on that equipment.

If there is breathing gas remaining in the cylinder, it should be sent to an appropriate laboratory for testing and comparison to the relevant air purity standard. This will reveal the composition including the oxygen percentage and the presence of any contaminants.

Where practical, it can sometimes be informative to take the equipment on a re-enactment dive to check its in-water functioning under similar circumstances to those of the accident. This will sometimes flush out problems that had not been obvious during bench testing or confirm suspicions of problems.

Dive computer data download

The victim's dive computer can act as a 'silent witness' and may provide valuable information about the diver's profile and the nature and timings of certain events. It may show changes in depth, ascents and descents and the associated rates, decompression violations, cessation of movement. However, the profile needs to be interpreted carefully and knowledgably.

Although the depth and time elapsed measurements of various computers are generally accurate, this is not always the case so these should be checked, and deviations noted. Dates and times shown on the profiles depend on whether the computer was correctly set prior to the dive. Decompression data will be reliant on the computer decompression model and any prior personal adjustments made by the user. On some computers, ascent rate alarms can be particularly sensitive, and a violation recorded with a very small and insignificant depth change. Water temperature and air consumption estimations vary between computers, some being more accurate than others.[10]

Computers vary in how they display the depths during the sampling intervals on the downloaded profile. Some models display the average depth during the interval, others the maximum depth and both can introduce inaccuracies, especially with longer sampling intervals. The shorter this interval, the closer the profile will match reality. For example, if the profile displays the maximum depth reached during each sampling interval and, if the interval is wide, significant changes in depth between the surface and the maximum depth will not be shown. This can mean, for example, that an ascent to or near the surface within an interval may be missed. On occasions, such as a with a profile shown with a single descent and a single recovery ascent of a deceased diver, this can confuse the interpretation of the relationship between the downloaded profile and the presence of arterial gas embolism.

Medical history

Family members may provide some insights into the victim's medical history, but consultation with the victim's primary care physician and/or diving medical examiner may yield important information about their health, medications, medical investigations and treatment. If the deceased diver was diving with a buddy or in a group, other divers may be aware of any pre-dive health complaints and possible use of over-the counter medications for seasickness or acute respiratory illness. There also may be a history of alcohol or drug use the evening before the dive. This information should be shared with the forensic pathologist as it may supplement autopsy findings and help to understand the accident scenario.

Diving history

The victim's diving history, including qualifications, period of diving, range of diving experience and the number and nature of dives completed can provide important insights. The diver's logbook or dive computer downloads assist this, including whether there had been a significant period of inactivity prior to the accident. A regular buddy or dive operator may also be able to provide information. The dive computer may contain evidence of diving behaviour such as habitual rapid ascents or the use or non-use of safety stops.

The postmortem examination

A complete forensic autopsy should be performed in all diving-related deaths, preferably by a forensic pathologist who possesses a knowledge of diving practices, diving physiology associated with breathing compressed gas underwater, and the correct interpretation of the autopsy findings in these challenging cases. The pathologist should

be aware of some modifications in autopsy technique that can be employed to determine if a gas embolism may have occurred. If the pathologist is not familiar with diving procedures and pathophysiology, it is highly recommended that consultation be obtained, either from another pathologist or from a diving physician. There are some excellent resources which explain the requirements and techniques for a diving autopsy.[11–15]

As is the case for all medicolegal autopsies, the primary goal is to establish a cause of death. However, in diving-related deaths it is even more important to establish the sequence of events leading up to the fatal outcome. For the proper interpretation of the autopsy findings the pathologist performing the postmortem examination needs to be made aware of the dive profile, the results of the equipment evaluation, the deceased diver's medical history, and the circumstances surrounding the dive.

Depending on jurisdiction, the coroner, pathologist, or medicolegal death investigator may go to the scene. This is preferable, as it provides context for the autopsy and it allows for interaction between the coroner/medical examiner staff and law enforcement, coast guard personnel, and witnesses which may include other divers. If the diver had been transported to hospital, the investigation may include visits to both the primary and secondary scenes.

The postmortem interval, the time between death and the autopsy, should be minimised to the extent possible to limit artifacts. In some diving-related deaths, the body is not recovered for days or even weeks after the dive. Decomposition changes and postmortem animal predation present a challenge to the pathologist and the information provided by the postmortem examination may be limited.

Postmortem radiographic imaging should be obtained prior to the autopsy. If available, postmortem computer tomography (PMCT) is preferred (Figure 1). Full body PMCT is desirable, but imaging of at least the head and thorax should take place, at a minimum. The scan can indicate the possible signs of arterial gas embolism (AGE), as well as bullae, blebs, parenchymal haemorrhage, signs of drowning, immersion pulmonary oedema, trauma, cardiac disease, and other natural disease processes.[16] Imaging should take place as soon as possible, even if the autopsy cannot be immediately performed. The longer the delay, the more likely the assessment of AGE will be complicated by gases from putrefaction and postmortem decompression artefact.

After postmortem imaging is completed, the autopsy should begin with a meticulous external examination. The body may be accompanied by the dive gear, and thermal protection, such as a wet suit or dry suit, may still be in place. All of this should be documented as should any evidence of trauma, including marine animal bites and stings. Findings such as subcutaneous emphysema, pedal oedema, or frothy fluid emanating from the nose and mouth are important to note. An examination of the tympanic membranes with an otoscope may reveal evidence of barotrauma. The goal of the external examination in forensic autopsies is to document how the body and environment interacted.

The internal examination should include not only documentation of any diving-related pathology such as intravascular and intracardiac gas, but also the presence of natural disease processes such as coronary artery atherosclerosis, cardiomegaly, and left ventricular hypertrophy. Potential intracardiac shunts, such as a patent foramen ovale or a ventricular septal defect, should be noted if present. Evidence of pulmonary issues that can result is air trapping, such as bullous emphysema or asthma, are also important to note.

A few modifications in autopsy technique are recommended, particularly if high-quality postmortem imaging is unavailable. One method for checking if a pneumothorax is present involves dissecting through the intercostal muscles on each side of the chest wall to see if the visceral and parietal pleurae are still adjacent (Figure 2). If the visceral pleura does not fall away from the parietal pleura as the chest cavity is breached, a pneumothorax is present.

A recommended technique for demonstrating the presence or absence of intracardiac gas involves opening the pericardial sac and filling it with water. The right and left ventricles of the heart can then be sequentially incised to determine if intracardiac gas is present (Figures 3, 4). Prior to opening the cranial cavity and removing the brain, tying off the carotid arteries may minimise the introduction of gas into the

Figure 1

Post-mortem computed tomography scan of a diver who died due to a gas embolism. Note the large amount of intravascular and intracardiac gas. (Courtesy of Michael Pickup, M.D., Provincial Forensic Pathology Unit, Toronto, Ontario)

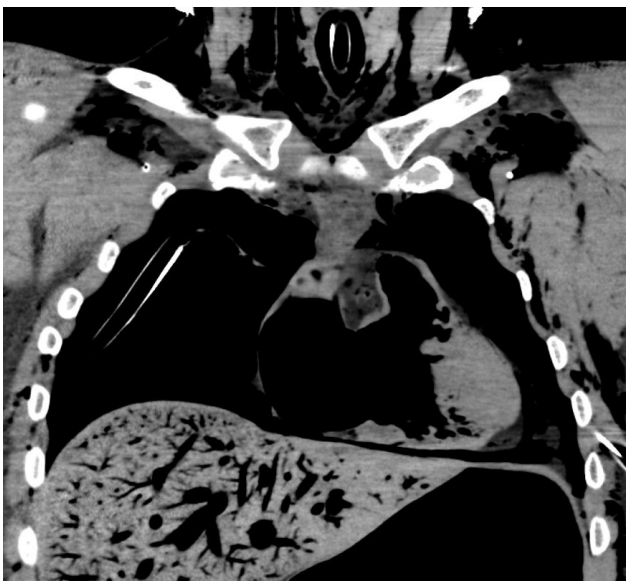


Figure 2
The intercostal muscles can be dissected in layers to expose the lungs as a test for the presence of a pneumothorax

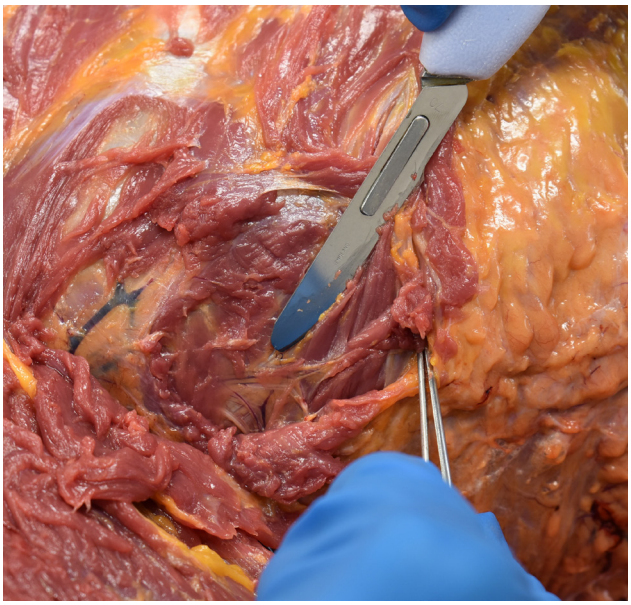


Figure 3
Filling the pericardial sac with water is one method that can be used to check for intracardiac gas in air embolism

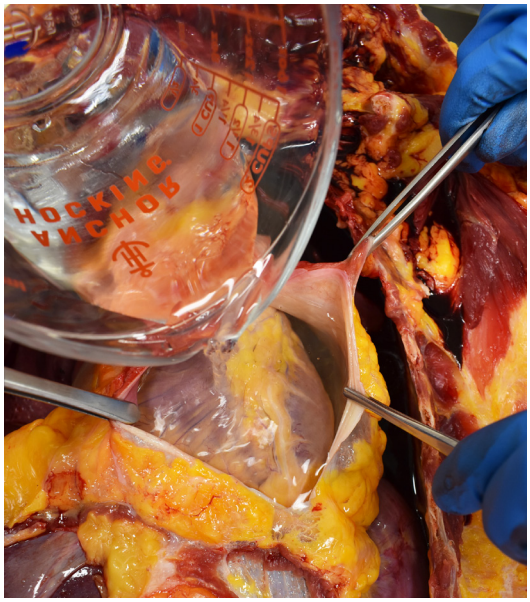


Figure 4
After the pericardial sac has been filled with water the right and left ventricles can be incised to see if any gas escapes

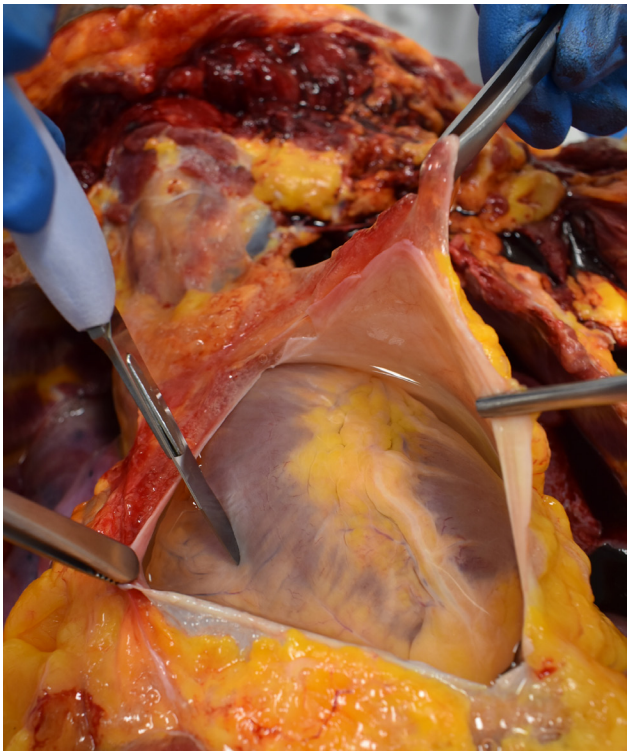
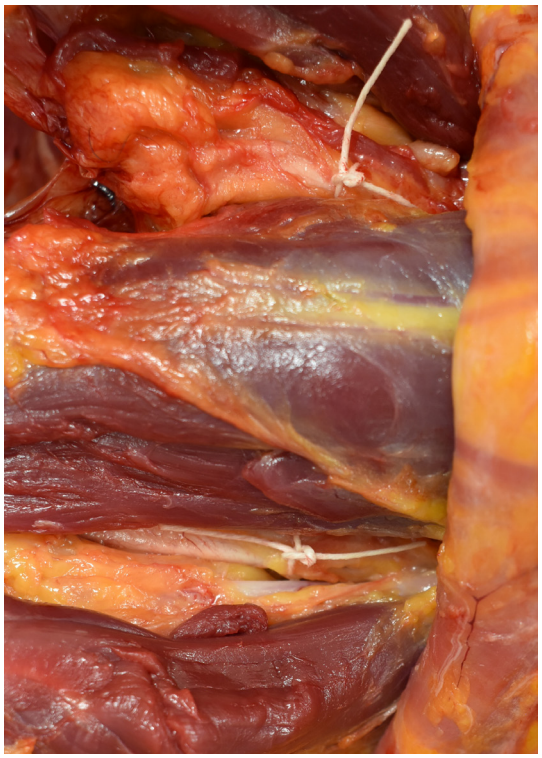


Figure 5
Tying off the carotid arteries prior to opening the head and checking cerebral arteries for intravascular gas can minimise artifact



cerebral vasculature (Figure 5). More extensive techniques to minimise postmortem artifact have been suggested.[17]

Ancillary studies should include obtaining comprehensive postmortem toxicology looking for ethanol, typical drugs of abuse, and select prescription and over-the-counter medications that may have affected the diver's performance in the water. Testing for exposure to carbon monoxide to exclude a contaminated gas supply is also necessary. At the pathologist's discretion, histologic examination of the major organs, particularly the heart and lungs, may reveal evidence of significant natural disease processes.

Autopsy interpretation

The presence of pulmonary barotrauma in the form of pneumothorax or subpleural haemorrhage corroborates the occurrence of a gas embolism. Intravascular gas noted at autopsy is commonly misinterpreted by pathologists who are unfamiliar with diving pathophysiology. This diagnosis has been made even in the absence of an ascent, e.g., when a diver has been entrapped in a cave or shipwreck. Anyone who has breathed compressed gas at depth and then surfaces, either on their own or is brought up from depth during body recovery, may have intravascular bubbles at autopsy. Postmortem off-gassing is the process by which dissolved gas that accumulates in tissues during the dive comes out of solution after death. Some of this gas will be intravascular and can mislead the pathologist to conclude that a gas embolism has occurred. Postmortem bubbles and gas accumulation in the left side of the heart and within the arteries at the base of the brain and on the epicardial surfaces are more indicative of a gas embolism having occurred. Bubbles in the inferior vena cava and right side of the heart may simply represent postmortem off-gassing.

If a patent foramen ovale or other potential intracardiac shunt is present, the possibility of paradoxical embolism may be considered. Venous bubbles can arterialise if right heart pressures transiently exceed left heart pressures, which may occur with strenuous lifting or performing the Valsalva manoeuvre. Deaths due to a paradoxical embolism are rare, but this would be a situation where gas embolism may be diagnosed without a rapid ascent or associated barotrauma.

The presence of middle ear, sinus or facial barotrauma may indicate an unconscious descent. The same is true for the presence of haemorrhage into the petrous portions of the temporal bones. In many cases the pathologist will correctly conclude that the cause of death is drowning. There is no definitive autopsy finding that allows the pathologist to make the determination that the diver drowned. However, the presence of congested and hyper-expanded lungs, dilation of the right ventricle of the heart and engorgement of the large central veins, frothy fluid in the large airways, fluid in the sphenoid sinus, watery fluid in the gastric contents are all consistent with drowning.[18]

The potential contribution of natural disease processes to a diving-related death cannot be overstated. Cardiovascular disease appears to be increasing in the diving population and therefore sudden cardiac death while diving or incapacitation from a cardiac dysrhythmia that results in drowning are increasingly common scenarios. In the absence of any definitive postmortem test to confirm whether an arrhythmia or dysrhythmia has occurred, a determination must be based on surrogate markers such as evidence of cardiac disease or abnormality at autopsy. A critical luminal narrowing is generally regarded as being greater than 70%. However, a cardiac event may be triggered simply by cardiomegaly and left ventricular hypertrophy, which are often present in divers who have hypertension. Atherosclerotic cardiovascular disease and hypertensive cardiovascular disease are often seen together at autopsy.

Rarely, the dive history and symptom presentation will indicate that a diagnosis of fatal decompression sickness should be considered. These deaths will typically involve very deep dives with omitted decompression or simply uncontrolled rapid ascents to the surface from excessive depth. Cardiopulmonary decompression sickness, also known as ‘the chokes’, has a characteristic clinical presentation and may rapidly progress to death. This presentation is more common in cases of altitude decompression sickness but also occurs in divers. Frothy blood in the pulmonary vasculature may be present at autopsy. Severe neurological symptoms from decompression sickness are generally more insidious in presentation and, when fatal, the diver has often received multiple treatments with hyperbaric oxygen during hospitalisation. On rare occasions, death will occur prior to recompression. There may be nothing specific in the autopsy findings and death is often due to a complication such as sepsis, pulmonary embolism or multiple organ failure. The pathologist should be provided all related treatment records and antemortem imaging studies. As with all diving-related deaths, proper interpretation of the dive profile is essential. For these cases, it is highly recommended to have the brain and spinal cord examined by a neuropathologist after fixation in formalin.

By utilising postmortem imaging and employing a few modifications in autopsy technique, the pathologist can correctly interpret the autopsy findings. However, it should also be appreciated that a diver may sustain a gas embolism and not have obvious evidence of intravascular gas or pulmonary barotrauma at autopsy. It follows that the circumstances surrounding the death, particularly the dive profile, are even more important than the results of a postmortem examination. That is why it is imperative that the forensic pathologist has at least a functional understanding of diving medicine and that they also be made fully aware of the diver’s medical history and the circumstances surrounding the dive, particularly the dive profile.

Analysis of a diving fatality

Identification of various contributory factors is hampered by the reality that most diving fatality reports are relatively sparse on detail. However, a diving fatality usually involves a series of related events culminating in death. A triggering event leads to a cascade of related events, some precipitated by the diver and some circumstantial. Preceding the trigger may be factors which predisposed to such an event.

An increasing number of studies of diver fatalities have utilised a chain of events analysis (CEA) to depict the suspected sequence of events within the accident. The concept was first applied to USA diving accidents[8] and later, with various modifications, to Australian fatalities,[19,20]

and elsewhere[21] and recently to non-fatal diving incidents.[22] The earlier CEAs divided the accident sequence into four components: a trigger, disabling agent, disabling injury and cause of death.[8] Later, a category of predisposing factors was added to the CEA to account for various factors present before the dive that might influence events.[23]

The major categories for the chain of events analysis are:

- Predisposing factors – A relevant factor(s) that was present prior to the dive, and/or prior to the trigger occurring and which is believed to have predisposed to the accident and/or to key components in the accident chain.
- Trigger – The earliest identifiable event that appeared to transform what would have been an unremarkable dive into an emergency.
- Disabling agent – Actions or circumstances associated with the trigger that caused injury or illness.
- Disabling condition (previously called disabling injury) – The condition that was directly responsible for death or for incapacitation followed by death from drowning.
- Cause of death (COD) – As reported by the medical examiner.
- The COD is no longer included in some CEA reports as it is often not as informative as the disabling condition. The COD of many divers is recorded as drowning. However, the drowning is often secondary to a disabling condition such as cardiac arrhythmia or CAGE, and, from the analysis and preventative perspective it is more valuable to determine the likely disabling condition. Additionally, the disabling agent often adds little to an analysis so is sometimes omitted.

In the absence of definitive criteria, there is a potential for subjectivity in the categorisation of events, and classifications can vary substantively from case to case and between investigators. To minimise subjectivity and increase consistency between investigators, a set of templates with various subcategories was created to provide some guidance when analysing a diving fatality. These are explained in detail elsewhere[23] and summarised in Figure 6.

An example of a fatality scenario and CEA is as follows. A diver with a faulty pressure gauge runs out of air, makes a rapid ascent to the surface, sustains a CAGE, loses consciousness in the water and drowns. In this incident, a predisposing factor was the faulty gauge, the trigger was exhaustion of air supply, the disabling agent was the rapid ascent, the disabling condition CAGE and the COD drowning.

Figure 6
Flowchart of the chain of events analysis for a diving accident

Conclusions

The investigation of dive accidents and fatalities requires a systematic approach based on sound principles and guidelines. The stages include witness interviews, field investigation, evidence collection and preservation, equipment assessment and a thorough and targeted autopsy in the event of a fatality. It is best done by investigators with a thorough familiarity with diving and the investigation processes. Specific expert assistance should be utilised where necessary.

Dive fatality research and reporting is valuable for learning from the misfortunes of the victims. More consistent reporting will help to better identify contributors to diving accidents and so enable the creation or reinforcement of appropriate countermeasures to help mitigate future deaths.

References

1 Walker D. Report of Australian Diving Deaths 1972-1993. Melbourne: J.L. Publications; 1998.
2 Cumming B, Peddie C. National Diving Committee (NDC) diving incidents report 2014. Ellesmere Port, Cheshire: British Sub Aqua Club. [cited 2022 May 3]. Available from: http://www.bsac.com/page.asp?section=1038&sectionTitle=Annual+Diving+Incident+Report.

3 Lippmann J, Taylor D McD, Stevenson C, Williams JW. Challenges in profiling Australian scuba divers through surveys. Diving Hyperb Med. 2018;48:23–30. doi: 10.28920/dhm48.1.23-30. PMID: 29557098. PMCID: PMC6467821.

4 Mozaffarian D, Benjamin EJ, Go AS, Arnett DK, Blaha MJ, Cushman M, et al. Heart disease and stroke statistics – 2015 update: a report from the American Heart Association. Circulation. 2015;131(4):e29–e322. doi: 10.1161/CIR.0000000000000152. PMID: 25520374.

5 Lippmann J, Stevenson C, Taylor D McD. Scuba diving fatalities in Australia, 2001 to 2013. diver demographics and characteristics. Diving Hyperb Med. 2020;50:105–14. doi: 10.28920/dhm50.2.105-114. PMID: 32557411. PMCID: PMC7481108.

6 Buzzacott P, editor. DAN Annual diving report: 2017 edition (A report on 2015 diving fatalities, injuries and incidents). Durham (NC): Divers Alert Network; 2016. [cited 2019 Sep 21]. Available from: https://www.diversalertnetwork.org/medical/report/AnnualDivingReport-2017Edition.pdf. PMID: 29553634.

7 Lippmann J, Taylor D McD. Medical conditions in scuba diving fatality victims in Australia, 2001 to 2013. Diving Hyperb Med. 2020;50:98–104. doi: 10.28920/dhm50.2.98-104. PMID: 32557410. PMCID: PMC7481113.

8 Denoble PJ, Caruso JL, de L Dear G, Vann RD. Common causes of open-circuit recreational diving fatalities. Undersea Hyperb Med. 2008;35:393–406. PMID: 19175195.

9 Lippmann J. Rescue and resuscitation factors in scuba diving and snorkelling fatalities in Australia, 2001–2013. Undersea Hyperb Med. 2020;47:101–9. doi: 10.22462/01.03.2020.11. PMID: 32176951.

10 Sayer MDJ, Azzopardi E. The silent witness: using dive computer records in diving fatality investigations. Diving Hyperb Med. 2014;44:167–9. PMID: 25311326. [cited 2024 Aug 8]. Available from: https://dhmjournal.com/images/IndividArticles/44Sept/Sayer_dhm.44.3.167-169.pdf.

11 Edmonds C. Investigation of diving fatalities. In: Edmonds C, Bennett M, Lippmann J, Mitchell SJ. Diving and subaquatic medicine, 5th ed. Boca Raton (FL): Taylor & Francis; 2016. p. 583–600.

12 Edmonds C, Caruso J. Recent modifications to the investigation of diving related deaths. Forensic Sci Med Pathol. 2014;10:83–90. doi: 10.1007/s12024-013-9491-x. PMID: 24166195.

13 Caruso J. Postmortem, how to. In: Denoble PJ, editor. Investigation of diving fatalities for medical examiners and diving physicians, Symposium Proceedings, June 18, 2014. Durham (NC): Divers Alert Network; 2014. p. 34–41. [cited 2024 Aug 8]. Available from: https://apps.dan.org/publication-library?&token=na.

14 Caruso J. The forensic investigation of recreational diving fatalities. In: Vann RD, Lang MA, editors. Recreational diving fatalities. Proceedings of the Divers Alert Network 2010 April 8-10 workshop. Durham (NC): Divers Alert Network, 2011. p. 34–40. [cited 2024 Aug 8]. Available from: https://www.researchgate.net/publication/51605460_Recreational_diving_fatalities.

15 Lawrence C, Cooke C. Fact File – Autopsy and the investigation of scuba diving deaths. Surry Hills: Surry Hills, NSW: The Royal College of Pathologists of Australasia; 2008.

16 Plattner T, Thali MJ, Yen K, Sonnenschein M, Stoupis C, Vock P, et al. Virtopsy-postmortem multislice computed tomography (MSCT) and magnetic resonance imaging (MRI) in a fatal scuba diving incident. J Forensic Sci. 2003;48:1347–55. PMID: 14640284.

17 Casadesus JM, Aguirre F, Carrera A, Boadas-Vaello P, Serrando MT, Reina F. Diagnosis of arterial gas embolism in SCUBA diving: modification suggestion of autopsy techniques and experience in eight cases. Forensic Sci Med Pathol. 2018;14:18–25. doi: 10.1007/s12024-018-9951-4. PMID: 29460254.

18 Di Maio VJ, Molina DK. Di Maio's forensic pathology, 5th ed. Boca Raton (FL): CRC Press; 2021.

19 Lippmann J, Baddeley A, Vann R, Walker D. An analysis of the causes of compressed gas diving fatalites in Australia from 1972–2005. Undersea Hyperb Med. 2013;40:49–61. PMID: 23397868.

20 Lippmann J, Taylor D McD. Scuba diving fatalities in Australia, 2001 to 2013: chain of events analysis. Diving Hyperb Med. 2020;50:220–29. doi: 10.28920/dhm50.3.220-229. PMID: 32957123. PMCID: PMC7819731.

21 Vinkel J, Bak P, Hyldegaard O. Danish diving-related fatalities 1999–2012. Diving Hyperb Med. 2016;46:142–9. PMID: 27723014. [cited 2024 Aug 8]. Available from: https://dhmjournal.com/images/IndividArticles/46Sept/Vinkel_dhm.46.3.142-149.pdf.

22 Turner BL, van Ooij PJAM, Wingelaar TT, van Hulst RA, Endert EL, Clarijs P, et al. Chain of events analysis in diving accidents treated by the Royal Netherlands Navy 1966–2023. Diving Hyperb Med. 2024;54:39–46. doi: 10.28920/dhm54.1.39-46. PMID: 38507908. PMCID: PMC11227959.

23 Lippmann J, Stevenson C, Taylor D McD, Williams J, Mohebbi M. Chain of events analysis for a scuba diving fatality. Diving Hyperb Med. 2017;47:144–54. doi: 10.28920/dhm47.3.144-154. PMID: 28868594. PMCID: PMC6159623.

Acknowledgment

Adapted and expanded from Chapter 5 Diving Accidents: Investigation of Diving Accidents and Fatalities – Dr John Lippmann – Oxford Specialist Handbook of Diving and Hyperbaric Medicine, In Press, Oxford University Press.

Conflicts of interest and funding: nil

Submitted: 23 May 2024
Accepted after revision: 9 August 2024

Short communication

Large lungs in divers: a risk for pulmonary barotrauma?

Robert A van Hulst[1], Pieter-Jan AM van Ooij[2,3]

[1] *Department of Anesthesiology/Hyperbaric Medicine, Amsterdam University Medical Center, location AMC, Amsterdam, the Netherlands*
[2] *Royal Netherlands Navy Diving and Submarine Medical Centre, Den Helder, the Netherlands*
[3] *Department of Pulmonology, Amsterdam University Medical Centre, Amsterdam, the Netherlands*

Corresponding author: *Professor Robert A van Hulst, Department of Hyperbaric Medicine, Anesthesiology, Meibergdreef 9, 1100 DD, Amsterdam, the Netherlands*
ORCiD: *0009-0002-6945-5508*
r.a.vanhulst@amsterdamuc.nl

Keywords
Air embolism; Lung function; Risk factors; Vital capacity

Abstract

(van Hulst RA, van Ooij PJAM. Large lungs in divers: a risk for pulmonary barotrauma? Diving and Hyperbaric Medicine. 2024 30 September;54(3):225–229. doi: 10.28920/dhm54.3.225-229. PMID: 39288928.)
This retrospective study analysed a series of investigations on lung function in military divers and the importance of computed tomography (CT) scans concerning fitness to dive. We examined the incidence of blebs and bullae in a population of military divers with large lungs prompted by six cases of pulmonary barotrauma. All of these divers' medicals were normal apart from having large lungs (FVC > 120% predicted). A subsequent survey of the database of all divers and submariners of the Royal Netherlands Navy (RNLN) found another 72 divers/submariners with large lungs who were then evaluated by a CT scan. This resulted in the identification of three further individuals with blebs and/or bullae, who were then declared unfit to dive. In total, the incidence of these lung abnormalities in this cohort was 11.5%. We discuss the possible consequences for fitness to dive with regard to the current literature on the subject, and also consider the most recent standards of reference values for pulmonary function indices. Based on our results and additional insights from other studies, we advise using the Global Lung Initiative reference values for pulmonary function, while performing high resolution CT scans only in divers with clinical indications.

Introduction

The occurrence of pulmonary barotrauma during scuba diving is relatively rare but it poses a serious event that can lead to fatalities. It is caused by the expansion of gas during voluntary or pathological air trapping during decompression. As per Boyle's law, upon ascent from a dive, as the ambient pressure decreases, the air in the lungs increases in volume. Any air trapped during ascent can cause rupture of the alveoli, resulting in pneumothorax, pneumomediastinum, subcutaneous emphysema, and/or air entering the pulmonary veins, developing a so-called arterial gas embolism.[1] These accidents are not related to the duration or depth of the dive and are observed mainly in inexperienced divers or in the case of 'blow up' (i.e., rapid) ascents.[2,3]

The Royal Netherlands Navy (RNLN) faced six cases of pulmonary barotrauma in experienced divers within two years (2009–2011). All six of these divers had been medically examined according to international standards, including chest X-ray at initial examination and extensive pulmonary function testing (spirometry and body-box plethysmography) every year.[1,4] Retrospective analysis of their medical files (including X-rays) did not demonstrate abnormalities except that all had 'large lungs' based on the reference values at that date. However, computed tomography (CT) scans made after the diving accidents showed abnormalities in all six cases, although they differed in terms of pulmonary pathology presentation, e.g., blebs, bullae, cysts, or air-trapping, which are contra-indications for fitness to dive.[1,4] After extensive discussions with radiologists, pulmonary physicians, and diving medical officers, it was concluded that these abnormal findings were pre-existing and not related to the accidents. Although initial chest X-rays were routine, we must realise this technique was not sensitive enough to detect minor pathology.

Large lungs are defined by spirometric volumes, and more specifically, according to the European Respiratory Society (ERS) standards at the date of these cases, as having a forced vital capacity above 120% of predicted value.[5] Large lungs in divers have been have been reported in the previous

literature, and are probably related to increased work of breathing during diving due to the density of the breathing gases.[6,7] Furthermore, the healthy worker effect may also account for this observation.[8]

Based on the above, our primary objective in this retrospective study was to identify abnormalities on CT scans in the Netherlands military diving and submariner population with large lungs.

Methods

According to national law, retrospective analyses are exempt from evaluation by a medical ethics committee.

The Royal Netherlands Navy Diving and Submarine Medical Center (DMC) performs yearly medical assessments of military divers in compliance with international standards,[4] while submariners are examined to the same standard every five years. Based on the six diving accidents resulting in pulmonary barotrauma in individuals identified as having large lungs, the Surgeon General of the Royal Netherlands Navy approved the protocol suggested by the National Military Board that the annual assessment of divers with large lungs should, in addition to spirometry, include body plethysmography, diffusion capacity and a CT scan (Min Def U138/DMC.MV/10-9 Dec 2010).

DATA COLLECTION

We retrospectively surveyed the Royal Netherlands Navy (RNLN) database of all divers and submariners to identify those with large lungs, which was determined as per the ERS guidelines at that time and identified another 72 subjects. The identified cohort were subject to CT scans in accordance with the National Military Board protocol described above, allowing this investigation of any lung abnormalities in this population.

PULMONARY FUNCTION TESTING

Pulmonary function was measured by qualified respiratory technicians using the Vmax Encore testing system (Cardinal Health, Bilthoven, the Netherlands) and performed according to the European Respiratory Society guidelines at that date (2010–2011).[5] Calibration of the testing apparatus was performed according to the manufacturer's guidelines. Reference values were calculated using validated prediction equations.

CT PROTOCOL

All CT scans were performed in the Military Hospital, Utrecht, in the Netherlands. In the six pulmonary barotrauma cases, the CT scans were made within one week of the accident. The additional study subjects were scanned in the period December 2010 to June 2011.

Volumetric paired inspiratory and expiratory CT was performed in all subjects and all examinations were conducted using the same scanner (Brilliance 16P, Philips Healthcare) in a single centre. Settings were 120 kVp at 130 mAs for inspiratory CTs and 90 kVp at 20 mAs for expiratory CTs in all subjects. Images were reconstructed with a slice thickness of 1 mm at 0.7 mm increments using a sharp reconstruction kernel (L- and E-filters, Philips Healthcare).[9]

STATISTICAL ANALYSES

After collection of the CT data, the population was split into two groups: those with no anomalies on the CT scan (CT-) and those with anomalies (CT+). All data were tested for normality using the Shapiro-Wilk test. Student's *t*-test was employed to compare data between the CT- and CT+ groups if normally distributed. If there was a non-normal distribution, the Wilcoxon rank-sum test was used. A *P*-value of < 0.05 was considered significant. Analyses were performed using Stata BE software (version 18, StataCorp, USA).

Results

Table 1 shows the age, years diving, and spirometry data pertaining to the original six pulmonary barotrauma cases. Including these six, a total of 78 out of 316 subjects complied with the selection criteria of large lungs and were included in this study. The demographic data of this total group of Navy divers with large lungs are presented in Table 2. In the newly found individuals with large lungs ($n = 72$), upon performing CT scans, we found three additional cases of substantial air trapping or blebs, and these individuals were declared unfit for duty as divers or submariners; in total individuals with pulmonary abnormalities made up 11.5% of this cohort. In another three cases, we found extra-pulmonary findings: haemangioma around one thoracic vertebra, and thymus residuals in two divers; these divers were not excluded from any further diving.

Lung function data, including spirometry and body box plethysmography, are shown in Table 3. Although there was a significant difference in forced vital capacity percent (FVC%) and forced expiratory volume in one second percent (FEV_1%) between the groups, with CT+ having lower values, the FEV_1/FVC data did not differ between the groups. Finally, the peak expiratory flow (PEF) and PEF% were also significantly lower in the CT+ group compared to the CT- group. In our opinion these differences, although significant, are not relevant from a clinical perspective.

Discussion

In this retrospective study of healthy divers and submariners with large lungs, we found an 11.5% (9/78) rate of abnormalities that rendered individuals either unfit for diving

Table 1

Spirometric data in six cases of pulmonary barotrauma; AGE – arterial gas embolism; CT – computed tomography; FEV_1 – forced expiratory volume in 1 second; msw – metres of seawater; ref – reference value; VC – vital capacity; y – years

Case	Age (y)	Smoking (pack years)	Years diving	Dive (depth/ duration)	Type of pulmonary barotrauma	CT findings	Slow VC ml (% ref)	FEV_1 ml (% ref)	FEV_1/VC (%)
1	21	0.1	0	20 msw/5 min	AGE + pneumothorax	Areas of airtrapping in both lungs. No bullae	6,590 (120)	5,300 (118)	80
2	29	0	7	Exit/re-entry 15 msw	Pneumothorax	Area of airtrapping both lungs. CT one year later no anomalies	8,250 (131)	5,870 (118)	71
3	29	4	2	9 msw/? min	Pneumothorax	Several large bullae right lung	7,100 (124)	5,280 (112)	74
4	31	Ex (4 yr) 1.5	8	18 msw/20 min	AGE	Several bullae low dorsal	6,960 (121)	5,140 (110)	73
5	40	0	8	30 msw/? min	Pneumothorax	Subpleural bullae right lung	6,450 (121)	5,130 (119)	80
6	47	0	20	20 msw/45 min	AGE	Bullae in both lungs	5,820 (120)	4,170 (100)	72

or for the free-escape exercises necessary for submariner training. These nine with abnormalities included six individuals who had experienced a pulmonary barotrauma and three who had abnormal CT scans.

High-resolution CT scans (HRCT) effectively detect intrapulmonary anomalies such as blebs, bullae, air trapping, and emphysema, all theoretical risks for pulmonary barotrauma in a population of divers and submariners.[1,2] These abnormalities have an inhomogeneous structure in the lung parenchyma, and lung expansion during ascent may cause lung rupture, leading to pulmonary barotrauma as was found the six cases in the present study.[1,10] However, the relevance of this concept for fitness to dive is still unclear, as the incidence of pulmonary barotrauma in divers is far lower than the occurrence of intrapulmonary abnormalities.[11] It is important that the clinical relevance is defined, as the ruling of an individual as unfit to dive is costly to both the diver and the employer.

The exact prevalence of pulmonary bullae or blebs in the normal population is unknown because it is unacceptable ethically to expose healthy individuals to a dose of radiation without a medical indication. However, two such studies have been made in the diving population over the last 10 years and showed different incidences of blebs and bullae in divers. One study demonstrated a 13% occurrence of abnormalities such as blebs, bullae or air trapping in 330 French military divers,[12] while another found bullae or blebs in seven of 94 subjects (8%).[13] In a systemic bilateral thoracoscopic study, a third study reported the prevalence blebs to be 6% among young healthy individuals.[14] In addition, post-mortem HRCT of 130 subjects without a history of pulmonary diseases found small bullae in approximately 30% of the cohort.[15] This greater incidence is possibly related to a higher radiation dose that can be used post-mortem, as higher dose produces greater sensitivity for detecting smaller blebs and bullae.

In our study, we defined large lungs as 120% above the predicted value of the vital capacity, which was commonly used according to European Respiratory Society standards of that time. Of the divers screened, 24.6% (78 of 316) met this criterion, suggesting that large lungs may be more common in the diving population than the 'normal' population. These European Respiratory Society standards have been discussed extensively due to a lack of accuracy for ethnic groups other than Caucasian, African, and Asian populations. Therefore, in 2008, the Global Lung Initiative (GLI) was established by the European Respiratory Society and the American Thoracic Society to develop all-age reference equations with corrections for gender and ethnic background.[16] Consequently, the Diving Medical Center now uses the GLI-2012 reference values to increase accuracy and prevent medical investigations and/or incorrect assessments of fitness to dive.[17] This protocol has been approved and in use since 2015. Based on the GLI-2012 reference values, we expect to have fewer divers who are outside the 90%

Table 2

Demographic data; data are mean (standard deviation) for normally distributed data, and median (interquartile range) for non-normal data. HRCT – high resolution computed tomography; HRCT- – no CT anomaly; HRCT+ – CT anomaly

Parameter	Total group (*n* = 78)	HRCT– (*n* = 69)	HRCT+ (*n* = 9)	*P*
Age (y)	33.5 (29–45)	34 (29–45)	35 (11)	NS
Height (cm)	184 (7)	184 (7)	184 (5)	NS
Weight (kg)	89 (9)	89 (9)	87 (5)	NS
Smoking (%)	36	33	55	NS
Pack years smokers (y)	1.6 (2.9)	1.6 (3.0)	1.5 (2.3)	NS

Table 3

Lung function values derived from spirometry and body-box plethysmography; data are mean (standard deviation) for normally distributed data, and median (interquartile range) for non-normal data; †*n* = 76; ‡*n* = 7 (2 missing); FEF – forced expiratory flow; FEV_1 – forced expiratory volume in 1 second; FVC – forced vital capacity; HRCT – high resolution computed tomography; HRCT- – no CT anomaly; HRCT+ – CT anomaly; PSF – peak expiratory flow; RV – residual volume; TLC – total lung capacity

Parameter	Total group (*n* = 78)	HRCT– (*n* = 69)	HRCT+ (*n* = 9)	*P*
FVC (L)	7.068 (0.725)	7.114 (0.719)	6.726 (0.717)	NS
FVC%	126 (121–134)	127 (121–135)	121 (120–124)	0.038
FEV_1 (L)	5.384 (0.561)	5.423 (0.555)	5.083 (0.545)	NS
FEV_1%	124 (8)	125 (8)	115 (7)	< 0.01
FEV_1/FVC (%)	76 (5)	77 (5)	76 (4)	NS
PEF (L)	12.4 (1.9)	12.5 (1.8)	11.1 (2.0)	0.039
PEF%	125 (16)	127 (15)	112 (19)	< 0.01
FEF25($L \cdot s^{-1}$)	9.75 (8.4–10.9)	9.9 (8.7–10.8)	9.2 (2.5)	NS
FEF25%	115 (22)	116 (21)	107 (29)	NS
FEF50 ($L \cdot s^{-1}$)	5.6 (4.9-6.4)	5.8 (1.3)	5.2 (1.0)	NS
FEF50%	104 (20)	106 (20)	94 (17)	NS
FEF75 ($L \cdot s^{-1}$)	2.15 (1.8–2.5)	2.3 (1.8–2.5)	1.9 (0.5)	NS
FEF75%	84 (74–100)	85 (76–102)	75 (15)	NS
TLC (L)	8.972 (0.982)†	9.018 (0.981)	8.521 (0.942)‡	NS
TLC%	118 (10)†	119 (10)	114 (106–125)‡	NS
RV (L)	1.936 (0.505)†	1.942 (0.482)	187.6 (0.747)‡	NS
RV%	92 (83–113.5)†	99 (22)	99 (43)‡	NS
RV/TLC	21.4 (4.3)†	21.4 (4.0)	21.6 (6.7)‡	NS

confidence intervals (z-score of 1.64) and therefore do not need additional screening (screening is a work in progress).

Recently, the Finnish Navy published a study on air trapping in 57 divers with large lungs (European Respiratory Society standards definition).[18] The amount of trapped air was calculated by subtracting the total lung capacity measured in a single-breath helium test from the total lung capacity in body plethysmography. In addition, extensive pulmonary function tests were performed including spirometry and airway resistance. They found that large lungs were associated with air trapping and concluded that this phenomenon could be disadvantageous for divers. However, they did not have any cases of pulmonary barotrauma in their population.[18] In our population, four of the nine cases with abnormalities had substantial air trapping; two of these were in the original pulmonary barotrauma case cohort and two were found during later screening.

In our recent follow-up studies based on these original data, we compared routine chest X-rays and HRCT findings in asymptomatic military divers.[17] We concluded that HRCT

detects more abnormalities, but the relevance for fitness to dive is still open to discussion. Therefore, we advise currently that HRCT is performed only in divers with clinical indications, not in all with large lungs. Abnormalities such as blebs bullae or air trapping found either by initial examination or later in the career leads generally to unfitness to dive.[1,4,10]

Conclusions

This study showed that large lungs (as defined by the older European Respiratory Society criteria) do not have a higher incidence of the blebs, or bullae whose presence is thought to pose a higher physiological risk for pulmonary barotrauma in divers. To avoid unnecessary unfit to dive rulings, we suggest using the newer GLI reference values in current populations of divers for interpretation of spirometric data. This is expected to narrow the definition of abnormal lung function and thus reduce the number of individuals deemed to have large lungs. High resolution CT should only be performed in divers with a clinical history or abnormal pulmonary function.

References

1 British Thoracic Society Fitness to Dive Group. British Thoracic Society guidelines on respiratory aspects of fitness for diving. Thorax. 2003;58:3–13. doi:10.1136/thorax.58.1.3. PMID: 12511710. PMCID: PMC1746450.

2 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.

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

4 Health and Safety Executive. The medical examination and assessment of commercial divers (MA1, rev 5, 2023). [cited 2024 Mar 1]. Available from: https://www.hse.gov.uk/pubns/ma1.pdf.

5 Quanjer PH, Tammeling GJ, Cotes JE, Pedersen OF, Peslin R, Yernault JC. Lung volumes and forced ventilatory flows: report working party standardization of lung function tests, European Community for steel and coal. Official Statement of the European Respiratory Society. Eur Respir J Suppl. 1993;16:5–40. PMID: 8499054.

6 Adir Y, Shupak A, Laor A, Weiler-Ravell D. Large lungs in divers: natural selection or a trainings effect? Chest. 2005;128:224–8. doi: 10.1378/chest.128.1.224. PMID: 16002939.

7 Crosbie WA, Reed JW, Clarke MC. Functional characteristics of the large lung found in commercial divers. J Appl Physiol Respir Environ Exerc Physiol. 1979;46:639–45. doi: 10.1152/jappl.1979.46.4.639. PMID: 457539.

8 Tetzlaff K, Thomas PS. Short- and long-term effects of diving on pulmonary function. Eur Respir Rev. 2017;26(143):160097. doi: 10.1183/16000617.0097-2016. PMID: 28356403.

9 Mets OM, van Hulst RA, Jacobs C, van Ginneken B, de Jong PA. Normal range of emphysema and air trapping on CT in young men. AJR Am J Roentgenol. 2012;199:336–40. doi: 10.2214/AJR.11.7808. PMID: 22826394.

10 Wendling J, Nome T. Medical assessment of working divers. Fitness to dive standards of European Diving Technology Committee.1st ed. Biele- Biene; hyperbaric edition. [cited 2024 Mar 1]. Available from: http://www.edtc.org/EDTC-fitnesstodivestandard-2003.pdf.

11 Lafère P, Germonpré P, Guerrero F, Marroni A, Balestra C. Decreased incidence of pulmonary barotrauma after discontinuation of emergency free ascent training. Aerosp Med Hum Perform. 2018;89:816–21. doi: 10.3357/AMHP.5003.2018. PMID: 30126514.

12 Bonnemaison B, Castagna O, de Maistre S, Blatteau JÉ. Chest CT scan for the screening of air anomalies at risk of pulmonary barotrauma for the initial medical assessment of fitness to dive in a military population. Front Physiol. 2022;13:1005698. doi: 10.3389/fphys.2022.1005698.2022. PMID: 36277200. PMCID: PMC9585318.

13 Wingelaar TT, Bakker L, Nap FJ, van Ooij PJAM, Endert EL, van Hulst RA. Routine chest X-ray are inaccurate in detecting relevant intrapulmonary anomalies during medical assessments for fitness to dive. Front Physiol. 2021;11:613398. doi: 10.3389/fphys.2020.613398. PMID: 33488401. PMCID: PMC7816860.

14 Almajid FM, Aljehani YM, Alabkary S, Alsaif HS. The accuracy of computed tomography in detecting surgically resectable blebs or bullae in primary spontaneous pneumothorax. Radiol Med. 2019;124:833–7. doi: 10.1007/s11547-019-01044-6. PMID: 31134432.

15 de Bakker HM, Tijsterman M, de Bakker-Teunissen OJG, Soerdjbalie-Maikoe V, van Hulst RA, de Bakker BS. Prevalence of pulmonary bullae and blebs in postmortem CT imaging with potential implications for diving medicine. Chest. 2020;157:916–23. doi: 10.1016/j.chest.2019.11.008. PMID: 31759963.

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

17 Wingelaar TT, Clarijs P, van Ooij PA, Koch DA, van Hulst RA. Modern assessment of pulmonary function in divers cannot rely on old reference values. Diving Hyperb Med. 2018;48(1):17–22. doi: 10.28920/dhm48.1.17-22. PMID: 29557097. PMCID: PMC6467825.

18 Wuorimaa T, Haukka J, Tikkinen J, Parkkola K, Piirilä P. Large lungs may predict increased air trapping in Navy divers. Physiol Rep. 2022;10(4):e15153. doi: 10.14814/phy2.15153. PMID: 35212176. PMCID: PMC8874342.

Conflicts of interest and funding: nil

Submitted: 4 March 2024
Accepted after revision: 7 June 2024

Case reports

Maxillary sinus barotrauma with infraorbital nerve paraesthesia after breath-hold diving

Kubra Canarslan Demir[1], Zeliha Yücel[2]

[1] *Department of Underwater and Hyperbaric Medicine, University of Health Sciences, Gulhane Training and Research Hospital, Ankara, Turkey*
[2] *Department of Neurology, Karaman Training and Research Hospital, Neurology Clinic, Karaman, Turkey*

Corresponding author: *Dr Kubra Canarslan Demir, SBÜ-Gülhane Eğitim ve Araştırma Hastanesi, Sualtı Hekimliği ve Hiperbarik Tıp Kliniği, Etlik/Ankara, Turkey*
ORCiD: *0000-0001-6911-2375*
drcanarslan@hotmail.com

Keywords
Diving incidents; Diving medicine; Neurological manifestations; Recovery of function; Valsalva manoeuvre

Abstract

(Canarslan Demir K, Yücel Z. Maxillary sinus barotrauma with infraorbital nerve paraesthesia after breath-hold diving. Diving and Hyperbaric Medicine. 2024 30 September;54(3):230–232. doi: 10.28920/dhm54.3.230-232. PMID: 39288929.) Barosinusitis, or sinus barotrauma, is a sinonasal injury and/or inflammation that results when the aerated spaces of the nose and sinuses are exposed to an uncompensated change in ambient pressure. We describe a 19-year-old male diver who presented to our clinic on the fourth day following a breath-hold diving session. During descent on a constant weight monofin dive at the South Cyprus World Championship he began to experience symptoms due to the inability to equalise the pressure, particularly in the Eustachian tubes and middle ear cavities. He felt pain and pressure in the upper left half of his face, left upper molars, and under his left eye at 60 metres, and he continued diving down to 74 metres. At presentation to our clinic, he still had ecchymosis under his right eye and pain in his upper right teeth, half of his face, and ear. He also described tingling in the lower left half of his nose and the left half of his upper lip. He received decongestants, B vitamins, and underwent endoscopic sinus drainage which alleviated his symptoms alleviated over time. The diver reported complete resolution of tingling, numbness, and pain after three months. It should not be forgotten that if appropriate treatment is delayed, permanent changes may occur as a result of long-term compression of the nerve, and therefore patients should be monitored closely.

Introduction

Barosinusitis, or sinus barotrauma, is a condition that describes the varying degrees of sinonasal injury and/or inflammation that result when the aerated spaces of the nose and sinuses are exposed to an uncompensated change in ambient pressure.[1] This causes mucosal injury, which most frequently manifests as headache, odontalgia, cloudy mucus, facial pain or pressure over the afflicted sinuses , and, more seriously, epistaxis. The paired frontal sinuses are the most frequently affected paranasal sinuses, followed in frequency by the maxillary sinuses and, less frequently, the sphenoid sinuses.[1,2] A small number of the more dramatic cases have some additional symptoms.These include nausea or vomiting, a sensation of impending syncope, and disorientation at the time of injury.[3]

Neurological symptoms are not common in sinus barotrauma. Maxillary sinus involvement has been reported less frequently than other sinuses. It was noted that the trigeminal nerve was impacted in certain instances. Pain was attributed to the upper teeth on the side of the maxillary sinus impacted in 4% of cases of maxillary sinus barotrauma. Most likely, the anterior superior alveolar nerve was involved in this. The remaining 4% experienced numbness on the same side of their cheeks due to involvement of the infraorbital nerve.[3–5]

We describe a diver who presented to our clinic with unilateral numbness in the lips and lower half of the nose secondary to sinus barotrauma after breath-hold diving (‘freediving’) to 74 metres.

Case report

The diver provided written consent for publication of his case history and imaging.

A 19-year-old male diver presented to our clinic on the fourth day following a freediving session. The diver experienced symptoms during a constant weight monofin free dive at the South Cyprus World Championship. As he descended to 50 metres of seawater (msw), he began to experience

symptoms due to the inability to equalise the pressure, particularly in the Eustachian tubes and middle ear cavities resulting in pressure difference between the middle ear and surrounding tissue. At 60 msw he felt pain and pressure in the upper left half of his face, left upper molars, and under his left eye, but he nevertheless continued down to 74 msw. The pain was relieved upon resurfacing. The diver had two episodes of epistaxis; immediately after the dive and then when he returned to the hotel one hour later. Tooth and ear pain started again and peaked after 5–6 hours. He took one 50 mg tablet of diclofenac sodium followed one hour later by one 500 mg tablet of paracetamol, and two hours later, another 50 mg tablet of diclofenac sodium. The day after the dive, ecchymosis occurred under the right eye.

The diver had dived for training on the same day or the previous days, but did not experience such symptoms. He had no colds, allergic symptoms, or signs and symptoms of acute or chronic sinusitis in the previous days and weeks.

When the diver presented to our clinic, he still had ecchymosis under his right eye and had pain in his upper right teeth, half of his face, and ear (Figure 1). He also described tingling in the lower left half of his nose and only the left half of his upper lip. Neurological examination revealed paresthesiae in a circular area of 2 cm in diameter including the lower left half of the nose and the upper left half of the upper lip. Light touch, two-point and hot-cold discrimination were not affected.

Complete blood count, erythrocyte sedimentation rate, and C-reactive protein were within normal limits. Paranasal computed tomography (CT) showed soft tissue density that filled the right maxilla and caused a total loss of aeration. The right ostiomeatal unit was found to be obliterated (Figure 2).

The diver was given a decongestant and vitamin B treatment. It was learned that the diver presented to the ear, nose and throat clinic at another hospital as his complaints did not improve, and endoscopic sinus drainage was performed. Although we could not access the report from the hospital, the diver stated that they eluted red-brown liquid. Following the procedure, the diver was given anti-inflammatory and antibiotic treatment. In our later interviews with the diver, he remarked that his symptoms gradually decreased after the surgical treatment and that the tingling, numbness and pain completely disappeared after three months.

Discussion

The paranasal sinuses are complex air-filled chambers enclosed by sturdy outer bony walls and delicate internal walls. Normally, the air pressure inside these sinuses adjusts to match the pressure in the nearby nasal passages via small openings called sinus ostia. In individuals experiencing barosinusitis, the sinus ostia can either be naturally smaller due to slight shifts in wall positions or become constricted and blocked due to local inflammation, swelling, or injury. This hinders the ostia's ability to adjust and support efficient air exchange. The uncompensated changes in intrasinus pressure can result in the mucosal injuries observed in barosinusitis.[6–8]

Risk factors for sinus barotrauma include inflammation of the mucosa, nasal or sinus polyps, significant nasal septum curvature and nasal turbinate hypertrophy.[7,9] Our diver did not have the above risk factors, but he admitted continuing the descent despite the inability to equalise the pressure, particularly in the Eustachian tubes and middle ear cavities to break the record.

Neurological symptoms such as headache and paresthesia in the region innervated by the infraorbital nerve, a branch of the trigeminal nerve, may occur secondary to paranasal sinus barotrauma, especially frontal or maxillary.[10,11] Barotrauma can occur both during descent (as described above), or during ascent due to a relative increase in pressure inside

Figure 1
Ecchymosis under the right eye (arrow) on the 4th day after diving

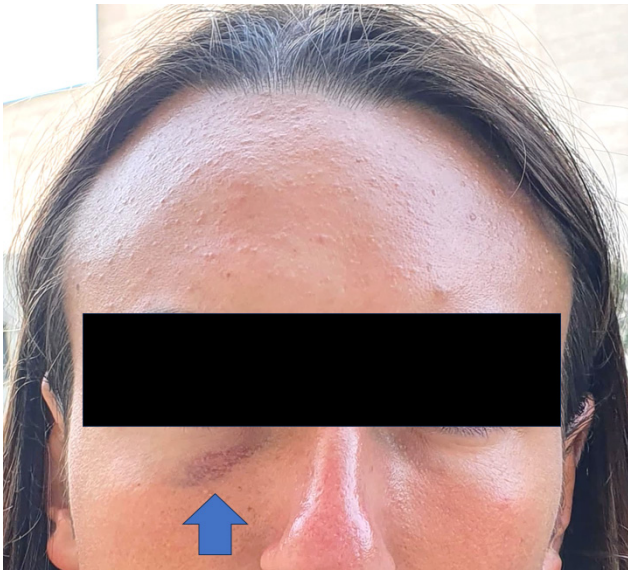
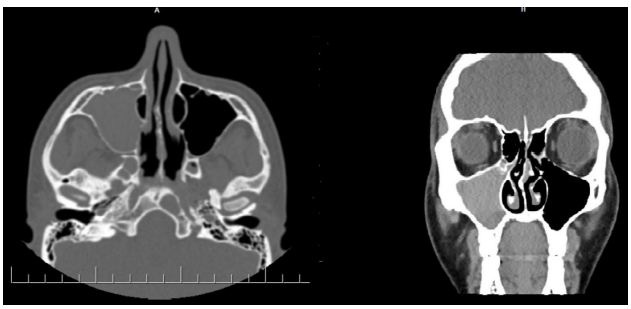


Figure 2
Soft tissue density that completely fills the right maxilla and causes total loss of aeration



the sinus if there is an obstruction to the free outward flow of gas via the antrum.

In 4% of maxillary sinus barotrauma, the pain was referred to the upper teeth on the affected side.[3] This is probably due to anterior superior alveolar nerve involvement. The infraorbital nerve and its branches, which branch from the maxillary branch of the trigeminal nerve and may be affected as they travel along the maxillary sinus wall.[3–5] Involvement of these nerves causes numbness or paresthesiae in the cheek, and numbness of the upper teeth, gums and mucosa on the ipsilateral side. In some cases, pain and hypersensitivity may occur. Problems with neuropraxias are more common following barotrauma of ascent rather than descent; this suggests that circulatory dysfunction or nerve compression is more important than intrasinus haemorrhage as the basis of the pathology.

Our diver had pain and numbness in the right upper teeth and paresthesia on the right half of the lip and at the edge of the nose. The diver's paranasal sinus CT showed congestion in the maxillary sinus and brown fluid during sinus drainage suggested that the diver had bleeding in the right maxillary sinus as a result of sinus barotrauma. The neurological symptoms suggest involvement of the infraorbital nerve and the anterior superior alveolar nerve; perhaps being affected by this bleeding. Even though we don't have definitive evidence. The recovery of the patient could be the result of the anti-inflammatory, the antibiotic, the surgical, or the other treatments or several combinations of them. There are a few related cases in the literature which were primarily treated conservatively, and maxillary sinus drainage was applied in patients who did not improve.[11] Similarly, our patient received initial conservative treatment followed by maxillary sinus drainage. Due to the small number of cases, it remains unclear how long to wait for the response of conservative treatment and when to decide on surgery.

It should not be forgotten that if appropriate treatment is delayed, permanent changes may occur as a result of long-term compression of the nerve, and therefore patients should be closely monitored. Maxillary sinus barotrauma with neurological complications is rarely reported. This case adds to the experience reported in the literature.

References

1 Vaezeafshar R, Psaltis AJ, Rao VK, Zarabanda D, Patel ZM, Nayak JV. Barosinusitis: Comprehensive review and proposed new classification system. Allergy Rhinol (Providence). 2017;8(3):109–17. doi: 10.2500/ar.2017.8.0221. PMID: 29070267. PMCID: PMC5662535.

2 Rusoke-Dierich O. Diving medicine. Barotrauma. Springer International Publishing AG, part of Springer Nature; 2018. p: 167–200. [cited 2024 Mar 20]. Available from: https://doi.org/10.1007/978-3-319-73836-9_26.

3 Edmonds C, Bennett M, Lippmann J, Mitchell SJ. Diving and subaquatic medicine. Boca Raton (FL): CRC Press; 2015. p. 98.

4 Garges LM. Maxillary sinus barotrauma – case report and review. Aviat Space Environ Med. 1985;56:796–802. PMID: 4038236.

5 Edmonds C. Dysbaric peripheral nerve involvement. SPUMS Journal. 1991;21(4):190–7. [cited 2024 Mar 20]. Available from: https://dhmjournal.com/images/IndividArticles/21Dec/Edmonds_SPUMSJ.21.4.190-197.pdf.

6 O'Reilly BJ, Lupa H, Mcrae A. The application of endoscopic sinus surgery to the treatment of recurrent sinus barotrauma. Clin Otolaryngol Allied Sci. 1996;21:528–32. doi: 10.1111/j.1365-2273.1996.tb01104.x. PMID: 9118575.

7 Weitzel EK, McMains KC, Rajapaksa S, Wormald PJ. Aerosinusitis: Pathophysiology, prophylaxis, and management in passengers and aircrew. Aviat Space Environ Med. 2008;79:50–3. doi: 10.3357/asem.2203.2008. PMID: 18225779.

8 Weitzel EK, McMains KC, Wormald PJ. Comprehensive surgical management of the aerosinusitis patient. Curr Opin Otolaryngol Head Neck Surg. 2009;17:11–7. doi: 10.1097/moo.0b013e32831b9caa. PMID: 19235278.

9 Mainardi F, Maggioni F, Lisotto C, Zanchin G. Diagnosis and management of headache attributed to airplane travel. Curr Neurol Neurosci Rep. 2013;13(3):335. doi: 10.1007/s11910-012-0335-y. PMID: 23335028.

10 Mohamad I. Aeroplane headache and sinus barotrauma: any missing link? Cephalalgia. 2012;32(14):1087. doi: 10.1177/0333102412456245. PMID: 22855522.

11 Baş MZ, Şahin SS. Maxillary sinus barotrauma with infraorbital nerve paresthesia after scuba diving: a case report. EurAsian Journal of Oral and Maxillofacial Surgery. 2023;2(4):97–9. [cited 2024 Mar 20]. Available from: https://dergipark.org.tr/en/download/article-file/3613751.

Acknowledgment

We would like to thank the diver for consenting to publication of his case

Conflicts of interest and funding: nil

Submitted: 21 March 2024
Accepted after revision: 21 May 2024

Lateral ST-elevation myocardial infarction from systemic air embolism after CT guided lung biopsy

Aung Myo Htay[1], Emma Wilson[1]

[1] *Department of Diving and Hyperbaric Medicine, Royal Hobart Hospital, Hobart, Tasmania, Australia*

Corresponding author: *Dr Aung Myo Htay, Department of Diving and Hyperbaric Medicine, Royal Hobart Hospital, 48 Liverpool Street, Hobart, Tasmania 7000, Australia*
htay.aung@ths.tas.gov.au

Keywords
Arterial gas embolism; Cardiac; Coronary; Iatrogenic; Complications and management

Abstract

(Htay AM, Wilson E. Lateral ST-elevation myocardial infarction from systemic air embolism after CT guided lung biopsy. Diving and Hyperbaric Medicine. 2024 30 September;54(3):233–236. doi: 10.28920/dhm54.3.233-236. PMID: 39288930.) Systemic air embolism is a rare but potentially life-threatening complication of computed tomography (CT)-guided lung biopsy. The largest lung biopsy audits report an incidence rate of approximately 0.061% for systemic air embolism, with a mortality rate of 0.07–0.15%. A prompt diagnosis with high index of suspicion is essential, and hyperbaric oxygen treatment (HBOT) is the definitive management. We report the case of a 44-year-old lady who developed a lateral ST elevation myocardial infarction from coronary artery air embolism following CT-guided lung biopsy for evaluation of a left lung lesion. The biopsy was performed in the right lateral decubitus position, and the patient reported chest pain after coughing during the procedure. The clinician decided to proceed, taking four biopsy samples as no pneumothorax was identified in the intraprocedural CT image. The patient was noted to have hypotension with ongoing chest pain post-procedure. Resuscitative measures were taken to stabilise her haemodynamics, and she was successfully treated with HBOT with total resolution of air embolism. She developed a left sided pneumothorax post-treatment and needed intercostal chest drain insertion. The left lung fully re-expanded, and the patient was discharged home after day two of admission.

Introduction

Computed tomography (CT)-guided needle biopsy of lung lesions is a common procedure used to evaluate lung nodules or lesions. The procedure is relatively safe. The most common complication is pneumothorax followed by other minor complications such as pulmonary haemorrhage and haemoptysis.[1] Major complications such as pneumothorax requiring intervention, systemic air embolism, haemothorax and death are rare.[1] Although systemic air embolism is a very rare complication, most deaths from this procedure have been attributed to fatal air embolism.[2] Systemic air embolisms can present as arrhythmias, seizures, cardiac ischaemia or stroke.[2] We present a case of cardiac ischaemia after a CT-guided lung biopsy.

Case report

The patient provided written consent for publication of her deidentified case details and imaging.

A 44-year-old lady, on evaluation for non-specific chest pain, was found to have a left peripheral lung lesion that required CT guided lung biopsy. Past medical history included hysterectomy for cervical cancer, thyroidectomy for thyroid cancer, and hypertension treated with amlodipine. She was a smoker with a 40-pack-year history.

A 20G Quick-Core® biopsy needle (IZI Medical, Baltimore, USA) was used for the procedure which was performed in right lateral decubitus position. Her pre-procedure vital signs were normal with BP 101/56 mmHg, heart rate 80·min^{-1} sinus rhythm, respiratory rate 18 with a normal oxygen saturation of 97% breathing room air. During the biopsy the patient coughed, and then complained of chest pain with shortness of breath. She became hypotensive with a systolic blood pressure of 61 mmHg with heart rate 65·min^{-1} and an intravenous fluid (crystalloid) 1 L bolus was given. Blood pressure improved to 85/52 mmHg with heart rate 72·min^{-1}, respiratory rate 28·min^{-1}, oxygen saturation of 97% on 2 L·min^{-1} of oxygen via nasal prongs. There was no pneumothorax or pulmonary haemorrhage noted on intraprocedural CT images.

A medical emergency team call was activated from the CT suites because of ongoing hypotension with chest pain. There were no arrhythmias on cardiac monitor throughout the procedure. A large air embolism was identified in the left ventricle on immediate CT chest (Figure 1). A 12-lead electrocardiogram (ECG) showed ST segment elevation in the lateral territory and ST depression in inferior leads (Figure 2); changes which were not present in the pre-procedure ECG.

Figure 1
Coronal computed tomography view showing biopsy needle placement (larger arrow) and air embolism in LV (smaller arrow)

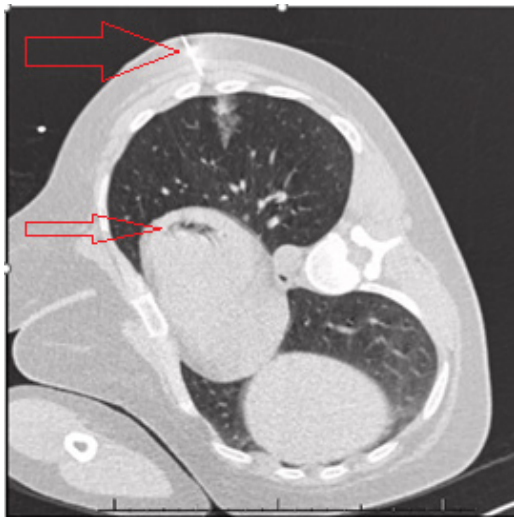


Figure 2
The electrocardiogram after biopsy and prior to hyperbaric oxygen treatment showing ischaemic changes (ST segment elevation in the lateral territory [particularly I and aVL] and ST depression in inferior leads [II, III, and aVF])

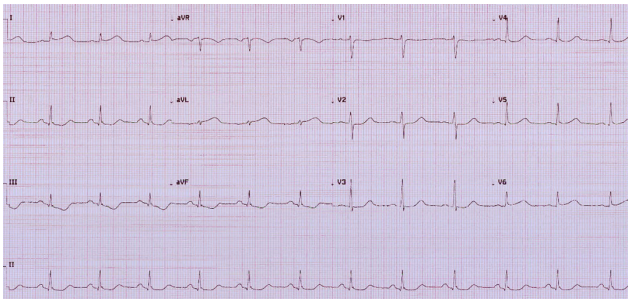
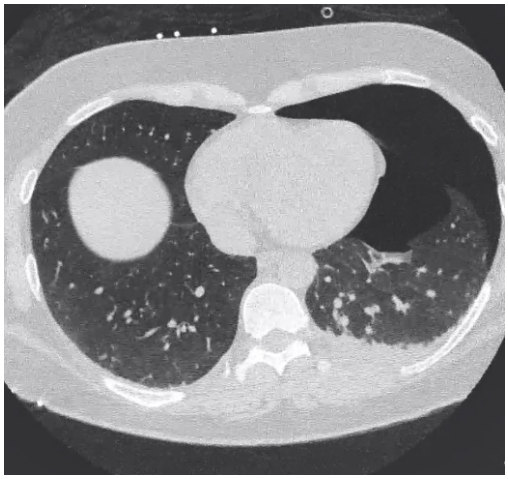


Figure 3
Coronal computed tomography view taken after decompression from hyperbaric oxygen treatment showing a large left pneumothorax but no intracardiac gas



Left ventricle lateral wall and apical hypokinesia was noted on a bedside echocardiogram by the cardiology team. Findings were consistent with a lateral territory ST elevation myocardial infarction from coronary artery air embolism leading to haemodynamic instability.

The patient was given crystalloid fluid boluses up to 2.5 L in total, and two doses of metaraminol (1 mg and 0.25 mg). She was maintained head down in the right lateral decubitus position, keeping the left ventricle up to avoid a systemic air embolism shower. Oxygen was given via humidified high-flow nasal prongs with 50 L flow with 50% FiO_2 by the medical emergency team but was changed to 15 L oxygen with a non-rebreather mask to avoid positive pressure ventilation. An indwelling urinary catheter was inserted before transfer to the hyperbaric unit for hyperbaric oxygen treatment (HBOT).

Compression commenced approximately 90 minutes after air embolism was diagnosed. The patient was compressed to 284 kPa (2.8 atmospheres absolute) over five minutes and breathed 100% oxygen for 45 minutes prior to decompression to surface pressure over 30 minutes with no air breaks.

We chose the treatment table to avoid inert gas (nitrogen) administration to a patient with high risk of pneumothorax and to ensure no decompression requirement for attendants. This provided the capability to decompress at any time in an emergency with the anticipated risks of unstable arrhythmia and cardiogenic shock.

The hyperbaric physician acted as the attendant in-chamber so as to provide the capability of in-chamber intercostal catheter insertion, should she become haemodynamically unstable from a possible expanding pneumothorax on ascent.

The patient complained of left-sided chest pain after finishing decompression, and reduced air entry was noted in the left lung field. A left sided pneumothorax was confirmed by point-of-care ultrasound, with loss of lung sliding. The patient remained haemodynamically stable without needing immediate intercostal catheter insertion. It was therefore decided to return to the CT suite as planned for evaluation of resolution of the left ventricle air collection and quantification of size of the left-sided pneumothorax (Figure 3). The left ventricle air had totally resolved post-HBOT, but she needed an intercostal chest catheter for a large left-sided pneumothorax.

The patient was admitted to the intensive care unit in a stable condition for close observation. The ST segment changes were resolved on serial ECGs. Her initial troponin drawn 40 minutes after initial hypotensive episode with chest pain was normal at 5 $ng{\cdot}L^{-1}$ and peaked at 72 $ng{\cdot}L^{-1}$ 20 hours post-event (normal < 15 $ng{\cdot}L^{-1}$). Her left lung totally re-expanded within 24 hours, and she was discharged home after the second day of admission.

The patient was readmitted on the day of discharge with a recurrent left-sided pneumothorax and large amount of subcutaneous emphysema, following a coughing fit. She was treated conservatively in the Emergency Medical Unit (short stay unit) with 15 L·min^{-1} of oxygen via a non-rebreather mask overnight, and was discharged home the following day, after total resolution of the pneumothorax.

Discussion

COMPLICATIONS FROM CT-GUIDED LUNG BIOPSY

CT-guided lung biopsy is a frequently performed procedure by interventional radiologists, and the rate of common complications are quoted as: pneumothorax (20–35%); pulmonary haemorrhage (4.7–11%); and haemoptysis (2–7%). Systemic air embolism is extremely rare (incidence 0.02% to 0.7%) but may potentially lead to a fatal outcome.[1–4]

MECHANISM OF SYSTEMIC AIR EMBOLISM

The most common mechanism is believed to be that the biopsy needle opens the pulmonary vein to the atmosphere leading to systemic air embolism. Following lung biopsy, fistula formation between the pulmonary vein and the alveoli is another possible mechanism.

RISK FACTORS

Coughing during lung biopsy, positive end-expiratory pressure ventilation, prone position, location of the lesion above the level of the left atrium, and depth of needle in the lesion are documented risk factors associated with systemic air embolism.[4–7] In this case, the lesion was above the level of the left atrium and coughing promoted entrance of air into the pulmonary vein by increasing the pressure gradient between the airway and pulmonary vein.

DIAGNOSIS

Systemic air embolism is a clinical diagnosis, based on a high index of suspicion in patients who develop unexplained hypotension, arrhythmia or new neurological symptoms in the context of lung biopsy. Imaging sensitivity for detecting cerebral air embolism may be as low as 25%.[8] Transoesophageal echocardiography is the most sensitive imaging modality for identification of cardiac air, detecting as little as 0.02 ml·kg^{-1} of air.[9] Imaging is not recommended to make the diagnosis of systemic arterial gas embolism, due to its low sensitivity and its potential to delay definitive management.

MANAGEMENT

Management includes supportive and definitive treatments. Initial supportive measures include maintaining a patent airway, 100% oxygen as first aid, intravenous fluid resuscitation with crystalloids as needed to treat hypovolaemia or hypotension, and inotropes to maintain circulation. Aggressive hydration is unnecessary for isolated arterial gas embolism due to the risk of cerebral and pulmonary oedema. It is recommended to avoid glucose containing intravenous fluid as it can worsen cerebral injury.

Traditionally patients were kept head down with lateral decubitus position for ventricular systemic air embolism, but studies from animal models showed buoyancy had little effect on distribution of air embolism.[10,11] Current consensus is to keep the patient in a supine position to avoid cerebral oedema and to avoid provoking posturally-induced mobilisation of further gas emboli from places like the left atrium or ventricle if cerebral gas embolism is suspected.[12]

High inspired oxygen concentration not only increases nitrogen gradient between bubble and tissue but also improves oxygenation to ischaemic tissue. Administering 15 L·min^{-1} oxygen via a non-rebreather mask is an initial first aid treatment option before HBOT.

Hyperbaric oxygen is the definitive treatment for systemic air/gas embolism. It is indicated for all cases with neurological, cardiopulmonary or other associated clinical abnormalities. Isolated venous gas embolism without arterial gas embolism or clinical manifestations is not an indication for HBOT. Early HBOT is associated with favourable outcome in patients with iatrogenic arterial gas embolism.[13]

Normobaric oxygen therapy is an option with variable results, if HBOT is not available or is contraindicated. In other cases arising from lung biopsy, one who was treated with normobaric oxygen therapy had complete resolution,[14] but another had residual neurological deficit post normobaric oxygen therapy.[15] In a recent third case the patient was initially treated with normobaric oxygen with some improvement, but subsequent deterioration prompted a change in plan and progression to HBOT.[16]

Conclusions

Most systemic air embolism cases related to diving are traditionally treated with US Navy treatment table 6 (Royal Navy table 62), but we treated our patient with modified treatment table with total resolution of air embolism. Decisions should be made on a case-by-case basis, depending on the severity of illness, time to HBOT, and amount of air/gas loading in patients. Further studies should be conducted to confirm if treatment with US Navy Treatment Table 6 is necessary in all cases.

References

1 Tomiyama N, Yasuhara Y, Nakajima Y, Adachi S, Arai Y, Kusumoto M, et al. CT-guided needle biopsy of lung lesions: a survey of severe complication based on 9783 biopsies in Japan. Eur J Radiol. 2006;59:60–4. doi: 10.1016/j.ejrad.2006.02.001. PMID: 16530369.

2 Richardson CM, Pointon KS, Manhire AR, Macfarlane JT. Percutaneous lung biopsies: a survey of UK practice based on 5444 biopsies. Br J Radiol. 2002;75(897):731–5. doi: 10.1259/bjr.75.897.750731. PMID: 12200241.

3 Sinner WN. Complications of percutaneous transthoracic needle aspiration biopsy. Acta Radiol Diagn (Stockh). 1976;17:813–28. doi: 10.1177/028418517601700609. PMID: 1016505.

4 Bou-Assaly W, Pernicano P, Hoeffner E. Systemic air embolism after transthoracic lung biopsy: A case report and review of literature. World J Radiol. 2010;2:193–6. doi: 10.4329/wjr.v2.i5.193. PMID: 21161035. PMCID: PMC2999016.

5 Hare SS, Gupta A, Goncalves ATC, Souza CA, Matzinger F, Seely JM. Systemic arterial air embolism after percutaneous lung biopsy. Clin Radiol. 2011;66:589–96. doi: 10.1016/j.crad.2011.03.005. PMID: 21530954.

6 Franke M, Reinhardt HC, von Bergwelt-Baildon M, Bangard C. Massive air embolism after lung biopsy. Circulation. 2014;129:1046–7. doi: 10.1161/CIRCULATIONAHA.113.004241. PMID: 24589698.

7 Freund MC, Petersen J, Goder KC, Bunse T, Wiedermann F, Glodny B. Systemic air embolism during percutaneous core needle biopsy of the lung: frequency and risk factors. BMC Pulm Med. 2012;12:2. doi: 10.1186/147-2466-12-2. PMID: 22309812. PMCID: PMC3608336.

8 Benson J, Adkinson C, Collier R. Hyperbaric oxygen therapy of iatrogenic cerebral arterial gas embolism. Undersea Hyperb Med. 2003;30:117–26. PMID 12964855.

9 Mirski MA, Lele AV, Fitzsimmons L, Toung TJK. Diagnosis and treatment of vascular air embolism. Anaesthesiology. 2007;106:164–77. doi: 10.1097/00000542-200701000-00026. PMID: 17197859.

10 Butler BD, Laine GA, Leiman BC, Warters D, Kurusz M, Sutton T, et al. Effect of the Trendelenburg position on the distribution of arterial air emboli in dogs. Ann Thorac Surg. 1988;45:198–202. doi: 10.1016/s0003-4975(10)62437-x. PMID: 3341824.

11 Mehlhorn U, Burke EJ, Butler BD, Davis KL, Katz J, Melamed E, et al. Body position does not affect the haemodynamic response to venous air embolism in dogs. Anesth Analg. 1994;79:734–9. doi: 10.1213/00000539-199410000-00020. PMID: 7943784.

12 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.

13 Fakkert RA, Karlas N, Schober P, Weber NC, Preckel B, van Hulst RA, et al. Early hyperbaric oxygen therapy is associated with favorable outcome in patients with iatrogenic cerebral arterial gas embolism: systemic review and individual patient data meta-analysis of observational studies. Crit Care. 2023;27(1):282. doi: 10.1186/s13054-023-04563-x. PMID: 37434172. PMCID: PMC10337083.

14 Galvis JM, Nunley DR, Zheyi T, Dinglasan LAV. Left ventricle and systemic air embolism after percutaneous lung biopsy. Respir Med Case Rep. 2017;22:206–8. doi: 10.1016/j.rmcr.2017.08.007. PMID: 28879078. PMCID: PMC5575445.

15 Al-Ali WM, Browne T, Jones R. A case of cranial air embolism after transthoracic lung biopsy. Am J Respir Crit Care Med. 2012;186:1193–5. doi: 10.1164/ajrccm.186.11.1193. PMID: 23204380.

16 Tsushima R, Mori K, Imaki S. Secondary deterioration in a patient with cerebral and coronary arterial gas embolism after brief symptom resolution: a case report. Diving Hyperb Med. 2024;54:61–4. doi: 10.28920/dhm54.1.61-64. PMID: 38507911.

Acknowledgements

Special thanks to Professor David Smart for guidance of treatment and Professor David Cooper for editing the manuscript.

Conflicts of interest and funding: nil

Submitted: 27 June 2023
Accepted after revision: 7 June 2024

Bispectral index with density spectral array (BIS-DSA) monitoring in a patient with inner ear and cerebral decompression sickness

Gerald Schmitz[1], Sharon Aguero[1]

[1] *Hyperbaric and Undersea Medical Service – Hospital CIMA, San Jose, Costa Rica*

Corresponding author: *Gerald Schmitz, Hyperbaric and Undersea Medical Service – Hospital CIMA, San Jose, Costa Rica*
ORCiD: *0000-0002-1138-8456*
gschmitzg@gmail.com

Keywords
Brain; Cerebral blood flow; Decompression illness; Diving; Inner ear; Electroencephalography

Abstract

(Schmitz G, Aguero S. Bispectral index with density spectral array (BIS-DSA) monitoring in a patient with inner ear and cerebral decompression sickness. Diving and Hyperbaric Medicine. 2024 30 September;54(3):237–241. doi: 10.28920/dhm54.3.237-241. PMID: 39288931.)
Bispectral index with density spectral array (BIS-DSA) monitoring during hyperbaric oxygen therapy of a case with inner ear and cerebral decompression sickness is described. During the initial treatment, a particular DSA pattern was found, which resolved after four treatments. Clinical resolution of the symptoms accompanied this improvement. The particular BIS-DSA pattern described in this case is concordant with a potential hypo-perfusion of the cortex related to decompression stress. This case suggests that BIS-DSA monitoring may be an easy, cost-effective, and viable form of neuro-monitoring during hyperbaric oxygen treatment for decompression sickness.

Introduction

Inner ear decompression sickness (IEDCS) is a challenging clinical presentation of decompression sickness (DCS), not just because there can be a confusing differential diagnosis with inner ear barotrauma, but also because of its uncertain pathophysiology and the high percentage of residual symptoms such as hearing loss and vestibular dysfunction.[1,2] Cerebral decompression sickness (CDCS) seems less frequent, and its diagnosis is mainly based on dysexecutive symptoms and sometimes focal manifestations such as weakness and sensory disturbance.[3] What both presentations of DCS have in common is their frequent association with a right-to-left shunt (RLS) which implicates tiny arterialised venous gas emboli in the pathophysiology.[3] The inner ear may be particularly vulnerable to harm from these tiny arterial bubbles because of persistent supersaturation early after diving[4] which allows even small numbers of arriving bubbles to grow and cause harm.[5] Decompression sickness involving the brain whose luxurious perfusion rapidly eliminates supersaturation, may occur when very large numbers of tiny bubbles arrive synchronously causing injury to micro-vessel endothelium with subsequent inflammatory change.[3] The potential vulnerability of the inner ear to arrival of small numbers of tiny arterial bubbles may explain the frequent observation of inner ear but not cerebral symptoms in individual cases.[6]

Electroencephalography (EEG) is a method for monitoring the electrical activity of the brain. It is widely used in the diagnostic workup of patients with chronic neurological disease but is not easily applied on a day-to-day basis for acute neurological patients. Bi-spectral Index (BIS) is based on the continuous monitoring of a processed frontal EEG, and because of the simplicity of its acquisition, it has been shown to be helpful in anaesthetised patients and some neuro-critical settings to monitor depth of anaesthesia.[7] Another important aspect of the processed frontal EEG is its capability to display the obtained data in clinically useful formats like the density spectral array (DSA) and the spectral edge frequency 95% (SEF). For example, SEF values of 8–13 Hz represent an adequate depth of anaesthesia with most anaesthetic drugs.[8]

Even though the use of EEG has previously been described for DCS, this was mostly done for investigational purposes and it is not used in the standard clinical evaluation of patients with DCS.[9,10] The use of BIS in general and specifically of the DSA has not been described up to this moment in relation to management of acute DCS.

Case report

The patient gave written consent for the publication of his case details.

The patient was a 40-year-old otherwise healthy man qualified as an advanced open water diver, with no dives during the six months prior to the diving accident. On the day of the accident, he dived twice, with a first dive to a maximum depth of 22.3 metres of seawater (msw) (327.3 kPa) for 44 minutes and a second dive after a one-hour

surface interval to 22.4 msw (328.3 kPa) for 45 minutes. Both dives were uneventful, with air as breathing gas.

About 20 minutes after the second dive, the patient started to experience vertigo, which worsened during the next hour and was accompanied by vomiting. Because of the severity of the symptoms, he was evacuated by helicopter and admitted to a general hospital. The patient underwent a central nervous and inner ear computed tomography scan, magnetic resonance imaging of the spine, audiometry, and tympanometry. All test results were normal. D-Dimers were within the normal range.

As DCS was suspected, the patient was transferred to our hyperbaric service, receiving oxygen by mask during transport. During the initial evaluation, the patient was completely alert and conscious. Diminished muscle strength was noted in his left quadriceps (3/5), and a strongly positive Romberg test was elicited, with swaying to the right in less than one second. The patient could not stand without assistance and was not able to walk. No signs of middle ear barotrauma were evident on otoscopic examination. No further neurological or dermatologic signs were apparent.

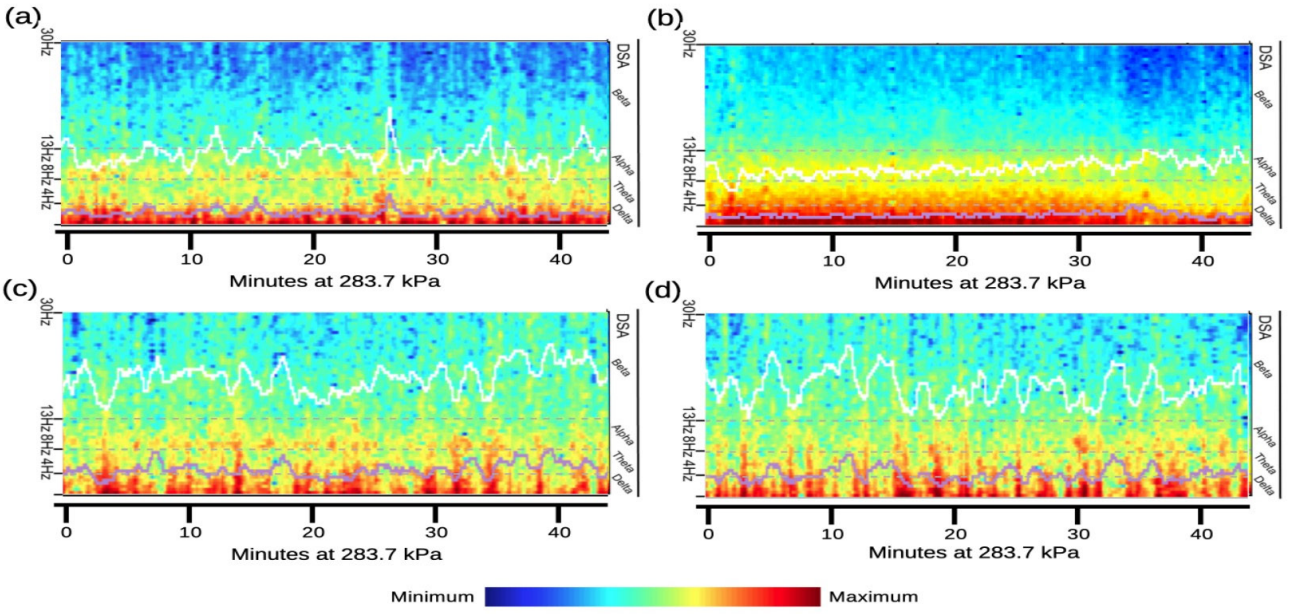
The initial IEDCS risk evaluation showed a value of 11 points on the severity score for inner ear decompression sickness,[11] and the patient was treated with hyperbaric oxygen starting approximately 10 hours after his last dive. The patient was treated in a Sechrist H3300 monoplace hyperbaric chamber (Sechrist Industries, Anaheim – USA). The initial treatment table was based on a traditional United States Navy Treatment Table 6. As the patient reported continuous improvement during the treatment at 283.7 kPa, he received a total of six periods (three extensions) at 283.7 kPa, reporting complete recovery of his muscle strength in his left leg during the last extension and major improvement of his vertigo. He subsequently completed four periods (two extensions) at 192.5 kPa. During the treatment, the patient was monitored according to the monitoring protocol for acute neurological patients of our hyperbaric centre, including electrocardiogram and oxygen saturation on a Mindray BeneVision N19 monitor and, for BIS and DSA on a Covidien BIS 4x monitor (Software Version 3.5) using a bilateral sensor (Ref#186-0212) in the standard position recommended by the manufacturer.

For statistical analysis, the SEF values of the first two periods at 283.7 kPa were considered. Each population of values was first checked for normality with a Shapiro-Wilk test, and the comparison between non-normally distributed samples was made using a Mann-Whitney test. Statistical significance was considered in the case of $P < 0.05$. In order to determine the statistical significance of changes in SEF values in air compared to oxygen periods, baseline SEF was recorded every 10 seconds during the last three minutes on oxygen before an air break and compared to the SEF recorded every 10 seconds during the last three minutes of the air break.

During the first HBO treatment, the patient exhibited a reduction of his heart rate by 16% during the first half hour

Figure 1

Density spectral array for each hemisphere during the first 40 minutes of the first and fourth hyperbaric oxygen treatments. The y-axis represents the wave frequency, while the x-axis shows the minutes elapsed at 283.7 kPa. The white line is the spectral edge frequency 95% limit, and the median frequency is shown as a gray line; a – treatment 1, left; b – treatment 1, right; c – treatment 4, left; d – treatment 4, right



of the treatment, stabilising at an average of 82·min^{-1}. The EEG and the DSA showed a predominance of delta waves, primarily in the right hemisphere, generating an asymmetry towards the right side of the DSA. Bearing in mind that mean SEF values of 23 (standard deviation [SD] 4.2) Hz have been reported normal for awake patients,[12] there was a bilateral reduction that was more pronounced on the right side (11–13 Hz left versus 8–10 Hz right) ($P = 0.02$), accompanied by an important reduction of the variability shown in the standard deviation being 0.44 Hz on the right and 1.1 Hz on the left ($P = 0.01$) (Figure 1a and b). No significant changes were found in the DSA during the air breaks. No signs of convulsions were found on the neuro-monitoring. BIS values oscillated between 82 and 97.

The patient was reassessed six hours after the first treatment, showing a complete resolution of the muscle strength deficit in his left quadriceps. The Romberg test remained positive, swaying to the right after 12 seconds. The patient could walk with limited assistance. Approximately 24 hours after diving he received a second HBO treatment on a Table 6 with two extensions at 283.7 kPa and one extension at 192.5 kPa.

There was a second follow-up evaluation 13 hours after the second treatment. At this time (late on day one after diving), he had a positive Romberg test, swaying to the right at 18 seconds, but could walk without assistance. His third HBO treatment was a traditional Table 6 without extensions. No signs of oxygen toxicity were evident either clinically or on frontal EEG.

The patient was reevaluated on day two after diving, and a completely negative sharpened Romberg test was observed. He could walk without assistance but complained about some limited sensation of instability with no positive signs on the clinical neurological evaluation. His fourth HBO treatment was a Brummelkamp table (283.7 kPa for three periods).[13] It is to be noted that all the treatments the patient received had the same structure at the beginning (283.7 kPa for 2–4 periods, each period composed of 20 minutes on oxygen and 5 minutes on air). This is the reason why, for statistical analysis of the measurements, just the first 50 minutes (two periods at 283.7 kPa) of each treatment were considered.

During the last treatment, the patient showed SEF values within the normal range, being significantly higher than in the first treatment on both sides ($P > 0.01$), without asymmetry on the DSA and with a considerably higher variability (standard deviation of 2.7 Hz compared to the measurements during the first treatment [$P < 0.01$]). During the last treatment, there were no significant differences between the left and the right side (Figure 1c and d).

During the evaluation on day three after diving, the patient was completely asymptomatic and was discharged from the hyperbaric treatment. After the initial management, a transthoracic echocardiogram showed an interatrial septal aneurism. On a follow-up call three months after the initial presentation, the patient informed us that he was diagnosed with a persistent foramen ovale.

Discussion

In the past, IEDCS was supposed to be present in 10–20% of patients with DCS and generally associated with other symptoms of DCS.[14] In actuality, the incidence is higher in some contemporary series, and the association with other symptoms of DCS is considered not to be frequent (75% of cases have no other symptoms of DCS).[4] In the current case, the patient had a clinical presentation of right IEDCS associated with symptoms of motor cortex involvement, expressed as muscle weakness in the left leg, and which is compatible with the findings in the right DSA. A predilection for right-sided IEDCS has been previously described, as has the typical short latency of inner ear and cerebral DCS.[15] The association of inner ear and cerebral DCS with right-left shunts is widely reported and discussed.[16] Based on this, the present case may be considered a typical case of IEDCS with right cerebral involvement.

Up to this moment, neuroimaging has not shown to be sensitive at the initial clinical evaluation of these patients[3] but may become more useful at later follow-ups,[16] when ischaemic areas may be found.[17] In contrast to neuroimaging, neuromonitoring is infrequently utilised in DCS, and most reports are limited to EEG in experimental settings. The clinical use of BIS-DSA monitoring during hyperbaric treatment of DCS has not been reported to our knowledge.

Electroencephalography is considered to have low sensitivity for cerebral DCS. Observed abnormalities include slow waves and sharp potentials primarily on the right temporal and occipital regions, with the slow wave being the most typical finding.[9] This predominance of slow waves is compatible with the lower SEF values seen in the present case, as the wave frequency distribution shifts towards lower frequencies, lowering the SEF value.

It is notable that in previous reports, the EEG was obtained after the HBO treatment. A controlled study estimated the incidence of abnormal EEG to be 57.1% before HBO treatment and observed a significant normalisation of the EEG after the HBO treatment.[10] In our case, we used continuous BIS-DSA monitoring during the HBO treatments, initially showing a particular pattern (low average SEF in the alpha range with reduced variability, primarily on the right side) that has not been described before. The pattern resolution was consistent with the clinical improvement during the progress of the treatments.

As previously mentioned SEF values below 13 Hz are typically associated with sedated states. In this case, the patient was completely alert. The pattern shown by the patient is compatible with the loss of high frequency in the EEG that may be seen by low blood flow, causing local

hypoxia in the tissue, mainly related to the oxygen-sensitive layers III, V, and VII of the cortex.[18] In animal models, a reduction of cortical blood flow related to DCS was previously demonstrated.[19] A recent animal study suggested that a neuro-inflammatory state via micro-particles may be involved.[20]

Autopsy reports in cases of cerebral DCS concluded that the patient exhibits alterations of the permeability of the blood vessels and activation of the blood clotting system, resulting in cerebral oedema, perivascular haemorrhages, and oedematous necrosis of the white matter.[21,22] A diffuse perivascular inflammation generated by decompression stress may cause a reduction of local blood flow and/or favour an intracellular hypoxic condition, affecting mainly cortex layers III, V, and VII, potentially causing the BIS-DSA pattern seen in this patient.

Conclusions

The symptoms of the patient indicated a right-sided cerebral involvement that goes in hand with the major changes of the DSA in the right frontal hemisphere.

The fact that the DSA on the left side also deviated from normal values without presenting any clinically evident symptoms points to the hypothesis that CDCS may be a more diffuse and mostly subclinical entity.

The DSA pattern seen in this case is characterised by a bilateral reduction of the SEF with a significant reduction of its variability, both more extensive on the symptomatic right side.

BIS-DSA may be considered a vital monitoring option during hyperbaric therapy in acute neurological conditions and may be a valuable clinical tool in the diagnosis of CDCS.

To our knowledge, this is the first report of BIS-DSA monitoring during HBO treatment in a clinical setting in general and in a patient with inner ear and cerebral DCS in particular.

References

1 Lindfors OH, Räisänen-Sokolowski AK, Hirvonen TP, Sinkkonen ST. Inner ear barotrauma and inner ear decompression sickness: a systematic review on differential diagnostics. Diving Hyperb Med. 2021;51:328–37. doi: 10.28920/dhm51.4.328-337. PMID: 34897597. PMCID: PMC8923696.

2 Sokolowski SA, Räisänen-Sokolowski AK, Tuominen LJ, Lundell RV. Delayed treatment for decompression illness: factors associated with long treatment delays and treatment outcome. Diving Hyperb Med. 2022;52:271–6. doi: 10.28920/dhm52.4.271-276. PMID: 36525684. PMCID: PMC10026386.

3 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.

4 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.

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

6 Tremolizzo L, Malpieri M, Ferrarese C, Appollonio I. Inner-ear decompression sickness: 'hubble-bubble' without brain trouble? Diving Hyperb Med. 2015;45:135–6. PMID: 26165540.

7 Mulvey DA, Klepsch P. Use of processed electroencephalography in the clinical setting. Curr Anesthesiol Rep. 2020;10:480–7. doi: 10.1007/s40140-020-00424-3. PMID: 33110400. PMCID: PMC7581499.

8 Jildenstål P, Bäckström A, Hedman K, Warrén-Stomberg M. Spectral edge frequency during general anesthesia: a narrative literature review. J Int Med Res. 2022;50:3000605221118682. doi: 10.1177/03000605221118682. PMID: 35971317. PMCID: PMC9386875.

9 Grønning M, Risberg J, Skeidsvoll H, Moen G, Aanderud L, Troland K, et al. Electroencephalography and magnetic resonance imaging in neurological decompression sickness. Undersea Hyperb Med. 2005;32:397–402. PMID: 16509281.

10 Sipinen SA, Ahovuo J, Halonen JP. Electroencephalography and magnetic resonance imaging after diving and decompression incidents: a controlled study. Undersea Hyperb Med. 1999;26:61–5. PMID: 10372424.

11 Gempp E, Louge P, de Maistre S, Morvan JB, Vallée N, Blatteau JE. Initial severity scoring and residual deficit in scuba divers with inner ear decompression sickness. Aerosp Med Hum Perform. 2016;87:735–9. doi: 10.3357/AMHP.4535.2016. PMID: 27634609.

12 Nieuwenhuijs D, Coleman EL, Douglas NJ, Drummond GB, Dahan A. Bispectral index values and spectral edge frequency at different stages of physiologic sleep. Anesth Analg. 2002;94:125–9, table of contents. doi: 10.1097/00000539-200201000-00024. PMID: 11772814.

13 DeMis J, Keuski BM, Due A. Hyperbaric treatment deviations for U.S. Navy Divers: spinal DCS. Undersea Hyperb Med. 2023;50:383–90. PMID: 38055878.

14 Kennedy RS, Diachenko JA. Incidence of vestibular symptomatology in 2,500 U.S. Navy Diving Accidents (1933–1970). Aviat Space Environ Med. 1975;46(4 Sec 1):432–5. PMID: 1080048.

15 Francis TJ, Pearson RR, Robertson AG, Hodgson M, Dutka AJ, Flynn ET. Central nervous system decompression sickness: latency of 1070 human cases. Undersea Biomed Res. 1988;15:403–17. PMID: 3067433.

16 Peppas S, Palaiodimos L, Nagraj S, Kokkinidis DG, Tiwari N, Kharawala A, et al. Right-to-left shunt in divers with neurological decompression sickness: a systematic review and meta-analysis. Healthcare (Basel). 2023;11:1407. doi: 10.3390/healthcare11101407. PMID: 37239692. PMCID: PMC10217987.

17 Schwerzmann M, Seiler C, Lipp E, Guzman R, Lövblad KO, Kraus M, et al. Relation between directly detected patent foramen ovale and ischemic brain lesions in sport divers. Ann Intern Med. 2001;134:21–4. doi: 10.7326/0003-4819-134-1-200101020-00009. PMID: 11187416.

18 Jordan KG. Emergency EEG and continuous EEG monitoring in acute ischemic stroke. J Clin Neurophysiol. 2004;21:341–52. PMID: 15592008.
19 Leitch DR, Hallenbeck JM. Somatosensory evoked potentials and neuraxial blood flow in central nervous system decompression sickness. Brain Res. 1984;311:307–15. doi: 10.1016/0006-8993(84)90093-3. PMID: 6498488.
20 Bhat AR, Arya AK, Bhopale VM, Imtiyaz Z, Xu S, Bedir D, et al. Persistent neuroinflammation and functional deficits in a murine model of decompression sickness. J Appl Physiol (1985). 2024;137:63–73. doi: 10.1152/japplphysiol.00097.2024. PMID: 38660728.
21 Funakoshi K, Yamada K, Tokufuji SI. A pathological study on cerebral lesions in divers decompression sickness (DCS). South Pacific Study. 1990;10:275. [cited 2024 May 21]. Available from: http://cpi.kagoshima-u.ac.jp/publications/southpacificstudies/sps/sps10-2/SouthPacificStudies10(2)pp275-285.pdf.
22 Waller SO. Autopsy features in scuba diving fatalities. Med J Aust. 1970;1:1106–8. doi: 10.5694/j.1326-5377.1970.tb84447.x. PMID: 5430490.

Conflicts of interest and funding: nil

Submitted: 18 May 2024
Accepted after revision: 6 July 2024

Decompression sickness in surface decompression breathing air instead of oxygen

Jan Risberg[1], Helle Midtgaard[2]

[1] *NUI, Bergen, Norway*
[2] *Oslo University Hospital, Oslo, Norway*

Corresponding author: *Dr Jan Risberg. NUI, Gravdalsveien 245, 5165 Laksevåg, Bergen, Norway*
jri@nui.no

Keywords
Decompression illness; Decompression tables; Diving tables; Incidents; Occupational diving

Abstract

(Risberg J, Midtgaard H. Decompression sickness in surface decompression breathing air instead of oxygen. Diving and Hyperbaric Medicine. 2024 30 September;54(3):242–248. doi: 10.28920/dhm54.3.242-248. PMID: 39288932.)
We report an unusual decompression sickness (DCS) incident in a commercial diving project. Eleven divers completed 91 dives to 23.5–36.2 m with bottom times ranging 23–67 min. The divers were breathing compressed air while immersed. Decompression was planned as surface decompression in a deck decompression chamber breathing oxygen typically for 15–30 min. Due to a technical error the divers breathed air rather than oxygen during the surface decompression procedure. Two divers suffered DCS. Both were recompressed on site with the same error resulting in them breathing compressed air rather than oxygen. One of them experienced a severe relapse with cardiovascular decompensation following recompression treatment. While DCS was expected due to the erroneous decompression procedures, it is noteworthy that only two incidents occurred during 91 dives with surface decompression breathing air instead of oxygen. Accounting for this error, the median omitted decompression time was 17 min (range 0–26 min) according to the Bühlmann ZHL-16C algorithm. These observations suggest that moderate omission of decompression time has a relatively small effect on DCS incidence rate. The other nine divers were interviewed in the weeks following completion of the project. None of them reported symptoms at the time, but five divers reported having experienced minor symptoms compatible with mild DCS during the project which was not reported until later.

Introduction

Decompression sickness (DCS) is a known complication of diving.[1] The likelihood of contracting DCS is reduced by adherence to recognised decompression tables, but the possibility can never be eliminated. Most surface-oriented diving is planned to allow uninterrupted ascent to surface ('no-decompression diving') or by staged in-water decompression stops at defined depths. An alternative approach to traditional in-water staged decompression stops is to shorten or completely remove the staged in-water decompression stops and rather recompress the diver immediately after surfacing in a deck decompression chamber. The divers will breathe oxygen through tight fitting masks integrated with the built-in-breathing-system (BIBS) of the deck decompression chamber. The benefits of surface decompression with oxygen ($SurDO_2$) include shortened in-water decompression time, avoidance of sea swell and thermal comfort during decompression. Oxygen breathing in the deck decompression chamber allows a shorter decompression time compared to air breathing conventionally used in in-water staged decompression. Surface decompression on oxygen has been suspected to cause a high DCS incidence.[2] However, probabilistic modelling suggests that dives planned according to the US Navy Diving Manual[3] may be completed with DCS probability in the same order as in-water air decompression, and with the above advantages.[4]

We report two cases of treated DCS caused by the incorrect administration of breathing gas (air instead of oxygen) during surface decompression. While we would expect a high DCS probability due to this error ('deserved DCS'), we discuss the fact that many other dives were completed uneventfully in spite of the same procedural error.

The SI unit for pressure is Pa, but decompression tables by convention use meters of seawater (msw) whether referencing to diver's ambient pressure immersed or in the dry environment. For this reason, we have retained the use of msw in this report (10 msw approximately = 100 kPa).

Case reports

The protocol for this work was presented to the Norwegian regional ethics committee west (REC west) for a submission assessment. REC west concluded that no formal ethical review was required. Both divers have read the contents of the manuscript before they gave their written permission to share the contents in public.

BACKGROUND

The commercial dives took place spring 2023 in the harbor of a Norwegian city. The diving company had been awarded a contract to extract oil from a sunken vessel. The wreck was located at a depth of approximately 30–35 msw. The objective of the diving project was to identify the locations of the bulkheads, clean areas using water jets, drill holes in the hull and connect flexible pipes to allow the extraction of the oil. The wreck had a significant list, and the divers could usually stand on the draught or on the sea bottom under the keel and work at fixed water depths. Divers' depth and bottom time were monitored electronically. Diving was done from a small diving support vessel equipped with a deck decompression chamber.

A total of 101 dives were completed of which 91 were $SurDO_2$ dives and 10 were no-decompression dives or conventional staged in-water decompression dives. All dives were planned with compressed air as breathing gas and the $SurDO_2$ decompressions followed nationally approved decompression tables (Norwegian Diving and Treatment Tables).[5] The Norwegian Diving and Treatment Tables $SurDO_2$ dives are slightly modified (metric converted) versions of the US Navy (USN) Diving Manual Rev 7 $SurDO_2$ tables.[3] None of the $SurDO_2$ dives called for staged in-water decompression stops preceding chamber recompression. The procedures instruct the diver to ascend at 10 msw·min^{-1} to surface, undress and move into the deck decompression chamber and then immediately start breathing oxygen by BIBS. The diver should be recompressed to 15 msw (253 kPa) within a 5 min surface interval. The first 15 min of oxygen breathing takes place at a chamber depth of 15 msw, followed by oxygen breathing at 12 msw. The diver should breathe chamber air for 5 min following every 30 min of BIBS oxygen breathing to reduce oxygen toxicity. Decompression from 15 or 12 msw to surface pressure is at 10 msw·min^{-1}.

Due to human error, stored compressed air rather than oxygen was connected to the BIBS system. For a period of 17 days 91 dives were completed with surface decompression breathing air rather than oxygen on BIBS until this error was discovered and corrected.

PREDICTING OMITTED DECOMPRESSION WHEN BREATHING AIR DURING $SURDO_2$

To illustrate the expected consequence of erroneously breathing compressed air rather than oxygen on BIBS during surface decompression, the optimal method would be to apply a probabilistic DCS model such as those developed by the USN.[6] However, such models were not available for us, so we decided to illustrate the extent of omitted decompression using the Bühlmann ZHL-16C model. For this purpose, we used the 'DecoPlanner' software version 4.6.5 (GUE, High Springs, FL) to calculate the required decompression for the entire procedure conducted breathing air. The software was provided such input data for each of the 91 surface decompression dives:

- Breathing gas air (FO_2 = 21%).
- Bühlmann algorithm, ZHL-16C, 100-100 gradient factors.
- Descent rate (compression) 18 msw·min^{-1}, and ascent rate (decompression) 10 msw·min^{-1}.
- Bottom time and depth as relevant for the specific dive.
- Ascent to 6 msw, isopressure for 4 min.
- Recompression to 15 msw.
- Air breathing at 15 and 12 msw as relevant for the specific dive.

It should be noted that the 'surface interval' was placed at 6 msw rather than surface which would have been correct based on the actual profile. The reason is that the software would not allow further diving or decompression calculation if any shallower stop than 6 msw was programmed after finished bottom time.

Following the data input, the software was requested to 'plan dive' and provided output as a recommended time of decompression at 3 msw. We will use the term 'Bühlmann-recommended decompression' for the time at 3 msw recommended by DecoPlanner and 'omitted decompression' for the discrepancy between the recommended time and the actual time spent decompressing.

Case descriptions

Two DCS incidents occurred during this diving project.

CASE ONE

The first affected a 48-year-old man, previously healthy with approximately 1,000 dives in his occupational diving career. The incident occurred on the fourth day of this project and his third day of diving. He had been diving a shallow no-decompression dive two days earlier and an uneventful dive to 27.9 msw for 42 min with surface decompression the preceding day without any symptoms. On the day of the incident, he was diving to 28 msw for 55 min. He was recompressed to 15 msw in the deck decompression chamber and followed a 45 min BIBS-schedule. Approximately 1 h after finishing surface decompression he experienced distorted vision, described as flickering, chest discomfort and left upper extremity paraesthesiae. He subsequently developed what was described by the supervisor as a mottling discoloration of the skin on the upper thorax and both upper extremities. He was recompressed therapeutically in the deck decompression chamber approximately 1 h 40 min after finishing surface decompression. The treatment table (Norwegian Diving and Treatment Tables[5] Table 6) is a slightly modified version of the US Navy Treatment Table 6.[3] He experienced immediate improvement of symptoms during recompression and remaining skin discoloration and symptoms disappeared during the initial part of recompression treatment. Due to

the erroneously connected compressed-air gas cylinder, he was breathing compressed air rather than oxygen through the BIBS during surface decompression as well as recompression treatment.

He was transported to Oslo University Hospital. Approximately 35 min after finishing treatment, and shortly before hospital arrival, he suffered visual disturbances (shimmering lights), loss of power and intense pain in right upper arm and relapse of the skin rash. On arrival at the hospital, he reported worsening discomfort, severe abdominal pain, photophobia, nausea, hypaesthesia in his upper extremity and some reduction of power in right upper extremity. However, neurological examination was normal, but a reticular blueish discoloration of chest and shoulder skin was evident as was a non-pitting edema in right upper arm. He needed analgesia for his abdominal pain.

He was provided hyperbaric oxygen treatment (HBOT) and experienced immediate relief of most symptoms during the treatment. Abdominal pain persisted for some time after he finished treatment, but a repeated neurological examination did not reveal any abnormal findings. He developed hypotension and haemoconcentration and was admitted to the intensive care unit for intravenous rehydration. He received a total of four HBOT sessions at the hospital before he was released. He suffered some minor discomfort in his right lower arm and right shoulder for a short time after treatment but was in a normal state when interviewed eight months following the event.

CASE TWO

The second incident occurred with a 35-year-old previously healthy man with approximately ten years of experience in occupational diving and some 300 dives. He had never experienced DCS. This event occurred on the 18th day of diving as dive number 100 in the diving project. He had completed 10 dives preceding the incident, nine of these with surface decompression. He was diving to 28 msw for 50 min and was obliged to complete 30 min of oxygen breathing during surface decompression. He experienced the first symptoms approximately 1 h after completing surface decompression. He complained of a rash, feeling of heat and itching in the skin of his left shoulder area. He was recompressed on site according to Norwegian Diving and Treatment Tables Table 6 and experienced immediate relief of itching during recompression and a gradual regress of the rash during the initial part of the treatment. Shortly before the planned decompression from 9 msw to surface pressure, it was realised that he had been breathing compressed air rather than oxygen on the BIBS and the treatment table was prolonged with two additional 25-min oxygen breathing periods at 3 msw. He remained asymptomatic. When examined at the hospital immediately following treatment no abnormalities were found and he has remained well since.

UNEVENTFUL DIVES AND DIVERS NOT TREATED FOR DCS

As mentioned earlier, eleven divers participated in this diving project with a total of 101 man-dives. The two divers suffering DCS completed 14 of these dives. The other nine divers completed 79 uneventful dives with surface decompression in addition to eight no-decompression and staged in-water decompression dives. The 91 surface decompression dives were completed to depths ranging 23.5–36.2 msw and bottom time ranging 23–67 min. Three, eighty-five and one man-dives were completed uneventfully with inadvertent compressed-air BIBS-breathing of 15, 30 and 45 min respectively during what was assumed to be $SURDO_2$. As described above, one dive with 30 min and one dive with 45 min inadvertent air breathing during surface decompression caused DCS (Figure 1).

As seen from Figure 1 many dives were completed to a depth of approximately 28 msw for a bottom time of approximately 50 min. This profile would call for 15 + 15 min of oxygen breathing at 15 and 12 msw according to the Norwegian Diving and Treatment Tables.[5] When air breathing through the BIBS an additional 20 min of decompression time should be added at 3 msw according to the DecoPlanner implementation of the Bühlmann algorithm. Figure 2 shows the difference between the 28 msw/50 min profile as it was dived in this project and how it compares to a recommended profile in DecoPlanner.

When all surface decompression profiles were analysed with DecoPlanner, the median omitted decompression time was 17 min (range 0-26 min, IQR 7 min).

The nine divers not treated for DCS were offered a consultation and medical examination with a diving physician on project day 21 (four days following the last DCS). Four of the divers accepted the offer, none of them exhibited abnormal clinical findings. In addition, they were all interviewed by the company diving physician by telephone in the weeks following the incidents. None of the other divers had raised any complaints following their dives, but when requested in the aftermath for symptoms they reported complaints as detailed in Table 1.

Discussion

Decompression sickness is a recognised occupational illness of diving.[1] The incidence rates per 100,000 dives have been reported to range 4–10 in recreational diving,[1] 0 to 100 in occupational diving[7] and 13 in military diving.[8] A study of 1980's North Sea offshore diving reports a much higher incidence for $SurDO_2$ dives with high inert gas load compared to no-decompression dives.[2] This observation is in agreement with statistical analysis (probabilistic models) of the decompression tables used at the time.[9]

Figure 1

Diving depth and bottom time for 97 dives in a diving project; no-decompression (NoDC) and in-water decompression dives (IWDC) and dives stipulated to include 15, 30 or 45 min of O_2 breathing on BIBS during assumed surface decompression on oxygen are presented with different symbols. Four additional NoDC dives to depths < 6 msw or bottom time < 10 min are excluded to allow better resolution. The two dives with decompression sickness (DCS) are indicated with red crosses

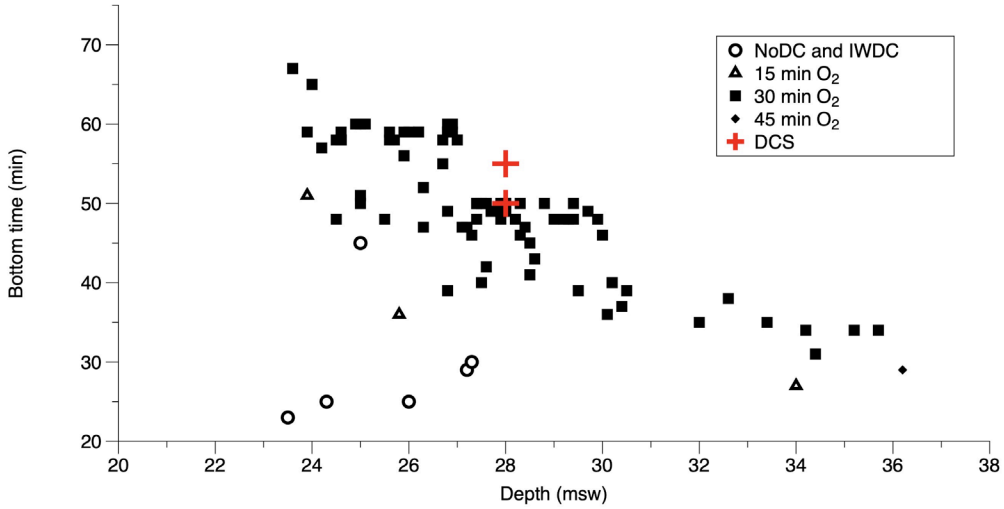
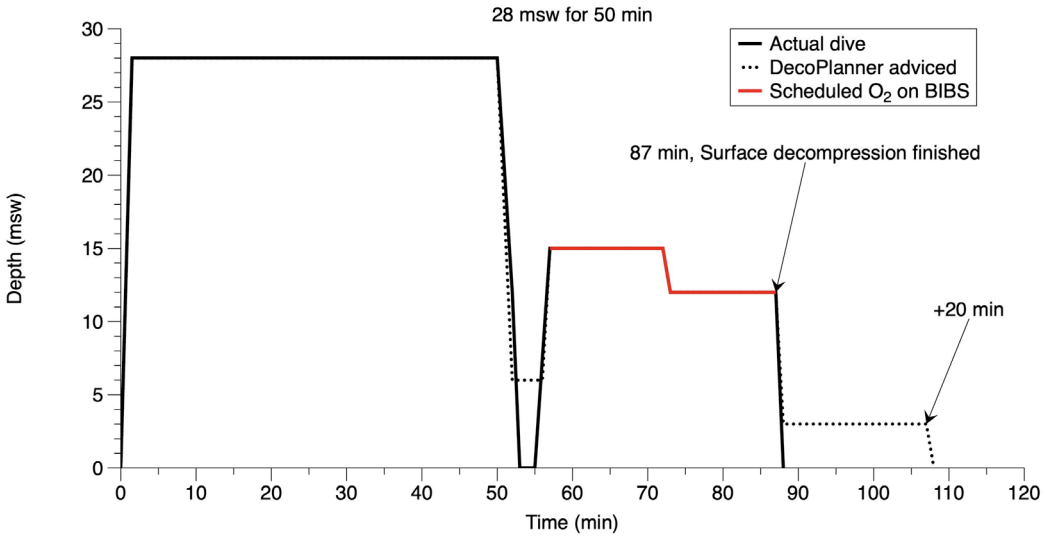


Figure 2

Comparison of a typical dive profile in the present diving project (continuous line) and a recommended decompression as calculated by DecoPlanner (dotted line) for the dive. As can be seen, DecoPlanner (Bühlmann algorithm) would advise a 20 min stop at 3 msw before surfacing. The red line indicates the time the diver should breathe oxygen on BIBS according to the Norwegian Diving and Treatment Tables[5]



In this work we have reported two cases of DCS occurring in 91 dives assumed to be conducted using $SurDO_2$, a 2.2% incidence rate. This incidence rate is much higher than commonly reported in Norwegian in-shore[5] and off-shore[10] surface-oriented diving and this can be attributed to the inappropriate breathing gas. As explained earlier, the $SurDO_2$ decompression tables in the Norwegian Diving and Treatment Tables[5] are slightly modified (metric converted) versions of $SurDO_2$ tables published in USN Diving Manual Rev 7.[3] The schedules used in this diving project would be expected[4] to have a DCS incidence rates ranging 2.5–3.1% with a 95% confidence interval of approximately ± 0.5% if the divers had been provided oxygen during surface decompression. It is tempting to speculate why the observed DCS incidence rate for schedules with inappropriate breathing gas didn't exceed that predicted by the probabilistic model. It is possible that divers under-report symptoms and that the true incidence of DCS in this project was higher. We will return to that later.

Table 1
Symptoms reported by the divers to the company diving medical advisor 14–26 days following the last incidence of DCS. None of the symptoms were reported to the dive supervisor or the company diving physician during the project period. NRS – numeric rating scale /10

Diver	Symptoms experienced during the project period
1	Case #1. No complaints until the day he experienced decompression sickness.
2	Case #2. Slight itching after one of the dives. Can't remember which.
3	Telephone interview day 14: Dived twice. Felt discomfort over left elbow after both dives. Relationship to dive unknown. Lasted for a couple of days.
4	Telephone interview day 14: Slight itching and numbness on dorsal aspect of left hand after dive #1/10. Slight bilateral shoulder discomfort after dive #4/10 persisting for 12 days, no remaining symptoms 23 days after the dive. Day 4 examination by diving physician: Slight shoulder discomfort as described above. No abnormal findings.
5	Telephone interview day 25: No symptoms experienced during project period. Day 4 clinical examination by diving physician: No symptoms. No abnormal findings.
6	Telephone interview day 14: He experienced slight headache after dive #3/11 and #4/11. Experienced foul smelling diesel fume in the helmet during these dives. Headache NRS 5 presenting approx. 1 h after finished dive. No headache in the morning on the succeeding days. Day 4 examination by diving physician: No remaining symptoms. No abnormal findings.
7	Telephone interview day 14: He might have experienced some additional fatigue in the evenings, but no focal symptoms.
8	Telephone interview day 14: He experienced pain in right knee and both elbows. Itching lower back, both wrists and both underarms. Headache after the first dives (NRS 2–3). Can't remember relationship between symptoms and diving in any further detail. No residual complaints. Day 4 clinical examination by diving physician: Complaints during previous dives as described above. No remaining symptoms. No abnormal findings.
9	Telephone interview day 14: He noticed bilateral elbow pain for a few days during the project, pain exaggerated by physical exertion. This coincided with a period of intense physical exercise. The pain appeared unrelated to diving and developed gradually. No remaining symptoms.
10	Telephone interview day 19: He did not experience any problems during the diving period
11	Email day 19 and telephone interview day 26: He experienced temporary itching after one of the dives (can't remember which). This occurred on the same day he started to use a new diving suit undergarment. No remaining symptoms.

It should be recognised that DCS incidence rates predicted by the probabilistic model are based on dives completed with diving depth and bottom time equal to the scheduled depth and bottom times in the USN Diving Manual Rev 7.[3] While working depth was almost constant for each dive in this diving project, it did not necessarily reach table depth, neither did actual bottom time equal the maximum allowed bottom time. This fact is shared with similar studies on DCS occurrence in operational diving and calls for a caveat when reporting decompression table safety based on epidemiological studies. While two DCS incidents occurred in 92 dives (2.2%) before the error was corrected, it should be appreciated that the binominal 95% confidence interval ranged from 0.3–7.6%, illustrating that the true underlying incidence might have been higher. A better analysis of the dives would require access to a probabilistic model which regrettably was unavailable for us. To partly compensate for this fact, we have estimated the amount of omitted decompression time if the dives had been planned according to Bühlmann ZHL-16C algorithm. Ninety-one dives had omitted 4–26 min of decompression time, while only two of these resulted in DCS. This finding seems to support the

notion that the effect of adding or subtracting 5–10 min of decompression, such as commonly applied when modifying the 'conservatism factor' of a dive computer, will have relatively small effect on the DCS risk. Others have stated that "Also large increases in TST are required to effect relatively small decreases in DCS risk due to the low slope of the P(DCS)min/TST line" (TST: Total stop time).[11]

Seven divers retrospectively reported having suffered minor symptoms during the project period. These were not reported to the dive supervisor or medically investigated (Table 1). It is the authors' opinion that five of these divers have experienced symptoms that might have been caused by DCS or alternatively a variety of trivial and not necessarily diving-related causes. It is not possible to retrospectively establish a diagnosis with any confidence, but the findings demonstrate symptom under-reporting in commercial diving. Such reporting bias can likely affect DCS estimates reported in epidemiological studies. Experimental studies designed to validate decompression tables typically report DCS incidence of 2–5% depending on study objective,[6] whereas much lower numbers are reported in epidemiological studies.[1,7,8] One group recently reported findings of a prospective study in which questionnaires were completed by 55 Finnish technical recreational divers.[12] They completed 2,983 dives during the one-year observation period, 27 (1%) resulted in symptoms that could have been caused by DCS. All but one was self-treated, i.e., the incidence of therapeutically recompressed DCS was 0.03%. A similar finding was reported in a 1994 questionnaire study involving 1,200 Norwegian recreational and 800 occupational divers.[13] The survey reached a 63% response rate. Forty-eight of 365 in-shore occupational divers had been treated for DCS. Fifty percent of the divers reported previous decompression-related but untreated symptoms. Central nervous system symptoms such as forgetfulness, irritation, depression, and attention deficits were reported by 21% of divers treated for DCS. However, 67% of the divers reporting decompression-related but untreated symptoms complained of such symptoms. The findings of these studies[12,13] suggest that unreported decompression-related symptoms may be common and possibly be associated with long term neurological sequelae.

Case 1 developed hypotension and needed fluid replacement following the inappropriate recompression treatment when he was breathing compressed air rather than oxygen on BIBS. A similar development was reported in another case report.[14] However, severe cardiopulmonary DCS (chokes) is usually associated with severe omitted decompression such as the one reported by Arjomand et al.[15]

Conclusions

We have reported two divers exhibiting DCS following surface decompression. This was most likely caused by erroneous breathing gas in the BIBS. One of them suffered relapse with severe pain, hypotension and intravascular hypovolaemia following a recompression treatment involving the same error (breathing air in the BIBS). While these two incidents were expected due to the inappropriate breathing gas, it should be noted that 89 dives (98%) were completed uneventfully despite incorrect breathing gas and omission of a median decompression time of 17 min.

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 Shields TG, Duff PM, Wilcock SE, Giles R. Decompression sickness from commercial offshore air-diving operations on the UK continental shelf during 1982 to 1988. Society for Underwater Technology (SUT) Subtech '89 «Fitness for Purpose». Aberdeen, Scotland: Kluwer Academic Publishers; 1989. p. 259–77.

3 Supervisor of Diving. U.S. Navy Diving Manual Revision 7 Change A. Washington, DC; 2018. Report No.: SS521-AG-PRO-010. [cited 2021 Dec 31]. Available from: https://www.navsea.navy.mil/Home/SUPSALV/00C3-Diving/Diving-Publications/.

4 Gerth WA, Doolette DJ. VVal-79 Maximum permissible tissue tension table for Thalmann algorithm support of air diving. Report No.: NEDU TR 12-01. Panama City (FL): Navy Experimental Diving Unit; 2012. [cited 2021 Dec 25]. Available from: https://apps.dtic.mil/sti/pdfs/ADA561928.pdf.

5 Risberg J, Møllerløkken A, Eftedal OS. Norwegian Diving- and Treatment Tables, 5th ed. Bergen: Personal publisher; 2019. [cited 2021 Dec 25]. Available from: http://dykketabeller.no/onewebmedia/NDTT%20Ed%205.pdf.

6 Thalmann ED, Parker EC, Survanshi SS, Weathersby PK. Improved probabilistic decompression model risk predictions using linear-exponential kinetics. Undersea Hyperb Med. 1997;24:255–74. PMID: 9444058.

7 Imbert JP. Decompression tables versus decompression procedures: an analysis of decompression sickness using diving data bases. EUBS annual meeting proceedings: Heraklion, Greece; 1991. p. 223–31.

8 Arness MK. Scuba decompression illness and diving fatalities in an overseas military community. Aviat Space Environ Med. 1997;68:325–33. PMID: 9096830.

9 Gerth WA, Doolette DJ. VVal-18 and VVal-18M Thalmann algorithm air decompression tables and procedures. Report No.: NEDU TR-07-09. Panama City (FL): Navy Experimental Diving Unit; 2007. [cited 2021 Dec 25]. Available from: https://diving-rov-specialists.com/index_htm_files/scient-c_298-VVal18-and-VVal18M-thalmann-algorithm.pdf.

10 Petroleumstilsynet. Rapport fra Petroleumstilsynets dykkedatabase DSYS – 2022 [Report from Petroleum Safety Authority Norway's diving database DSYS – 2022]. 2023. [cited 2024 Apr 13]. Available from: https://www.havtil.no/contentassets/7284426234ae40cdaa62e2037ed2bf35/dsys_2022-rapport.pdf.

11 Tikusis P, Gerth WA. Decompression theory. In: Brubakk AO, Neuman TS, editors. Bennett and Elliott's physiology and medicine of diving, 5th ed. Edinburgh: Saunders; 2003. p. 419–54.

12 Tuominen LJ, Sokolowski S, Lundell RV, Raisanen-Sokolowski AK. Decompression illness in Finnish technical

divers: a follow-up study on incidence and self-treatment. Diving Hyperb Med. 2022;52:78–84. doi: 10.28920/dhm52.2.74-84. PMID: 35732278. PMCID: PMC9527095.
13 Brubakk AO, Bolstad G, Jacobsen G. Helseeffekter av luftdykking. Yrkes og sportsdykkere. Revisjon 1 [Health effects of air diving. Occupational and recreational divers. Rev 1]. Report No.: STF23 A93053. Trondheim: SINTEF UNIMED; 1994.
14 Klapa S, Meyne J, Kähler W, Tillmans F, Werr H, Binder A, et al. Decompression illness with hypovolemic shock and neurological failure symptoms after two risky dives: a case report. Physiol Rep. 2017;5(6):e13094. doi: 10.14814/phy2.13094. PMID: 28325788. PMCID: PMC5371546.
15 Arjomand A, Holm JR, Gerbino AJ. Severe decompression sickness associated with shock and acute respiratory failure. Case Rep Crit Care. 2020;2020:8855060. doi: 10.1155/2020/8855060. PMID: 33204543. PMCID: PMC7661127.

Acknowledgements

We appreciate the kind support of David Humphreys (NUI a.s.) reviewing the text.

We appreciate the generous support from SubseaPartner providing funding to let this manuscript to be released immediately for public access.

Conflicts of interest and funding: nil

Submitted: 13 April 2024
Accepted after revision: 9 August 2024

Hyperbaric oxygen treatment (HBOT) in a case of traumatic chondronecrosis of the cricoid cartilage

Subhranshu Kumar[1], HBS Chaudhry[1], Chandrasekhar Mohanty[1], Sourabh Bhutani[1], Muhammed Risham[1], Kshitij Lanjekar[1]

[1] *Department of Marine Medicine, Institute of Naval Medicine, INHS Asvini, RC church, Colaba, Mumbai, India*

Corresponding author: *Dr Subhranshu Kumar, Department of Marine Medicine, Institute of Naval Medicine, INHS Asvini, RC church, Colaba, Mumbai, India*
subhranshukumar19@gmail.com

Keywords
Chronic wounds; Larynx trauma; Hyperbaric medicine; Outcome; Tracheostomy complication

Abstract

(Kumar S, Chaudhry HBS, Risham M, Lanjekar K, Mohanty C, Bhutani S. Hyperbaric oxygen treatment (HBOT) in a case of traumatic chondronecrosis of the cricoid cartilage. Diving and Hyperbaric Medicine. 2024 30 September;54(3):249–251. doi: 10.28920/dhm54.3.249-251. PMID: 39288933.)

Cricoid chondronecrosis is a rare entity and is scarcely reported in the literature. Its prevalence is increasing in the form of chondroradionecrosis among the survivorship of head and neck carcinoma patients treated with radiotherapy. We have reported a case of cricoid chondronecroisis caused by trauma from repeated tracheostomy. The patient presented with hoarseness and dyspnoea. Radiological findings in multidetector computed tomography showed disintegration of the cricoid and confirmed the diagnosis. Conservative treatment was given in the form of antibiotics, steroids and nebulised anticholinergics and bronchodilators. However, the patient did not improve and his condition worsened throughout two months of hospitalisation. He was referred for hyperbaric oxygen treatment, which was given over 30 sessions. This was associated with improvement in his condition and he was able to be decannulated from tracheostomy. Six monthly follow up of the patient showed a well-healed tracheostomy scar.

Introduction

Traumatic chondronecrosis is a rare consequence of cricoid trauma.[1] Trauma to the cartilage either from direct assault or during airway management can lead to chondronecrosis, which is difficult to manage.[2] Treatment typically consists of the use of antibiotics, steroids and surgery in severe life-threatening conditions, but prognosis remains poor.[1,2] Hyperbaric oxygen treatment (HBOT) has occasionally been reported as effective in cases failing to heal with other interventions. We report a case of traumatic chondronecrosis of the cricoid in which HBOT was used after the condition failed to improve with antibiotics and steroids.

Case report

The patient gave written consent for publication of his case details and medical images.

A 31-year-old male presented to our centre with difficulty breathing and hoarseness of voice. He had been injured in a hand grenade blast while participating in a military training activity eight months previously. He suffered from a severe head injury, his left thumb was blown away, and he also sustained a comminuted fracture base of the first metacarpal of the same hand. After initial stabilisation at the nearby hospital, he was transferred to a tertiary care hospital. His definitive management included right decompressive hemicraniectomy and surgical debridement of the left hand under general anaesthesia. On the sixth day of surgery, he developed difficulty in breathing due to poor cough reflex and retention of secretions. His oxygen saturation was decreasing and he was managed with a planned percutaneous tracheostomy, which remained *in situ* for five days. After stabilisation, decannulation of his tracheostomy was performed. The patient remained in the hospital for another two weeks for observation. Once, he appeared stable without any active intervention, he was discharged from the hospital for recovery. During the convalescent period at home, he developed acute breathlessness and again reported to a hospital near his home. He was diagnosed with acute laryngotracheobronchitis and managed conservatively with medication. He responded well and fully recovered.

After two months, the patient again developed severe breathlessness and hoarseness. An emergency tracheostomy procedure was performed with 8.0 mm inner diameter, single lumen, cuffed, non-fenestrated tracheostomy tube. After 10 days, the tracheostomy tube was changed to a 7.0 mm inner diameter, double lumen, fenestrated tube. Pre- (without contrast) and post- (with contrast) multidetector computed tomography (CT) views of the neck were obtained from the base of the skull to the carina. This revealed fragmentation of the cricoid cartilage lamina predominantly along the

posterior aspect with extensive soft tissue component causing partial airway narrowing (Figure 1). Contrast media was used for better visualisation of the soft tissue. Based on the above findings he was diagnosed with chondronecrosis of the cricoid cartilage. He was managed with antibiotics, corticosteroids and nebulisation, for around three months.

The hoarseness and dyspnoea did not improve with the medication and his clinical condition deteriorated over the three months while the tracheostomy tube was in situ. He was referred to our centre for HBOT, given the poor wound healing even after adequate treatment. Problem wounds are an approved indication for HBOT per the Undersea and Hyperbaric Medical Society (UHMS). After ascertaining fitness for HBOT, a trial session was given at 243 kPa (2.4 atmospheres absolute) for 60 minutes without an air break. For the purposes of treatment, the tracheostomy tube was temporarily closed and HBOT was given via a face mask. A hyperbaric physician remained in the chamber with the patient in case of any complications arising from closure of the tracheostomy. The patient tolerated the trial session without any complaints or complications. During HBOT, antibiotics were stopped and steroids were tapered off slowly. Tracheostomy care was given simultaneously with the ongoing HBOT to avoid late complications of the tracheostomy. Although HBOT can cause acute complications in terms of barotraumas, oxygen toxicity and chronic complications like pulmonary changes and cataracts none of these problems were observed.

Breathing started becoming comfortable at the 8th to 10th session of HBOT and dyspnoea improved at around the 22nd session. Decannulation was successfully performed after the 26th session but HBOT was continued for six days a week for a total of 30 sessions. The hoarseness improved significantly during and after HBOT. Indirect laryngoscopy after the completion of 30 sessions of HBOT showed oedema subsidence in the glottis and subglottic area, a decrease in the size of granulation tissue, and a reversal of narrowing in the subglottic area. The patient underwent repeat neck CT at one month follow-up which confirmed the remodelling of the cricoid cartilage architecture with opening up of the airway (Figure 2). At the 3rd month follow up, complete resolution of dyspnoea was noted, and the tracheostomy scar had healed well without any complication.

Discussion

While cricoid chondronecrosis is a rare entity and is scarcely reported in the literature, the prevalence of chondroradionecrosis is increasing among survivors of head and neck carcinoma treated with radiotherapy.[3] We present a case of traumatic chondronecrosis of cricoid cartilage. The presentation of cricoid chondronecrosis may include hoarseness, dyspnoea, dysphagia and open discharging sinuses over the neck. All of these symptoms have highly debilitating consequences for the patients.

Figure 1
Computed tomography scan of neck region showing fragmented cricoid cartilage (yellow arrow)

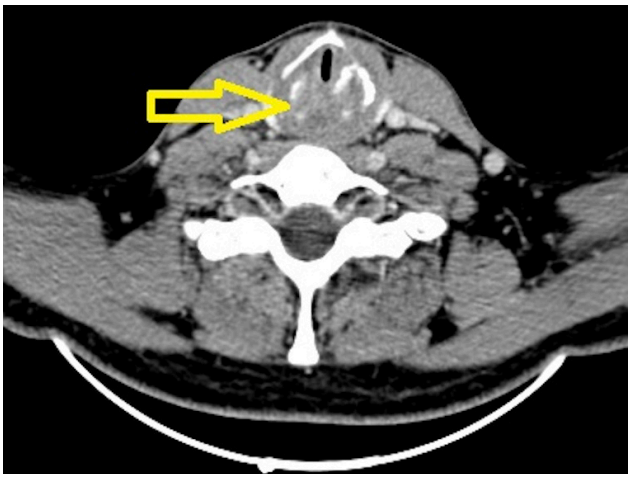
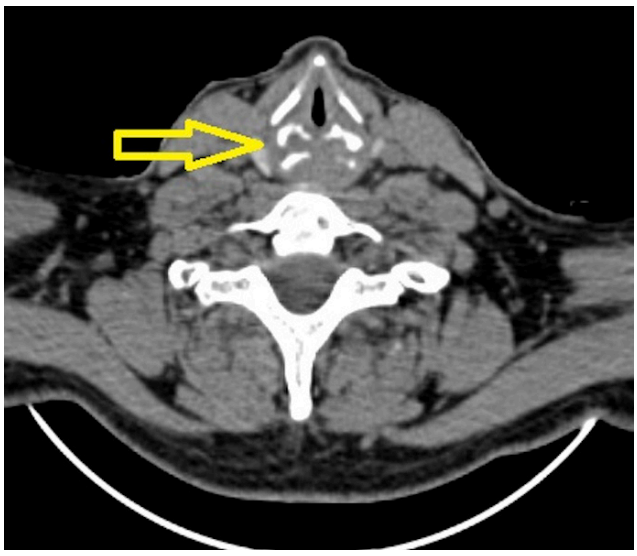


Figure 2
Computed tomography scan of head and neck region showing restoration of cricoid cartilage (yellow arrow) with resolution airway narrowing



Understanding the anatomy of cricoid cartilage including its blood supply is necessary to interpret the pathology behind its necrosis. This cartilage is the only cartilage of the laryngeal box which completely encircles the airway. It is made up of hyaline cartilage and resembles a signet ring which is narrow in the anterior side and widens posteriorly. It is avascular and oxygen and nutrients through diffusion from the surrounding capillaries. Its capacity for regeneration is consequently poor and this is a slow process.[4] The external capillaries supply only the submucosa of the cartilage and the internal perichondrial plexus provides the majority of nutrients to the cartilage. The compromise of the internal perichondrial plexus often leads to chondronecrosis of the cricoid cartilage.[2] In our case, repeated episodes of tracheostomy were thought to be the causative factor.

In radiation-induced necrosis, radiation causes hypoxic, hypovascular, and hypocellular changes, which impairs cell replication and repair and causes cartilage breakdown.[5] Apart from the radiation exposure, any mechanical trauma in the form of intubation, direct mucosal damage by high cuff pressure of a tracheostomy tube, poorly fitted or excessive movement of the tube can cause breach in the mucosa. This exposure can trigger an inflammatory response which can lead to fibrosis or necrosis[6,7] A detailed history of the patient regarding exposure to the factors causing chondronecrosis of cricoid cartilage will generally indicate the pathology causing the disease. After a detailed history, investigation modalities like laryngoscopy and CT help in diagnosis. Laryngoscopy can find the narrowing of the airway, oedema and granulation tissue but it will be a non-specific sign. Computed tomography scanning can find and quantify the glottic and subglottic narrowing, the presence of air in the central lamina of the cricoid, a hypodensity or disruption or fragmentation of the cortex of the cricoid and helps in confirming the diagnosis.[8]

Conservative treatment of cricoid chondronecrosis includes antibiotics and corticosteroids. Although the use of HBOT has been supported by studies in radiation-induced soft tissue injury (including cartilage), it may also be useful in chondronecrosis of cricoid caused by mechanical trauma or other etiologies like aseptic necrosis. The use of HBOT in patients with tracheostomy tubes *in situ* needs special consideration, as airway narrowing may causes difficulty in using a face mask. Hoods, T-tubes or the use of a monoplace chamber may be preferred but it all depends upon the available resources. Hyperbaric oxygen has multiple potentially relevant mechanisms in this setting. Periodic reversal of hypoxia and supports oxygen dependent wound healing processes. The standing PO_2 gradient and hyperoxia-induced vasoconstriction may reduce local tissue oedema and thus reduce the airway narrowing causing symptomatic improvement.[9] The anti-inflammatory role of HBOT in decreasing the pro-inflammatory cytokines and increasing the anti-inflammatory cytokines is widely discussed and documented in the literature.[10] The intermittent increases in oxygen tension can induce many of the mediators and cellular mechanisms that are usually induced during hypoxia. This is called hyperoxic-hypoxic paradox.[11] The potential benefits to healing include elevation of hypoxia inducible factor 1 and 2, mobilisation of vasculogenic stem and progenitor cells, and local elaboration of vasculogenic cytokines. These cell-signaling effects seem induced by transient increases in reactive oxygen species.[12] All these factors work in tandem and help in healing.

Conclusions

In cases of chondronecrosis of the cricoid, a multidisciplinary approach to treatment must be considered. The treatment protocols for this rare entity are variable because of its scarce mention in the literature. Clinicians should be aware of HBOT as an option in management, particularly in serious or refractory cases. Although a randomised controlled trial of HBOT in this indication would be desirable, the rare and sporadic nature of cases make such a study unlikely. More reports or observational series are encouraged.

References

1 Palled K, Bhat V, Hassan S, Ganamukhi M. Imaging appearance of a post-intubation cricoid chondronecrosis. BJR Case Rep. 2016;2(3):20150442. doi: 10.1259/bjrcr.20150442. PMID: 30459988. PMCID: PMC6243369.

2 Mims MM, Leclerc AA, Smith LJ. Cricoid chondronecrosis: case report and review of literature. Ann Otol Rhinol Laryngol. 2020;129:662–8. doi: 10.1177/0003489420904974. PMID: 32070112.

3 Roh JL. Chondroradionecrosis of the larynx: diagnostic and therapeutic measures for saving the organ from radiotherapy sequelae. Clin Exp Otorhinolaryngol. 2009;2:115–9. doi: 10.3342/ceo.2009.2.3.115. PMID: 19784402. PMCID: PMC2751874.

4 Mathews S, Jain S. Anatomy, head and neck, cricoid cartilage. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan. PMID: 30969643.

5 Marx RE, Johnson RP. Studies in the radiobiology of osteoradionecrosis and their clinical significance. Oral Surg Oral Med Oral Pathol. 1987;64:379–90. doi: 10.1016/0030-4220(87)90136-8. PMID: 3477756.

6 Beswick DM, Collins J, Nekhendzy V, Damrose EJ. Chondronecrosis of the larynx following use of the laryngeal mask airway. Laryngoscope. 2015;125:946–9. doi: 10.1002/lary.24967. PMID: 25345975.

7 Charlin B, Dehon A, Bergeron D, Mongeau CJ, Grondin P. Aseptic necrosis of the cricoid: a complication of tracheal intubation. J Otolaryngol. 1987;16:377–81. PMID: 3694746.

8 Ali AA, Shweihat YR, Bartter T. Cricoid chondronecrosis: a complication of endotracheal intubation. J Ark Med Soc. 2012;108(9):192–4. PMID: 22435316.

9 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.

10 Woo J, Min JH, Lee YH, Roh HT. Effects of hyperbaric oxygen therapy on inflammation, oxidative/antioxidant balance, and muscle damage after acute exercise in normobaric, normoxic and hypobaric, hypoxic environments: a pilot study. Int J Environ Res Public Health. 2020;17(20):7377. doi: 10.3390/ijerph17207377. PMID: 33050362. PMCID: PMC7601270.

11 Hadanny A, Efrati S. The hyperoxic-hypoxic paradox. Biomolecules. 2020;10(6):958. doi: 10.3390/biom10060958. PMID: 32630465. PMCID: PMC7355982.

12 Milovanova TN, Bhopale VM, Sorokina EM, Moore JS, Hunt TK, Hauer-Jensen M, Velazquez OC, Thom SR. Hyperbaric oxygen stimulates vasculogenic stem cell growth and differentiation in vivo. J Appl Physiol (1985). 2009;106:711–28. doi: 10.1152/japplphysiol.91054.2008. PMID: 19023021. PMCID: PMC2644249.

Conflicts of interest and funding: nil

Submitted: 27 March 2024
Accepted after revision: 9 August 2024

Letter to the Editor

Hyperbaric medicine and climate footprint

The world is going through a vicious circle. The current climate crisis negatively impacts population health. And heath care contributes to aggravate the climate crisis. The 2019 Health Care Without Harm reports that the climate footprint of the healthcare sector represents 4.4% of global net emissions, i.e., the equivalent of 2 gigatons of carbon dioxide (CO_2) annually.[1]

Several healthcare fields have already taken actions. For example, anaesthesiology is already moving towards a more ecological vision of healthcare, and notably reduced or eliminated the most detrimental anaesthetic gases.[2] The hyperbaric medicine specialty has yet to embrace this movement. In this letter, we describe the process and results of an intervention aimed at reducing plastic waste at a hyperbaric medicine centre in a large university hospital. Through this simple and effective example, we would like to encourage each hyperbaric medicine centre to take part in reducing carbon emissions globally.

In our hyperbaric centre at the Geneva University Hospital, Switzerland, we used to distribute plastic bottles of water to each patient to help them equalise the pressure across their tympanic membranes during the compression phase of hyperbaric oxygen treatment (HBOT).

Before our initiative, approximately 5,300 bottles were distributed annually, i.e., one bottle per session and per patient (both urgent and elective patients). Based on the life cycle assessment (LCA),[3] the bottles represented 100 kg of plastic and the equivalent of 5 tons of CO_2 each year at our centre. This calculation was carried out by our institution's environmental experts and involved modelling the environmental impact of all material and energy flows throughout the product's life cycle. The extraction of raw materials or the use of recycled materials, the transformation, use and treatment of end-of-life waste are analysed, resulting in the carbon footprint. There are global databases that contain references for carbon data, allowing to estimate the carbon footprint of a given object.

Our intervention consisted of acquiring: (1) a single water cooler fountain for our hyperbaric medicine centre as an initial investment of 1,200 CHF, (2) 330 ml reusable metal bottles at 4 CHF each, and (3) 10 reusable plastic cups (virtually no cost because returnable). For elective indications, a dedicated metal bottle was given to each patient (kept for their HBOT series and given to them at the end as a goodie). For urgent indications, the reusable cups were used, then washed and used again for future patients, and their carbon footprint is minimal. Five thousand HBOT sessions corresponded to approximately 200 elective patients annually, which means 200 metal bottles per year. The carbon footprint of the metal water bottles for one year represents the equivalent of 80 kg of CO_2, compared with five tons of CO_2 for plastic bottles previously (i.e., divided by a factor 62). In addition, the cost of 200 metal bottles was 800 CHF, compared with 5,300 plastic bottles at a total cost of 1,620 CHF. The cost of the fountain is compensated in less than two years. In this example, the environmental and financial benefits are indisputable.

In conclusion, we showed that a simple intervention can effectively reduce waste and carbon printing in the field of hyperbaric medicine while being cost neutral. It's time for the hyperbaric and diving medicine sector to take responsibility for its ecological footprint and respond to the climate emergency, not only by treating those who are ill, injured or dying as a result of the climate crisis and its consequences, but also by introducing primary prevention and drastically reducing its own emissions.

References

1 Karliner J, Slotterback S, Boyd R, Ashby B, Steele K. The health care's climate footprint [Internet]. 2021. [cited 2024 March 14]. Available from: https://healthcareclimateaction.org/sites/default/files/2021-11/French_HealthCaresClimateFootprint_091619_web.pdf.

2 McGain F, Muret J, Lawson C, Sherman JD. Environmental sustainability in anaesthesia and critical care. Br J Anaesth. 2020;125:680–92. doi: 10.1016/j.bja.2020.06.055. PMID: 32798068. PMCID: PMC7421303.

3 The sustainable development notebooks. Analysing the life cycle of a product or service [Internet]. 2024. [cited 2024 Mar 14]. Available from: http://les.cahiers-developpement-durable.be/outils/analyse-du-cycle-de-vie/.

doi: 10.28920/dhm54.3.252. PMID: 39288934.

Alice Varichon[1], Rodrigue Pignel[1], Sylvain Boet[1,2]

[1] Diving and hyperbaric Unit, Division of Emergency Medicine, Department of Anesthesiology, Clinical Pharmacology, Intensive Care and Emergency Medicine. Geneva University Hospitals and Faculty of Medicine, University of Geneva, Geneva, Switzerland
[2] Hyperbaric Medicine Unit, Department of Anesthesiology and Pain Medicine, Ottawa Hospital Research Institute, The Ottawa Hospital, University of Ottawa, Ottawa, ON, Canada

***Corresponding author:** Dr Sylvain Boet, Diving and hyperbaric Unit, Division of Emergency, Geneva University Hospital, 4 Gabrielle-Perret-Gentil Street, 1205 Geneva, Switzerland*
sylvain.boet@hug.ch

Submitted: 5 July 2024
Accepted: 14 July 2024

Keywords
Climate footprint; Ecology; Hyperbaric oxygen; Hyperbaric oxygen treatment; Life cycle assessment

Errata

Elliott EJ, Price K, Peters B. Formulating policies and procedures for managing diving related deaths: a whole of state engagement from frontline and hospital services in Tasmania. Diving and Hyperbaric Medicine. 2024 30 June;54(2):86−91. doi: 10.28920/dhm54.2.86-91. PMID: 38870949.

The authors have requested an update be made to the **Acknowledgements** statement in their article.

The **Acknowledgements** should read:

The authors would like to thank Senior Constable Scott Williams, Dr Chris Lawrence, Dr Andrew Reid, and Dr John Lippmann. The authors would also like to acknowledge and thank the support from the Tasmanian frontline agency representatives, and representatives from the Royal Hobart Hospital, Launceston General Hospital, North West Regional Hospital, Mersey Hospital, and Ochre Medical Group.

doi: 10.28920/dhm54.3.253. PMID: 39476422.

Advertising in DHM

Commercial advertising is welcomed within the pages of *Diving and Hyperbaric Medicine*. Companies and organisations within the diving, hyperbaric medicine and wound-care communities who might wish to advertise their equipment and services are welcome. The advertising policy of the parent societies – EUBS and SPUMS is available for download on Diving and Hyperbaric Medicine website. Scan the QR code above for more information.

Further information:
Can be obtained by contacting our Editorial Manager, Nicky Telles.
editorialassist@dhmjournal.com

South Pacific Underwater Medicine Society

Notices and news

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

President's report

Neil Banham

I am very appreciative and proud to be able to announce the establishment of the Mike Bennett Scholarship, which has recently been created to honour our great friend and colleague Professor Mike Bennett AM. Dr Sue Pugh, Mike's wife, has bequeathed funds to create this scholarship to fund the successful applicant to attend a scientific meeting of relevance to diving and hyperbaric medicine. Further details regarding the scholarship are to be found in the 'Notices' section, as well as on the SPUMS website.

Thank you Sue for instigating this fantastic initiative.

The venue for our 53rd Annual Scientific Meeting (ASM) in 2025 has been confirmed as Bali, Indonesia. Diveplanit has again been contracted as our travel provider to assist our Bali ASM Convenors Xavier Vrijdag and Hanna van Waart. See information flyer on the next page.

Confirmed dates are 18–23 May 2025.

Registration is now open follow this link to register. South Pacific Underwater Medicine Society – SPUMS ASM 2025

Theme: '*Oxygen: Too little, too much or just right*'

Venue: Ramayana Candidasa, Bali, Indonesia.

Thanks Hanna and Xavier and to Simon Mallender and Deb Dickson-Smith from Diveplanit.

Workshops were held at our recent 52nd SPUMS ASM in Fiji to update the SPUMS and the United Kingdom Sports Diving Medical Committee (UKSDMC) Joint Position Statement (JPS) on persistent foramen ovale (PFO) and diving of 2015 and to develop one for return for diving (or not!) following an episode of Immersion Pulmonary Oedema (IPO), as well as diver information for both. Development of these JPS and diver education e-brochures are progressing, with a view to publication in *Diving and Hyperbaric Medicine* and on the SPUMS website, by the end of 2024. The Paediatric Diving Position Statement which was workshopped at our 51st ASM in Cairns is in the final stages of editing and should be available on the SPUMS website soon and then published in *Diving and Hyperbaric Medicine*. Thank you to Lizzie Elliot, the lead for this and to the group of SPUMS members assisting.

Consideration for future venues for the 2026 and 2027 SPUMS conferences were canvassed at the ASM. Popular options were Palau and the Maldives, but a decision is yet to be made. Please advise me regarding other suitable ideas for a venue and/or if you wish to convene. Any proposed venue should have a conference room seating at least 100 people and the capacity to cater for at least 60 divers at one time.

David Smart and I have updated the SPUMS ASM Convenor Manual such that the organising of future ASMs is much easier.

The ANZHMG Introductory Course in Diving and Hyperbaric Medicine will be next held 17–28 February 2025, again in Fremantle. The 2025 course is now fully subscribed, with a wait list. I strongly suggest that you register your interest if you are considering attending the course in 2026; dates will again be from mid to late February 2026 for two weeks. Link to information on this course.

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.

Just prior to the 2025 AGM, nominations for the position of SPUMS President-elect will be sought, with the position being decided at the Bali AGM. The incoming President-elect will have a year to 'learn the ropes' prior to the completion of my second 3-year term as President at the 2026 AGM. Please consider yourself for this.

Dr Neil Banham
President

SPUMS Facebook page
Find us at:
SPUMS on Facebook

Royal Australian Navy Medical Officers' Underwater Medicine Course

Dates: 14–25 October 2024 and 17–28 March 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*

HBOE
HBOEvidence

HBOEvidence

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

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

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: 17–28 February 2025
Venue: Hougoumont Hotel, Fremantle, Western Australia
Cost: AUD$3,200.00 (inclusive of GST) for two weeks

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

The course content includes:

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

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

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.

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 Prof 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.

Closing date: 31 December 2024

Dr Neil Banham MBBS, FACEM, DipDHM, ANZCA DipAdvDHM

SPUMS President

SPUMS
South Pacific Underwater Medicine Society

Email: president@spums.org.au
Website: https://www.spums.org.au

SPUMS Diploma in Diving and Hyperbaric Medicine

Requirements for candidates (May 2014)

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

- 1 be medically qualified, and remain a current financial member of the Society at least until they have completed all requirements of the Diploma;
- 2 supply evidence of satisfactory completion of an examined two-week full-time course in diving and hyperbaric medicine at an approved facility. The list of such approved facilities may be found on the SPUMS website;
- 3 have completed the equivalent (as determined by the Education Officer) of at least six months' full-time clinical training in an approved Hyperbaric Medicine Unit;
- 4 submit a written proposal for research in a relevant area of underwater or hyperbaric medicine, in a standard format, for approval before commencing the research project;
- 5 produce, to the satisfaction of the Academic Board, a written report on the approved research project, in the form of a scientific paper suitable for publication. Accompanying this report should be a request to be considered for the SPUMS Diploma and supporting documentation for 1–4 above.

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

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

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

Additional information – prospective approval of projects is required

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

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

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

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

Projects will be deemed to have lapsed if:

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

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

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

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

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

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

SPUMS 53rd Scientific Meeting

Registrations are now open

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

Dear Colleagues, at the time of writing this, the focus of the EUBS is on the upcoming congress of our distinguished society, which will take place in September 2024 in the city of Brest, France. This congress is more than just an excuse to enjoy Breton crêpes or gaze at lighthouses in the rain. It is held under the auspices of the University of Western Brittany, a center of excellence in underwater and hyperbaric science, where brilliant minds from across the globe have been trained. These students, coming from all over the world to study the mysteries of diving as part of the European PHYPODE project, once walked through the doors of this university and left with remarkable theses on a variety of subjects, ranging from the development of decompression-resistant rat strains to the benefits of enjoying Belgian chocolate before a dive. Brest has allowed these young researchers to become recognised experts, and now they are luminaries in their field.

I know some of you may hesitate to travel because of the environmental impact of such journeys. And you are right. The issue of our carbon footprint is weighing more and more on our consciences. From the exchanges between ancient Greek cities to the voyages of the Enlightenment across Europe, meetings between scholars have played a major role in the advancement of knowledge. This remains true in modern science: a fruitful exchange with a colleague halfway around the world can open up perspectives that years of solitary work may not achieve. The production of knowledge is most often a collective endeavor, where the density of exchanges and networks plays a crucial role. Moreover, attending scientific conferences also helps maintain knowledge and plays a significant role in the evaluation of scientific careers.

So, how can we resolve the dilemma of reducing our environmental impact while maintaining sufficient scientific exchange?

This question is being thoughtfully considered by many universities around the world. In the face of ecological challenges, it's common to prioritise actions as follows:[1]
1. Reduce non-essential uses / sobriety measures,
2. Decrease the carbon impact of necessary uses / efficiency measures,
3. Try to offset the negative impact / compensation measures.

So, to account for our carbon footprint while staying true to the spirit of in-person scientific meetings, allow me to suggest a few reflections applied to our field:

Sobriety measures

» Favor combining underwater and hyperbaric medicine conferences, as is already partly the case with joint EUBS and SPUMS meetings during TRICON conferences. In the future, it would be desirable to hold these joint conferences more frequently, involving other scientific societies such as the UHMS.
» Prioritise interpersonal exchanges during conferences, as these are the parts of the event that are the hardest to replace with virtual conferences.
» Develop virtual conferences. EUBS successfully organized two webinars during the COVID pandemic. Organising a webinar must be approached very differently than an in-person conference. Interaction is certainly possible, but it requires dedicated technical means. Webinars can be an excellent tool for ongoing training in our field.

Efficiency measures

» Opt for low-emission transportation methods.
  » Take the train when possible. Train journeys are not only more eco-friendly, but also a great opportunity to review your notes for your presentations. Of course, this requires some extra time and available connections.
  » Recently, certain airlines have started using planes that run on recycled cooking oil.

Compensation measures

» Universities or institutions could establish the possibility of offsetting the greenhouse gas emissions associated with travel.
» Allocate part of the budgets for emission compensation.
» Ensure the effectiveness and relevance of the compensation methods.

In total, this is primarily an individual effort that requires calculating one's carbon footprint to assess one's ecological impact. It seems pertinent to catalog conferences by theme to avoid duplication and eliminate unnecessary events. When possible, organise and participate in teleconferences. Finally, grouping trips by extending their duration and combining multiple objectives also seems like a good approach.

In short, dear colleagues, it is clear that attending the Brest congress in September 2024 is an unmissable adventure.

Between exciting scientific sessions, joyful reunions, and brilliant ideas for saving our planet while continuing to explore its depths, this congress promises to be unforgettable.

See you very soon.

Jean-Eric Blatteau
The President, EUBS

References

1 Bouchet F. Diminuer l'impact écologique de notre recherche scientifique. [cited 2024 Aug 29]. Availablefrom: http://perso.ens-lyon.fr/vincent.pilloni/Diminuer.pdf.

EUBS Notices and news

EUBS Member-at-Large elections

The EUBS Elections again held electronically, using the ElectionRunner software, to elect one Member-at-Large for the four-year period 2024–2028. There were three candidates: Dr Pedro Barata Coelho from Portugal, Dr Mihaela Ignatescu from the United Kingdom and Dr Anders Kjellberg from Sweden. The candidate who scored best was Anders Kjellberg, and he will take office as from our General Assembly on 20 September 2024.

We will be saying goodbye to our 2020 Member-at-Large, Dr Oscar Camacho, for having served faithfully on the ExCom for four years. We thank Oscar 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).

EUBS2024 Annual Scientific Meeting, Brest, France, from 16–20 September 2024

As this issue of *Diving and Hyperbaric Medicine* journal is published, we will have had the pleasure to unite again in Brest, from 16–20 September 2024. 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 48th Annual Scientific Meeting of EUBS will have been a great success.

Next year, the EUBS meeting will be in Helsinki, France, from 3–6 September 2025, please keep these dates free in your busy schedules. Also, a good idea would be to plan to have some days off before and after the conference to enjoy Finland, which is extra beautiful at that time of the year.

EUBS General Assembly

This is a formal invitation to participate in our EUBS annual General Assembly, which will take place during the EUBS Annual Scientific Meeting, on Friday 20 September from 11.00 to 12.00, in the main conference hall. The agenda will be customary to discuss all items relevant to the function of the Society, as discussed by ExCom during their ExCom meeting on Monday 16 September and will be posted on the information board. All EUBS members with voting rights are cordially invited.

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.

BHA ANNUAL SCIENTIFIC MEETING 2024

Hull, East Yorkshire, UK, 6-7 November 2024

The British Hyperbaric Association (BHA) is holding its 2-day Annual Conference in the Kingsley Suite, Canham Turner Building, University of Hull Cottingham Rd HU6 7RX, Wednesday 6 and Thursday 7 November 2024.

The theme **"Nitrogen Narcosis and Bubbles"** provides an opportunity for all involved in diving and diving medicine to acquire, maintain and develop their professional profile.

Each day has registered for 6 CPD points

The program is designed for a wide range of participants including: Occupational, Emergency Department, ICU and Basics Doctors, Prehospital Responders, Nurses, Paramedics, dive systems Life Support Technicians, persons with an interest in the Physiology and Medicine of Diving and importantly **Divers themselves**

Abstracts for oral presentation are welcome, applications should be 250 words or less and submitted to BHA.Conference@mimirmarine.com on or before 14 October 2024.

Registration & payment

https://www.BHA-conference2024.com
Credit / Debit Card / Paypal / by Phone

Attending 6th & 7th Nov:	£ 400
6th Nov only:	£ 200
7th Nov only:	£ 200
Nurses, Divers, Students Non-Medics (per day):	£ 150
Conference Dinner:	£ 70
Dinner Guests:	£ 50

Contact us on +44(0)1482 672 462
BHA.Conference@mimirmarine.com

The current program includes the following speakers:

DAY 1 – 6TH NOV

Towards Gas Narcosis Monitoring in Compressed Gas Diving. Dr Xavier Vrijdag. Department of Anesthesiology at the University of Auckland.

Nitrogen Narcosis, It's a Risky Business. Pauliina Ahti. Centre for Interdisciplinary Brain Research, Department of Psychology, University of Jyväskylä. Finland.

Nitrogen Narcosis, never any hangover, or? Mikael Gennser MD PhD. Division of Environmental Physiology. Royal Institute of Technology, Sweden.

Acclimatisation to Diving: A Systematic Review. Dr Jan Risberg. Norway

Fatality Reporting International Database Dr. Frauke Tillmans DAN USA Research Director

DAY 2 – 7TH NOV

History and Evaluation of Intra-vascular Bubble Detection and Scoring. Lesley Blogg. BSc (hons), PhD

Bubbles detected, or not, now what? Mikael Gennser MD PhD. Division of Environmental Physiology. Royal Institute of Technology, Sweden.

National Network Acute Interventional Neuroradiology Dr Paul Maliakal. Consultant in diagnostic & Interventional Neuroradiology.

National Network Treatment of Arterial Gas Embolism Dr Pieter Bothma Consultant in Anaesthesia and Intensive Care.

AGE from shallow depth escape training Dr Jan Risberg.

The Paediatric Patient. Jaqui Painter Hull Hyperbaric Unit

Conference Website and Registration: www.BHA-conference2024.com
Video hook-up is available to overseas delegates, contact: BHA.Conference@mimirmarine.com

Courses and meetings

Scott Haldane Foundation

As an institute dedicated to education in diving medicine, the Scott Haldane Foundation has organized more than 320 courses all over the world, over the past 32 years. SHF is targeting on an international audience with courses world wide.

Below the schedule of upcoming SHF-courses in the second half of 2024.

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

2024

16–23 November	31st in-depth course Diving Medicine "Hear, smell, feel" (level 2d) Watamu, Kenya
23–30 November	31st in-depth course Diving Medicine "Hear, smell, feel" (level 2d) Watamu, Kenya
On request	Internship HBOt (level 2d) NL/Belgium

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

DHM Journal Facebook

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

Diving and Hyperbaric Medicine Journal copyright statement 2024

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/

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

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

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

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?

DIVER EMERGENCY SERVICES PHONE NUMBERS

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

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

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

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

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

USA – DAN
+1-919-684-9111

JAPAN – DAN
+81-3-3812-4999 (Japan)

ADSF
AUSTRALASIAN DIVING SAFETY FOUNDATION

Scholarships for Diving Medical Training for Doctors

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

There are two categories of scholarships:

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

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