Diving and Hyperbaric Medicine

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World first: clinical ECMO in the hyperbaric chamber

Outcome after iatrogenic arterial gas embolism

Cardiac arrest and defibrillation during HBOT (two articles)

Pulmonary air-containing lesions in HBOT patients

Quality in hyperbaric medicine clinical trials

Perceptions of mouthpiece retainers in rebreather divers

DCI and in-water recompression in freediving (two articles)

Fluid loss during rebreather dives

HBOT for acute idiopathic sudden hearing loss

Critical flicker fusion frequency through a chamber porthole

Three case reports

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EDITORIAL OFFICE

PURPOSES OF THE SOCIETIES

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

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The Editor's offering

It's not often that any journal can report a consequential clinical world first, but courtesy of our colleagues in Melbourne, we are privileged to do so in this issue of DHM. Following a severe multi-trauma injury which, among many other things, required a pneumonectomy and institution of venovenous extracorporeal membrane oxygenation (ECMO), a normally fit and well young man developed a vaso-invasive fungal infection of the face with associated sepsis. This seemed on an inexorable fatal trajectory despite maximal debridement and anti-fungal treatment. Based on previous positive case reporting in mucormycosis, hyperbaric oxygen treatment (HBOT) was considered a potential adjunctive treatment-of-last-resort. However, there were no prior reports of clinical ECMO in a hyperbaric environment. In the space of 36 hours, a multidisciplinary Alfred Hospital team overcame the ethical, administrative, technical and medical challenges associated with providing HBOT to a critically ill patient on ECMO and began treatment. Within just a few treatments the infection appeared to be regressing, and after 13 the infection appeared eliminated and the massively debrided facial wound had established healthy granulation. The patient was eventually discharged home.

The three associated papers published here (respectively describing the case; the process, logistics and governance issues; and the ECMO circuit itself) are important for two reasons. First, though the authors are appropriately cautious in drawing conclusions about efficacy on the basis of a case report, it is undeniable that there was a compelling temporal relationship between institution of HBOT and the reversal of a seemingly hopeless pattern of deterioration. The case reminds us that refractory, life-threatening fungal infections may respond to HBOT. Second, the authors have demonstrated that for a suitable indication and under the right circumstances, ECMO can be run in a hyperbaric chamber. They provide a template for the considerations and processes required to achieve this.

To complement these ground breaking papers, in this issue of the journal we have a range of high-quality articles across a range of subjects. Raoul Fakkert and our colleagues from the Netherlands report outcomes after iatrogenic arterial gas embolism. It is clear that even with appropriate treatment, there is considerable associated morbidity. There are two papers relating to defibrillation in hyperbaric environments: Sophia Nohl and colleagues review the global literature on this subject, and Anja Beilharz and colleagues review cases and protocols in Australian and New Zealand hyperbaric units. Osman Turkmen and colleagues assess the risks associated with pulmonary gas containing lesions during HBOT, and discuss justification (or not) for screening strategies. Yeonjung Yoo and colleagues audit the quality of reporting in randomised trials and observational studies in our field against established standards. It seems clear there is room for improvement there. Emmanuel Gouin and colleagues extend their fascinating series of studies into technical diving practices with a survey on use of and related beliefs around mouthpiece retaining devices by rebreather divers. It remains a mystery to me why divers have been slow to adopt this strategy whose net effect is (almost certainly) to increase safety, and the Gouin study sheds some light on this. Two papers by the UCSD / Stanford group address decompression illness events in freedivers and the use of in-water recompression in its treatment. These are issues of increasing importance as the 'freediving renaissance' continues. Laura Tuominen and colleagues report on fluid / weight loss over the course of cold-water rebreather dives, and found no correlation between fluid loss and post-dive venous gas emboli production. Annemarie Newth and colleagues provide an up-to-date systematic review and meta-analysis of HBOT in treatment of idiopathic sudden sensorineural hearing loss. There are three case reports relating to HBOT as delayed salvage treatment for post-traumatic sensorineural hearing loss, cutaneous decompress sickness in a commercial diver with persistent foramen ovale despite breathing oxygen during a surface decompression procedure, and HBOT as delayed treatment of persisting hypoglycaemic encephalopathy.

This issue also sees the first iteration of an "*images in diving and hyperbaric medicine*" segment which I hope will become a regular feature in the journal. This was the brainchild of EUBS immediate past-president Jean-Eric Blatteau who has provided the first. We are looking for interesting images around which a multiple-choice question (on a topic like diagnosis or management) can be constructed with a supporting narrative that poses the question, then provides an answer with explanation. These need to be short (preferably less than 500 words) with up to five supporting references. See Professor Blatteau's contribution in this issue.

This issue brings 2025 to a close for DHM. We have had a record number of submissions this year; more than the upward blip we saw during COVID. This certainly tests our production capacity, but the journal is in good health! I would like to thank Lesley Blogg for her work as deputy editor, and the societal excoms and the Journal Governance Committee for their ongoing support. Particular thanks to Dr Stephan Roehr, SPUMS Treasurer, for his work as the de facto journal accountant. Finally, as always, my sincere gratitude to Nicky Telles for her extremely hard work, efficiency and accuracy as editorial manager. It just would not work without her.

Professor Simon J Mitchell
Editor, Diving and Hyperbaric Medicine

Cover photo: The case described by Devaney et al. in the present issue during management in the Alfred Hospital hyperbaric unit, Melbourne Australia. Photo courtesy of Dr Bridget Devaney.

Featured articles

Novel use of hyperbaric oxygen treatment for treatment-resistant disseminated *Saksenaea* and *Fusarium* in a patient on extracorporeal membrane oxygenation (ECMO): a case report

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Keywords
Case reports; Infection; Mucormycosis; Vaso-invasive fungal disease; Trauma

Abstract

(Devaney B, Mathew J, Ferris S, Roberts L, Covelli C, Orosz J, Nanjayya VB, Fuller A, Millar I. Novel use of hyperbaric oxygen treatment for treatment-resistant disseminated *Saksenaea* and *Fusarium* in a patient on extracorporeal membrane oxygenation (ECMO): a case report. Diving and Hyperbaric Medicine. 2025 December 20;55(4):309–314. doi: 10.28920/dhm55.4.309-314. PMID: 41364853.)

Filamentous soil moulds such as *Saksenaea* and *Fusarium* are angioinvasive fungi responsible for severe disseminated infections. *Saksenaea* causes zygomycosis, with disseminated cases having over ninety percent mortality. *Fusarium*, a hyphomycetes mould, can also cause disseminated infections in immunocompromised individuals, with high mortality. We describe the case of a normally healthy 20-year-old male who survived major traumatic injuries resulting from an aviation incident. He subsequently developed disseminated cutaneous zygomycetes *(Saksenaea)* and *Fusarium* infection with associated immunosuppression, multiorgan dysfunction and sepsis. Treatment strategies included repeated and extensive surgical debridement (inferior orbital region to carotid sheath in the neck, to a depth of buccal mucosa and zygomatic bone in the cheek), antifungal agents including intravenous (IV) liposomal amphotericin B and voriconazole, and IV immunoglobulin and granulocyte colony stimulating factor. Despite maximal medical and surgical treatment, disease control was not achieved. After multi-specialty consensus that current management had failed to control the disease process, hyperbaric oxygen treatment (HBOT) was added to standard therapy on an experimental basis based on several case reports, pathophysiological rationale, and institutional experience with angioinvasive *Mucor*. The patient was on venovenous extracorporeal membrane oxygenation for all HBOT sessions; details are reported separately. Thirteen treatment sessions (243 kPa [2.4 atmospheres absolute], 95 min) were successfully delivered. Local and systemic disease control was achieved within several days of commencing HBOT, and after a prolonged period of rehabilitation and reconstruction, the patient was discharged home. We conclude that HBOT may have an important role in the management of angioinvasive fungal disease.

Introduction

Angioinvasive mould infections usually occur in immunosuppressed patients, or after major trauma. Filamentous hyphae invade and obstruct small blood vessels leading to downstream tissue hypoxia, lactic acidosis and tissue death. Hyperbaric oxygen treatment (HBOT) has been postulated to improve survival primarily via restoration

of normoxia to hypoxic tissues; restoring host immunity, improving viability of marginal tissues and penetration of antifungal therapies, and by stimulating the bone marrow.[1]

We report a case of severe refractory fungal infection with an extremely poor prognosis in which infection control was associated with undertaking HBOT.

Case report

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

We present the case of a healthy 20-year-old male non-Australian citizen who was involved in a military aviation incident in dense tropical bushland in the Northern Territory of Australia. His initial vital signs after being extricated from the immediate vicinity of the aircraft were GCS 14–15, heart rate 140·min^{-1}, systolic blood pressure 85 mmHg. The patient developed a left sided tension pneumothorax and respiratory arrest, for which bilateral finger thoracostomies and intercostal drain insertion were performed at the scene. He was intubated at scene and received eight units of packed red blood cells enroute to the receiving hospital. The pre-hospital time was three hours.

The patient's injuries included grade II liver laceration, splenic hilar injury, splenic flexure retroperitoneal haematoma, pancreatic tail bruising, left lung laceration, multiple spinal fractures (C7 vertebral body, T2-4 superior endplate, T8-10, L1-3 left transverse process, left S3 body and sacral ala), left acetabular fracture, bilateral inferior scapula fractures, 9th–11th left posterior rib fractures, right medial epicondylar avulsion fracture, small subarachnoid haemorrhage, left posterior thigh and gluteal haematoma and penile shaft and scrotal bruising.

In the operating theatre at the receiving hospital, damage control resuscitation and surgery was undertaken including laparotomy, thoracotomy, splenectomy and liver packing. During the left lateral thoracotomy a left pneumonectomy was performed due to uncontrollable hilar bleeding. A brief period of pulseless electrical activity cardiac arrest occurred intra-operatively, requiring internal cardiac massage.

The patient remained severely hypoxic (SaO_2 80–85%) despite FiO_2 1.0, inhaled nitric oxide (20 ppm) and prone mechanical ventilation in the first 24 hours of admission. He was vasoplegic and required high doses of vasopressors (noradrenaline 140 mcg·min^{-1}, vasopressin 3 U·min^{-1}). The extracorporeal membrane oxygenation (ECMO) outreach and retrieval service from our institution was mobilised to the receiving trauma hospital and he was commenced on venovenous (VV) ECMO for ongoing refractory hypoxaemia. A patent foramen ovale was noted during cannulation. He was transferred over 3,000 km by air to our centre on VV ECMO. He remained severely vasoplegic and went on to develop the following complications of his injuries: right renal infarcts and anuric acute kidney injury (managed with continuous renal replacement therapy); cardiac contusion (peak troponin 3,600 ng·L^{-1}); left sided infected haemothorax with radiological evidence of tension and haemodynamic instability (requiring multiple relook thoracotomies for bleeding, washouts of infected collections in the left chest, and left pneumonectomy stump revision); embolic infarcts of the right middle and posterior cerebral arteries and posterior inferior cerebellar artery (with resolution on later imaging); digital ischaemia of his right thumb and left 2nd and 3rd digits (later resolved with minimal intervention); compartment syndrome of the anterior and lateral compartments of the left leg (requiring fasciotomy); rhabdomyolysis; right cephalic vein deep venous thrombosis (DVT) (treated with low dose heparin); and polymicrobial pneumonia of the remaining lung with *Stenotrophomonas maltophila, Burkholderia territorii*, *Burkholderia cepacia complex* and *Acinetobacter baumanii complex* (treated with meropenem and later cefiderocol and sulfamethoxazole-trimethoprim). The patient developed persistent inflammation, immunosuppression, and catabolism syndrome (PICS).[2]

A minor penetrating cheek wound had been noted and irrigated on arrival at our centre. On Day 10 after the initial injury, the surrounding area became erythematous, and the patient developed signs of sepsis and profound sepsis-related lymphopenia. He was taken to theatre for a wash-out of the wound. Biopsy results showed angioinvasive fungal infection, which raised suspicion for mucormycosis. He was found to have disseminated disease with distant infection of the left leg and chest wall.

Two angioinvasive fungal organisms were confirmed: *Fusarium neocosmosporiellum* and *Saksenaea vasiformis* both of which are filamentous soil moulds (Figures 1, 2). Two other soil moulds of uncertain significance, *Curvularia species* and *Pseudothielavia subhyaloderma,* were grown from a forearm wound and the chest wound respectively. The infection progressed locally and systemically. The patient was increasingly septic and underwent multiple, extensive, right facial debridements, including a right radical parotidectomy with complete facial nerve sacrifice (Figures 3, 4). Despite maximal surgical debridement, anti-fungal agents (intravenous liposomal amphotericin B and voriconazole), granulocyte colony stimulating factor (G-CSF) and intravenous (IV) immunoglobulin, fungal invasion continued to progress. Further surgical efforts were discontinued once debridement depth extended to buccal mucosa, to zygomatic, maxillary and mandibular bone in the cheek and fungal growth was found to have invaded the right carotid sheath, threatening the carotid artery. It should be noted that despite repeated up-titration of the voriconazole dose, serum levels remained sub-therapeutic due to sequestration within the ECMO circuit.

At this stage, a multidisciplinary team of US and Australian clinicians considered several treatment options, including

Figure 1
Parotid biopsy with possibly two types of fungi seen (broad hyphae – *Saksenaea*; narrower hyphae invading blood vessel wall – *Fusarium*)

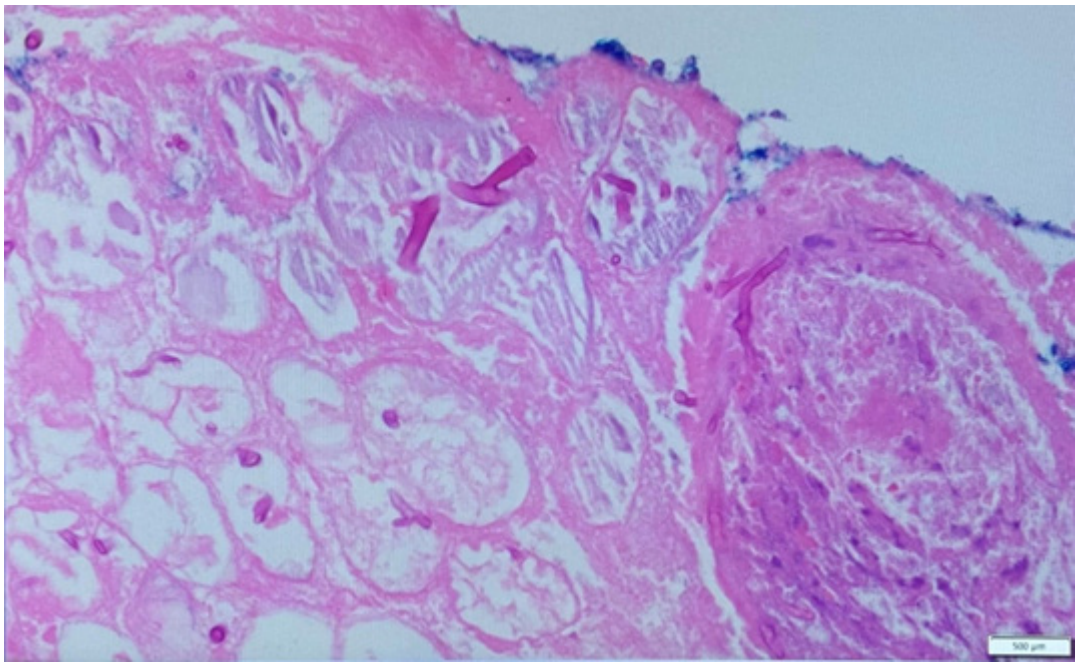
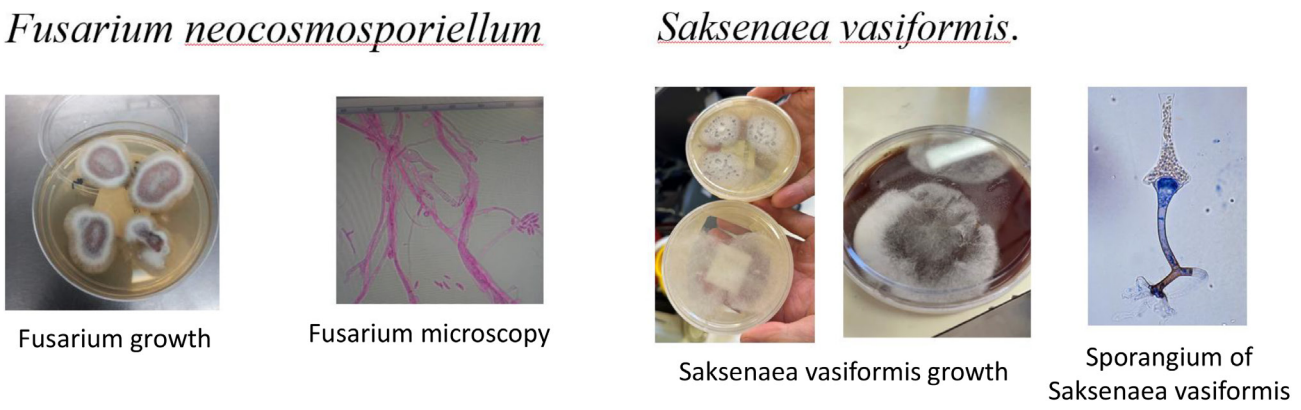


Figure 2
Microbiology plates showing fungal growth and microscopy



total facial debridement followed by a facial transplant, carotid ligation, and flap surgery to enhance circulation to the facial area. However, these options were ruled out after considering the patient's family's wishes, the significant disability, morbidity, and deformity associated with these procedures, and the patient's own preferences regarding quality of life as advised by his family. This decision prompted the exploration of experimental therapies. Based on attractive pathophysiological mechanisms, positive anecdotal and institutional experience with HBOT for advanced *Mucor*, and several published case reports, a decision was made to pursue HBOT as a 'last resort' experimental therapy. A further review in the operating theatre was performed prior to commencing HBOT, which demonstrated macroscopic fungal spread beyond the previous margins however this was not debrided (Figure 4).

A validation process had previously commenced for the use of ECMO in hyperbaric conditions, and is detailed elsewhere.[3,4] Importantly, the device was determined to be safe and to not pose a fire or safety risk to infrastructure or staff. After an expedited planning process, emergency ethics review and clinical innovations committee approval for the inaugural clinical use of ECMO in the hyperbaric chamber, HBOT was commenced daily and in total the patient received 13 treatment sessions over 15 days. Each treatment lasted 95 minutes at a treatment pressure of 243 kPa (2.4 atmospheres absolute). On surgical review after four hyperbaric treatments, the wound bed was noted to be sloughy with no frank mould, and on subsequent reviews the wound became clean and granulating (Figure 5). No significant further surgical debridement was required. Serum voriconazole

Figure 3
Clinical photography: first debridement once microbiology known; seven days prior to commencing hyperbaric oxygen treatment

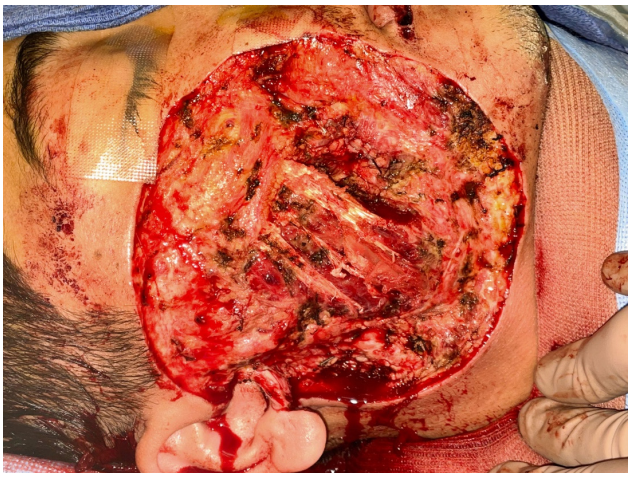


Figure 4
Clinical photography: day of commencement of hyperbaric oxygen treatment

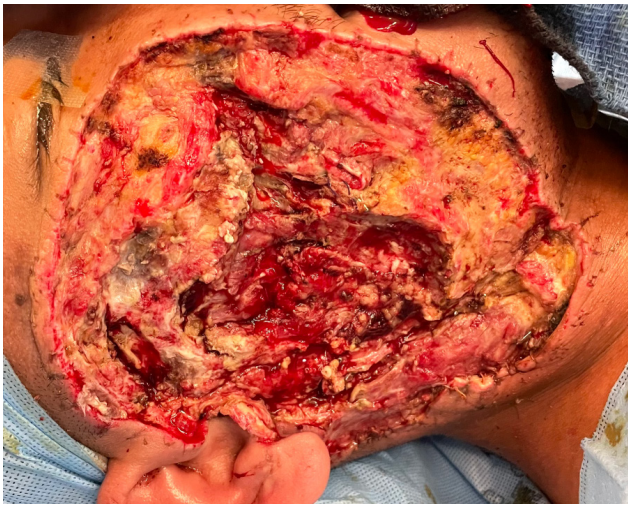


Figure 5
Clinical photography after completion of hyperbaric treatments

did not reach therapeutic levels prior to cessation of HBOT. The patient's lymphopenia resolved and was replaced by leucocytosis coinciding with reduced requirement for vasopressor support. Due to recurrent mucous plugging of the bronchus to the single remaining lung, the patient was kept on VV ECMO as a safety precaution whilst he was subsequently repatriated to his home country.

After an extensive hospital stay complicated by low-grade empyema, and profound weight loss requiring significant physical therapy and nutritional supports, the patient was decannulated from VV ECMO after approximately three months. Wound cultures and biopsies were negative for fungal disease, and his facial wounds healed with grafting. He was discharged home to the community.

Discussion

Within four days of commencing HBOT for disseminated *Saksenaea* and *Fusarium* in this patient, the previously extensive and progressive fungal growth (which was not able to be acceptably debrided and was left in-situ during the 'final' re-look surgery) had macroscopically resolved. This corresponded with clinical improvement and reduced requirement for haemodynamic support. Complete resolution of fungal disease was later demonstrated by negative wound swab and biopsy cultures.

As the patient was on ECMO when he developed angioinvasive fungal disease, and ECMO had not previously been validated for hyperbaric use, HBOT was only considered when consensus from all treating teams was that current treatment had failed to gain disease control. In this context, the resolution of fungal invasion within days of commencing HBOT makes a convincing case for the role HBOT played in controlling the disease process. Additionally, resolution of fungal invasion in the absence of therapeutic serum voriconazole levels, further underscores the potential significance of the role of HBOT.

The pathogenesis of zygomycosis involves rapid angioinvasion with resultant thrombus formation leading to localised tissue hypoxia and subsequent ischaemia.[5,6] Tissue necrosis limits the ability of the host immune system to deliver leucocytes and anti-fungal agents to the foci of infection and reduces the oxygen carrying capacity of infected tissues. Phagocytic leucocyte function is highly dependent on adequate oxygenation to tissues as leucocytes rely on oxidative radicals to kill invading pathogens and the rate of radical production is directly related to the oxygen tension of the local environment. Hypoxic

(below 30 mmHg) or anoxic environments result in loss of function and killing ability of the leucocytes.[7] Angioinvasive properties of these organisms allow dissemination to other organs. Disseminated disease, such as described in this report, has been reported to have mortality rates approaching 96%.[5,8]

Current mainstays of treatment for zygomycosis include urgent and aggressive surgical debridement and concomitant medical therapy.[9] The combination of surgical management and anti-fungal therapy has been associated with decreased mortality rates. In a review of 693 cases of cutaneous mucormycosis (of which only 13% had disseminated disease), Skiada et al., found that combined therapy had a lower mortality rate (29.6%) compared to medical management alone (36%) or surgical management alone (46.2%).[10] However, mortality remains high, necessitating the introduction of adjuvant therapies. There is limited high quality data to demonstrate the efficacy of HBOT but several case reports and case series describe positive outcomes amongst patients who received HBOT.[11–15]

There are several mechanisms by which HBOT may exert beneficial effects in the management of zygomycosis. HBOT involves the patient breathing 100% oxygen in a pressurised hyperbaric chamber. Treatment pressures are generally 203–304 kPa (2–3 atmospheres absolute); assuming normal gas transfer at the lungs this allows the partial pressure of oxygen in the arterial blood to reach up to ~2,000 mmHg. By accentuating the oxygen gradient between blood and tissue, the diffusion distance of oxygen is increased, and tissue hypoxia can be reversed. Reversal of tissue hypoxia and therefore acidosis, helps maintain local tissue survival.[7,16] Correcting acidosis within the tissues has the added benefit in zygomycoses of potentiating the oxidative action of Amphotericin B.[1,11,17] Periods of restoration of normoxia help combat the thrombotic and ischaemic effects of angioinvasive fungal infections, likely significantly boosting the anti-microbial capacity of leucocytes in the region of the infection.[1,17] Hyperbaric hyperoxia also improves tissue healing by restoring normal fibroblast function, increasing collagen deposition, promoting the secretion of inflammatory cytokines and, after repeated exposure, promoting angiogenesis.[1,18] Hyperoxia has been shown to have direct fungicidal effects on Zygomycetes, with *in-vitro* testing demonstrating complete inhibition of Zygomycetes growth at 24- and 72-h when exposed to 100% O_2 at 203 or 304 kPa (2 or 3 atmospheres absolute).[16]

This patient had two different moulds grown from his tissue samples: *Saksenaea vasiformis,* a Zygomycetes *and Fusarium.* Zygomycotic infections are a life-threatening group of diseases caused by moulds belonging to the class Zygomycetes and orders Mucorales and Entomophtorales.[6,19] The infections may occur in cutaneous, rhino-orbital-cerebral, pulmonary, gastrointestinal, renal or uncommon sites, each with associated risk factors and complications.[8] This report describes the use of HBOT in the management of disseminated skin and soft tissue mucormycosis and *Fusarium* infection resistant to standard management.

Fusarium exhibits resistance to a number of azoles and echinocandins but remains susceptible to natamycin, amphotericin B, voriconazole and posaconazole.[20] In resistant cases, a combination of antifungal drugs should be considered as this increases the efficacy and spectrum of action.[20] Surgical debridement and granulocyte colony stimulating factor have been used in the management of *Fusarium* with resection of all infected tissues recommended.[20] From the searches conducted, there appear to be no reports on the use of HBOT for *Fusarium*, however we suspected that HBOT may play a similar role in the management of *Fusarium* as has been reported in the management of other zygomycosis.

Given the patient was unable to be weaned from VV ECMO prior to HBOT and that validation processes for the Rotaflow Series I ECMO console with PLS1 circuit (Maquet, Rastatt, Germany) and Quadrax-i oxygenator was still underway at our centre, express ethics, clinical innovations committee and executive approvals were sought before proceeding with treatment. The technical details relating to validation of the device, and the governance and workflow procedures utilised to safely deliver VV-ECMO in the hyperbaric chamber are detailed in separate manuscripts.[3,4]

Conclusions

We report a rare case of disseminated cutaneous Zygomycetes (*Saksenaea*) and *Fusarium* in a previously healthy young adult male with severe trauma and associated multiorgan dysfunction, sepsis and immunosuppression. This case indicates that HBOT may rapidly interrupt progression of angioinvasive fungal disease, even in advanced stages. We hypothesise that the primary mechanism involved is the restoration of normoxia (or even hyperoxia) in areas made hypoxic by angioinvasion of fungal hyphae, enabling resumption of oxygen-dependant phagocytic functions. We suggest that clinicians should consider the use of HBOT as an additional treatment modality for angioinvasive fungal diseases such as those caused by Zygomycetes (*Saksenaea*) and *Fusarium,* even, in the right setting, for those requiring VV-ECMO.

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The process, logistics and governance behind a high-stakes novel intervention: the use of extracorporeal membrane oxygenation (ECMO) in the hyperbaric chamber

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Hyperbaric oxygen; Intensive care medicine; Perfusion; Risk assessment; Risk management; Workflow

Abstract
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Introduction: A multi-trauma patient receiving extracorporeal membrane oxygenation (ECMO) developed severe disseminated vaso-invasive fungal disease. In the absence of any remaining treatment escalation options, hyperbaric oxygen treatment (HBOT) was considered as a last effort at gaining disease control. Previously, the use of modern ECMO devices had not been validated for hyperbaric use at our centre or, to the best of our knowledge, at any other centre around the world. We had, however, identified a potentially hyperbaric compatible ECMO device and had commenced a validation process. The aim of this report is to highlight the practical, operational and governance processes undertaken to safely provide HBOT utilising ECMO at short notice.
Methods: A detailed risk assessment, development of risk reduction strategies and workflows, emergency out-of-session ethics review, clinical innovations committee review, legal advice, executive approvals and informed consent were undertaken over a 32-hour period prior to commencing HBOT.
Results: We present the identified risks, governance approvals, workflow, staffing model, chamber layout and safety checklist utilised to successfully deliver thirteen HBOT sessions to a patient on venovenous (VV) ECMO.
Conclusions: Through an extensive and coordinated effort involving multiple specialties and disciplines at our service, we were able to safely deliver HBOT to a patient supported by VV ECMO.

Introduction

A 20-year-old male was involved in a Class A aviation incident in dense tropical bushland and sustained major traumatic injuries. Following a damage-control pneumonectomy he was unable to be adequately ventilated and was commenced on venovenous extracorporeal membrane oxygenation (VV ECMO) prior to transfer to our centre. Several weeks into his hospital stay he developed disseminated vaso-invasive fungal disease which failed to respond to maximal medical therapy and repeated, extensive, debridement. In the absence of any remaining treatment escalation options hyperbaric oxygen treatment (HBOT) was considered in a critical-care capable hyperbaric facility, as an essentially experimental treatment. The rationale included attractive patho-physiological mechanisms, and previous published and unpublished reports suggesting a potential benefit of HBOT in the treatment of vaso-invasive fungal disease.[1–5] At this time ECMO had not been validated for in-chamber use in our centre or to the best of our knowledge in any other centre worldwide, however a validation process was underway at our centre.[6]

The aim of this report is to highlight the practical, operational and governance processes that were undertaken to safely provide HBOT as a last effort to a patient dependent on VV ECMO using the Maquet (Getinge) original series Rotaflow (Rotaflow 1) at short notice. The technical report and clinical components of the case are beyond the scope of this report and are detailed elsewhere.[6,7]

Methods

A series of inter-dependent working groups were urgently convened to work together and in parallel to navigate the complexities of safely delivering ECMO in the hyperbaric environment. The aims of the working groups were to: i) configure and perform additional testing of the Rotaflow 1; ii) establish workflows, logistics, staffing and contingency plans; iii) identify risks and risk reduction measures; and iv) navigate the clinical governance and consent requirements for a novel and experimental intervention.

The working groups consisted of senior hyperbaric, intensive care and ECMO clinicians (medical and nursing), and biomedical engineers. A scribe role was allocated.

RISK ASSESSMENT

Equipment safety

As part of a longer-term project to develop ECMO capability in the hyperbaric chamber, a Rotaflow 1 console had previously been reconfigured to run without a battery on a dedicated and highly redundant medical power supply installed in the hyperbaric chamber, with preliminary testing performed on a circuit primed with saline.[6] However, at the time this patient was considered for HBOT we had not yet validated the use of ECMO with a circuit primed with blood or applied *in vivo* conditions. An assessment of risks was undertaken (Table 1), and risk reduction strategies are described in Results.

Table 1
Summary of key risks assessed prior to delivery of ECMO in the hyperbaric environment

Equipment
Fire safety
Device integrity under pressure
Device performance under pressure
Patient
Injury or death relating to equipment failure or malfunction
Oxygen toxicity
Barotrauma
Staff
Staff decompression injury in event of an emergency decompression procedure
Well-being
Reputational
Legal
Organisation
Organisational risk
Opportunity cost impact

Staff and patient safety

A customised HBOT treatment table was developed to maximise potential therapeutic benefit whilst ensuring that the inside attendants did not acquire a decompression obligation. Attendants could therefore be rapidly decompressed at any point in an emergency. Consideration was given to the unknowns around oxygen toxicity in the setting of oxygen delivery via both a ventilator and the ECMO circuit.

Emergency scenarios

Emergency management plans specific to this patient on VV ECMO in the hyperbaric chamber were developed for the following scenarios: pulmonary barotrauma, air embolism, loss of primary and secondary gas supply, cardiac arrest, ECMO pump or circuit failure.

CLINICAL GOVERNANCE APPROVALS

Given the novel and untested nature of delivering HBOT to a patient on ECMO, it was considered that ethics committee review, clinical innovations committee review and executive approvals were required. At our centre, the ethics committee is primarily responsible for research governance but also can be called to provide advice to management on case specific dilemmas. The clinical innovations committee includes a wide range of senior clinicians, technical and management personnel who normally conduct scheduled considerations of the appropriateness of technical, equipment and process innovations proposed for introduction at our centre. A briefing document was prepared for these committees and emergency 'out of session' meetings were scheduled with hyperbaric and intensive care leadership input. Approvals and consent were sought on the proviso that all parties were willing to accept that in addition to identified risks, there were potentially unidentified and unquantifiable risks to the patient.

NEXT OF KIN CONSENT

The patient was unconscious throughout the planning, discussions and approvals processes, and therefore unable to provide consent. When a clear strategy was established to deliver treatment in the safest possible fashion and organisational approvals had been granted, a discussion was held with his next of kin. The medical treatment decision maker was provided with written information about the proposed treatment and a comprehensive informed consent process was undertaken.

LOGISTICS

Staffing

A staffing model was developed to ensure maximal safety during this novel intervention, with specific consideration to the skill-mix and roles of staff inside and outside of the chamber.

Workflow

A workflow plan was established by the working group prior to initiation of HBOT. A specific hyperbaric-ECMO chamber checklist was developed for use alongside pre-existing hyperbaric checklists.

Patient and equipment positioning

Planning of patient and equipment positioning in the chamber was performed by hyperbaric nursing staff.

Briefing document

A team briefing document was prepared which detailed the clinical situation, planned intervention, workflows, emergency procedures, administrative tasks, roles and responsibilities.

Results

ADDRESSING THE IDENTIFIED RISKS

Equipment safety

The Rotaflow 1 was deemed safe from a fire-safety perspective in the hyperbaric environment and had performed consistently with a circuit primed with saline during preliminary testing.[6] Additional technical tests did not demonstrate any macroscopic bubbles visually, microscopic bubbles via ultrasonic detection methods, or any leaks in the system.[6]

ECMO components used at our centre include a console, pump, patient circuit with oxygenator, gas supply, blender and blood warmer. During HBOT, the blood warmer and gas blender were excluded from the setup as they had not undergone testing. Traditional temperature management techniques were utilised including hyperbaric chamber heating and blankets. Oxygen was supplied directly to the oxygenator via flexible oxygen tubing from a standard oxygen flow meter connected to a medical oxygen outlet in the chamber, in line with ECMO transport practice at our institution.

Staff and patient safety

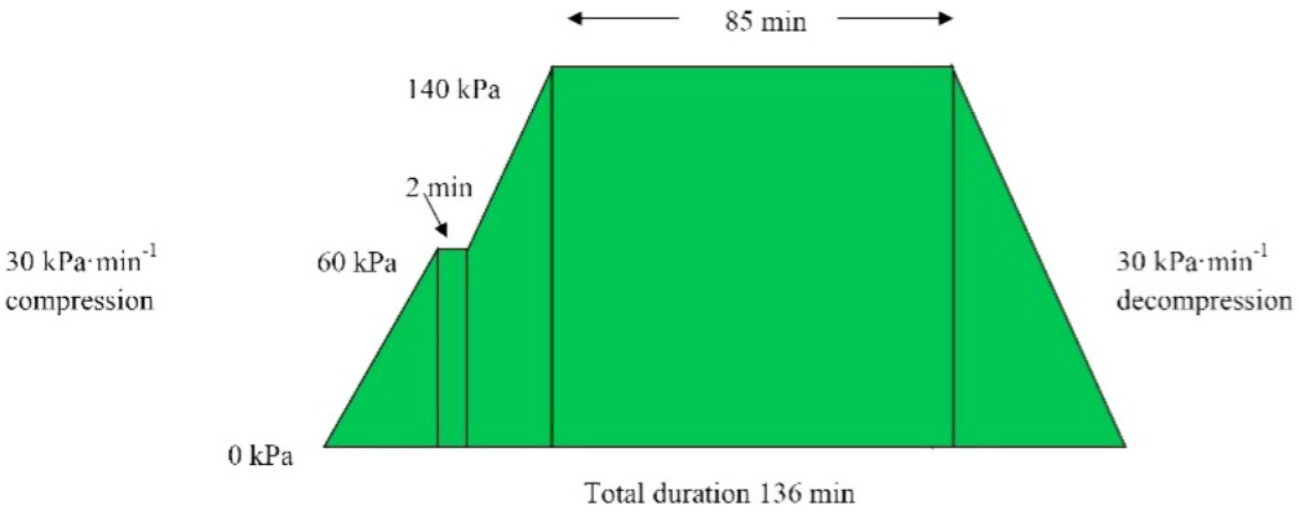
The HBOT treatment table (Figure 1) involved a pressurisation rate of 30 kPa·min^{-1} to a maximum pressure of 140 kPa (gauge pressure) (2.4 atmospheres absolute [atm abs]). A two-minute hold was introduced on reaching 60 kPa to allow for equipment checks and examine for bubbles in the circuit, and also for personnel checks and to ensure all key clinical parties inside and outside of the chamber, were comfortable and willing to proceed. The total hyperbaric oxygen delivery time at 140 kPa was 85 minutes. There were no air breaks and the patient remained on 100% FiO_2 via the ventilator (and ECMO oxygenator) for the duration of the treatment table. Attendants breathed 100% oxygen from five minutes prior to commencing decompression until completion of the treatment table. The chamber was decompressed at a rate of 30 kPa·min^{-1}.

Emergency scenarios

In addition to close observation for known complications of HBOT including hypercapnia, oxygen toxicity and hypoglycaemia, particular consideration was given to the critical scenarios and contingency plans described below.

Pulmonary barotrauma is a rare complication of exposure to the hyperbaric environment. The patient treated was at increased risk due to single lung ventilation complicated by acute respiratory distress syndrome and a friable bronchial

Figure 1
Hyperbaric ECMO treatment table; note that treatment pressures are gauge pressure



stump post-pneumonectomy.[8,9] The patient had mixed restrictive and obstructive ventilatory challenges, and a mixed ventilation strategy was employed using the Getinge Servo-i HBO Ventilator. Medical staff were equipped with a needle and finger thoracostomy kit inside the chamber for chest decompression if required.

In the event an air embolism was detected in the ECMO circuit, the circuit was to be immediately clamped and emergency decompression procedure commenced to facilitate a circuit change. The patient's intensive care unit (ICU) ECMO console was a Getinge Rotaflow II (Rotaflow 2); this remained on charge at all times with a spare primed permanent life support (PLS) circuit, immediately outside the hyperbaric chamber.

In the unlikely event of interruption to both primary and back-up oxygen supply systems, rapid decompression would occur and cylinder oxygen would be utilised.

Given the patient was supported with a VV ECMO configuration, there was no circulatory support in the event of a cardiac arrest. In this instance, the in-chamber nursing attendant would commence chest compressions and the hyperbaric team lead would coordinate a rapid decompression and initiate an emergency (Code Blue) response. After chamber decompression, the patient would be transported down to a resuscitation area outside the chamber for ongoing advanced life support and defibrillation if required, with ECMO blood flow maintained by utilising the hand-crank.

In the event of an ECMO pump failure, the inside hyperbaric ECMO clinician would transfer the pump-head onto the back-up hand crank and restore flow, the technicians would commence an emergency decompression and on return to 1 atm abs the extracorporeal life support (ECLS) team would exchange the console.

In the event of circuit failure, the circuit would be clamped, emergency chamber decompression would occur, followed by a rapid assessment and circuit change by the two ECMO clinicians in the hyperbaric resuscitation area.

GOVERNANCE APPROVALS

The final consensus from both the ethics and the clinical innovations committees was to endorse the recommendation to conduct hyperbaric oxygen treatment on this patient. Given the unique situation, the hospital legal team confirmed indemnity from the hospital's insurance provider relating to the provision of HBOT for this patient on ECMO. Hospital executive provided approval to proceed.

MEDICAL TREATMENT DECISION MAKER CONSENT

The medical treatment decision maker provided informed consent amidst the uncertainty of this novel delivery of HBOT. This decision was made in the interest of the patient, who, it was felt, would have wanted to proceed with HBOT despite the uncertainty. It was also believed that he would have liked to be a part of validating the safe delivery of HBOT with ECMO for the benefit of other patients in the future.

STAFFING

The provision of hyperbaric treatment to a patient on ECMO required a large collaborative effort among senior ECMO and hyperbaric physicians, clinical nurse specialists and biomedical engineers.

Inside the chamber two models of staffing were utilised:
1) a critical care nurse qualified in both ECMO and hyperbaric nursing, and a senior critical care physician with both hyperbaric and ECMO credentialing; or
2) two critical care nurses qualified in both ECMO and hyperbaric nursing and a critical care hyperbaric physician.

All staff working inside the chamber were required to have current certificates of fitness to work in a compressed air environment.

Outside the chamber, the team consisted of a dedicated team leader, the chamber operator, a hyperbaric nurse, an ECMO clinical nurse consultant, two ECMO physicians, the ICU bedside nurse, and the operations manager.

A dedicated team leader role was seen as critical to manage any interactions between the clinical team and external parties and to provide overall coordination and monitoring of clinical processes and leadership of emergency response should that have been needed, noting the large number of personnel and roles involved. This role was fulfilled by the (outside) senior hyperbaric physician.

WORKFLOW

Preparation to commence HBOT

The first treatment session commenced 32 hours after the initial clinical determination to pursue HBOT. The patient had middle ear tympanostomy tubes inserted to reduce the risk of barotrauma. Prior to the first HBOT, the HLS ECMO circuit was changed to a PLS circuit compatible with the Rotaflow 1 and Rotaflow 2 consoles and the Quadrox i-adult HMO 70000 (Quadrox) oxygenator. There was a right femoral multi-stage 25 Fr access cannula and a left femoral single-stage 21 Fr return cannula.

Pre-treatment

In ICU before each transfer to the hyperbaric chamber, the patient was prepared using a standardised intra-hospital transport checklist.

All involved teams (Hyperbaric, ECMO & ICU) gathered for a briefing one hour prior to each planned HBOT session. The structured brief prepared in the planning phase was used to ensure consistent delivery of all important information.

Prior to each treatment session, sedation was deepened to reduce the risk of awareness, and for the early treatments a continuous infusion of muscle relaxant was given to prevent ventilator desynchrony and ECMO access insufficiency (a state where the suction pressure at the access cannula is excessive in relation to the venous return). The patient's ECMO flow requirements and fluid state were also optimised to prevent access insufficiency.

Transport and arrival at chamber

The patient was transported on the Rotaflow 2 by the ECMO and ICU teams to the hyperbaric chamber, where a primary survey was completed upon arrival. Hyperbaric nurses performed a sequential changeover of ICU equipment in communication with the ECMO team and critical care hyperbaric physician team leader – the oxygenator to the chamber/wall source of oxygen, monitoring cables to the chamber monitor and endotracheal tube to the hyperbaric ventilator. Ventilator settings were confirmed, and the endotracheal tube cuff inflator was connected to the pilot balloon of the endotracheal tube.

Standard pre-treatment chamber checklists were completed, which include in-chamber safety equipment checks, attendants' oxygen mask checks, confirmation of the pressure fitness of attendants and medical emergency plans, a safety time-out, and patient safety checks.

Once standard intensive care hyperbaric checks were completed, the ECMO team commenced a swap of consoles onto the hyperbaric-modified Rotaflow 1 and the flow was zeroed. Patient vital signs were closely observed during this period. Once the console swap was complete, final pre-treatment attendant checks were completed and chamber pressurisation (HBOT) commenced.

During treatment

Once pressurisation began, the hyperbaric ECMO physician monitored the circuit both visually and using Doppler ultrasound for air bubbles. The hyperbaric nurse monitored the patient's ventilation and haemodynamic parameters. During pressurisation, the chamber fan and chiller were left off for noise reduction and heat conservation. When the chamber reached the target pressure of 140 kPa (2.4 atm abs), the first arterial blood gas (ABG) was taken to assess the adequacy of patient ventilation, blood flow and fresh gas flow (FGF) to the oxygenator. Adjustments were made as required. The first of two samples were then taken for pre- and post-oxygenator blood gas analysis. A second arterial blood gas was taken later in the treatment.

Throughout the treatment, the patient was monitored with continuous five lead ECG, arterial blood pressure, oxygen saturation, waveform capnography and nasopharyngeal temperature monitoring. Pre- and post-oxygenator pressure transducers were not connected during treatment, as is standard practice during transport.

The inside attendants reassessed the patient prior to donning their own oxygen masks for decompression. Particular consideration was given to vasoactive medication adjustments that had occurred during the treatment, and a reduction in hyperbaric oxygen-mediated vasoconstriction during decompression was anticipated. The patient's ventilation status was monitored closely on decompression for signs of pneumothorax. Pressure bags were adjusted to avoid rupture of the normal saline fluid bag within.

Post-treatment

Once the treatment had concluded, a staged process was undertaken in the chamber to transfer the patient back onto ICU transport equipment and the Rotaflow 2 console. After a final patient assessment and a pre-return transport checklist were performed, handover of patient care was given and the ICU/ECLS teams transported the patient back to ICU. It was recommended to slowly wean the patient's FiO_2 via the ventilator (not via the ECMO blender) over 60 minutes after return to ICU, as per standard practice.

ECMO chamber checklist

Consistent with standard procedures for both hyperbaric and ECMO operations in our organisation, a hyperbaric ECMO checklist was developed (Table 2).[10] This checklist was further refined as experience was gained during the course of treatment. The checklist was deemed an important safeguard to mitigate human factor elements in a dynamic clinical environment supported by a large multidisciplinary team.

CHAMBER LAYOUT AND PATIENT POSITIONING

We elected not to alter the positioning of the ventilator, equipment trolley, or IV pole and medication infusion pumps from our standard critical care practice. The patient's bed was centrally located within the main lock (feet toward exit), and the ventilator was placed at the patient's left shoulder connected to oxygen, air, nitrogen and power outlets at a central utility station. The IV pole and infusion pumps were placed to the right side of the head of the bed, in proximity to the patient's central venous access device (Figure 2). The Rotaflow 1 console was placed in close proximity to the foot end of the bed and the power cable, fresh gas flow and nitrogen purge tubing were routed together via the chamber ceiling to maintain clear access to vital circuit components.

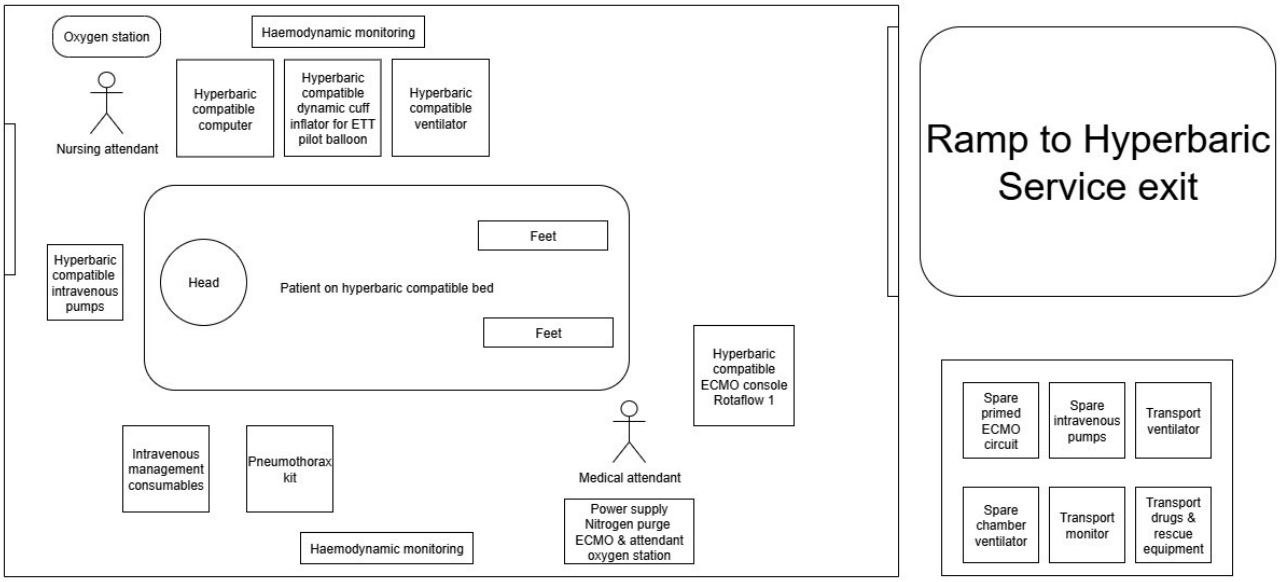
Table 2

Hyperbaric ECMO checklist; ECMO – extracorporeal membrane oxygenation; HBS – hyperbaric service; ICU – intensive care unit

Start-up procedure for hyperbaric ECMO device (device will not start unless connected to in-chamber power)
Confirm ECMO oxygenator connected to oxygen flowmeter and confirm oxygen set to fresh gas flow required – pre-pressurisation □ – at pressure □ – post decompression □
Confirm nitrogen purge is connected and ON (check with Technician)
Confirm compatible ECMO console and primed circuit are plugged in and available at bottom of chamber ramp
Measure ECMO cannula insertion length to ensure unchanged since leaving ICU
Confirm ECMO circuit and cannula secured with no visible bleeding
Confirm four ECMO clamps present
Confirm the following HAVE BEEN REMOVED from the ECMO trolley and chamber: – blood warmer – blender – all oxygen cylinders – all alcohol and skin adhesives
Visualise yellow cap is in-situ on ECMO oxygenator
Confirm hyperbaric ambient cooler is off and patient temperature probe is in-situ
Check ECMO console alarms are set

Figure 2

Layout of equipment in the main lock of the multiplace chamber for hyperbaric ECMO



Discussion

Prior to the development of cardiopulmonary bypass equipment suitable for small infants, cases of paediatric congenital heart disease underwent cardiothoracic surgery in the hyperbaric chamber and there are reports of early work in the 1960s to 1980s involving animals or patients undergoing HBOT whilst on cardiopulmonary bypass or ECMO.[11,12] A conference abstract describes a series from China including 48 children and adults who underwent cardiothoracic surgery

with extracorporeal circulation under hyperbaric conditions in 1984.[13] However, to the best of our knowledge, this case represents the first time that a patient has received HBOT whilst on ECMO, and the first documented use of the Rotaflow 1 console and Quadrox-i oxygenator under hyperbaric conditions. This paper is also the first instance where processes and workflow for safe delivery of HBOT with ECMO have been documented.

The patient had previously been on an ECMO console which was assessed as not suitable for hyperbaric use, and an initial circuit change was therefore required to facilitate HBOT. He was then transported each day from ICU on the Rotaflow 2 (of which the circuit is compatible with the original series Rotaflow), switched to the Rotaflow 1 on arrival in the hyperbaric unit, and back to Rotaflow 2 after hyperbaric treatment for transfer back to ICU. This was required because preliminary testing and power modifications had been made on the Rotaflow 1 device, and without battery power it was not suitable for use in transport.

Thirteen HBOT sessions were delivered for this patient over fifteen days, with a pause over the second weekend.

Skill-mix and inter-operability between hyperbaric, ECMO and intensive care teams were critical in ensuring the clinical capacity to deliver ECMO safely in the hyperbaric environment. Critically, the preliminary testing and modifications of the device would not have been achieved without dedicated hyperbaric biomedical engineers employed within our service. Technical details of this work are outside the scope of this manuscript and can be found in a corresponding technical paper.[6]

Strategic actions to mitigate risks and harm reduction in a realm of uncertainty were also key to delivering ECMO in the hyperbaric chamber. Non-technical skills were emphasised in the planning sessions and utilised throughout, including shared mental models, closed loop communication, clear task delineation and anticipation of problems in advance. A process-focused approach was used, with repeated evaluation of impacts and outcomes being identified through discussion and feedback. Consideration was given to the psychological safety of staff, and to the potential impact of an adverse event or adverse patient outcome. Risk of moral injury in such a scenario was considered and staff were given the opportunity to voice any concerns they had about the safety or appropriateness of proceeding with treatment.

Given this is a first-of-a-kind experience, in the world to our knowledge, we hope that these documented processes will help organisations worldwide develop the capability to safely deliver HBOT to a patient supported with ECMO. On a local level, these processes will be consolidated into a guideline should the need arise to provide HBOT to a patient on ECMO in the future.

Conclusions

The novel approach of providing HBOT to a patient supported with VV ECMO was complex and required a whole-of-system approach with meticulous planning. A collaborative approach in our organisation allowed the delivery of a considered plan incorporating risk assessments, mitigation strategies, workflows and checklists. Together with expedited organisational safeguards through the clinical innovations, ethics and executive committees, we delivered the world-first in-vivo HBOT course to a patient supported on VV ECMO. Having demonstrated the safe and successful use of the Rotaflow 1, we envisage developing the capacity to treat patients supported with ECMO with a round-the-clock service in the future. We hope the experience shared here will encourage the development of similar capabilities in other health services worldwide.

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Validation and clinical use of the Maquet (Getinge) original series Rotaflow extracorporeal membrane oxygenation device in hyperbaric conditions: a technical report

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Keywords
ECMO; Equipment; Hyperbaric oxygen treatment; Intensive care; Life support; Medical devices; Perfusion

Abstract
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Introduction: Extracorporeal membrane oxygenation (ECMO) has not been previously used clinically in the modern hyperbaric chamber. We describe the modifications, validation and clinical performance of the Maquet (Getinge) original series Rotaflow (Rotaflow 1), Quadrox-i adult microporous membrane oxygenator and permanent life support (PLS) circuit under hyperbaric conditions.
Methods: A Rotaflow 1 and Quadrox oxygenator underwent power supply modifications and rigorous safety testing in the hyperbaric environment using a PLS circuit primed with normal saline. Clinical validation was subsequently undertaken during a 'last resort' course of 13 hyperbaric oxygen treatment (HBOT) sessions for a patient suffering a life threatening vaso-invasive fungal infection requiring support with venovenous ECMO.
Results: Preliminary testing and subsequent clinical application in the hyperbaric chamber demonstrated steady flow through the circuit based on pump revolutions per minute, with up to 180 mL (10%) variability demonstrated between the console display compared to the independent flow meter. No significant changes to flow variability were noted during pressurisation and decompression phases. Device temperature remained within safe limits. No bubbles were visually or sonographically detected. There were no performance or integrity issues detected through compression, maintenance and decompression phases. During clinical use, the patient remained stable and hyperoxygenation targets were achieved. Membrane oxygenator oxygen inflow set at up to 8 $L \cdot min^{-1}$ maintained CO_2 clearance.
Conclusions: After safety related modifications to the console's power supply, the Rotaflow 1 console, Quadrox oxygenator and PLS circuit performed satisfactorily up to 243 kPa during repeated clinical use.

Introduction

Hyperbaric oxygen treatment (HBOT) is used in the treatment of arterial gas embolism, decompression sickness, necrotising soft tissue infections, late tissue radiation injury and complex wounds.[1] Treatment capability varies between hyperbaric centres, ranging from those which treat low-acuity ambulant patients only through to those that treat critically unwell intensive care patients requiring haemodynamic and ventilatory support. Our hyperbaric biomedical engineers have expertise in the validation of equipment for use under hyperbaric conditions, including validations of the HeartMate III left ventricular assist device, a pleural vacuum relief device for use with an underwater seal and various syringe drivers.[2–4]

Our hyperbaric centre regularly treats critically unwell patients.[5] Over recent years, a number of patients receiving extracorporeal membrane oxygenation (ECMO) at our centre have had a clinical indication for HBOT but were unable to undergo HBOT as ECMO had not been validated for use within the hyperbaric environment. Validation of ECMO for the hyperbaric environment would allow patients to access potentially life-saving HBOT whilst continuing to receive cardiopulmonary support.[1,6,7]

A patient in our intensive care unit (ICU) was critically ill following multi-trauma and was supported with veno-venous (VV) ECMO for severe respiratory failure due to acute respiratory distress syndrome in his remaining lung following a pneumonectomy for refractory haemothorax. Complicating the patient's burden of traumatic injury was a disseminated cutaneous filamentous fungal infection refractory to maximal medical and surgical therapies. In the absence of other treatment escalation options, HBOT was considered as a final line of therapy.[8] The challenge in delivering HBOT was the ability to continue VV ECMO support within the hyperbaric environment. Fortuitously, biomedical adaptation of the power supply of the Maquet original series Rotaflow (Getinge AB, Göteborg, Sweden) console (Rotaflow 1) to ensure electrical safety had previously occurred, as well as testing of the console in the hyperbaric environment using a Permanent Life Support (PLS) Set circuit (Maquet, Rastatt, Germany) primed with normal saline (Figure 1). Ethics approval was pending to complete further testing under hyperbaric conditions while using blood in the circuit. No patient trials with this device had ever been conducted in hyperbaric conditions at our centre or reported in the literature.

Methods

HYPERBARIC SAFETY ASSESSMENT (*IN-VITRO*) AND MODIFICATIONS

Equipment validation for use in the hyperbaric environment is performed by experienced hyperbaric biomedical engineers at our centre. The testing matrix includes an assessment of general safety, oxygen safety and device performance, and is outlined in Table 1.

Biomedical engineering, technical, medical and nursing staff undertook a preliminary risk assessment for the ECMO console and circuit and identified several critical areas requiring focused attention to ensure patient safety and system reliability: equipment malfunction such as pump failure, battery run-time, clotting of the Quadrox-i adult microporous membrane oxygenator (specifically HMO 70000, Maquet Cardiopulmonary AG, Hirrlingen, Germany) (Quadrox oxygenator) and the potential for components such as relays, motors or actuators to be ignition sources. Mitigation strategies identified for risks determined to be particularly important for ECMO devices in the hyperbaric environment included: physical inspection at component level, formal temperature testing of components identified on

Figure 1
PLS System; Rotaflow console, drive and permanent life support (PLS) set. Image source: https://www.getinge.com/int/products/pls-set/

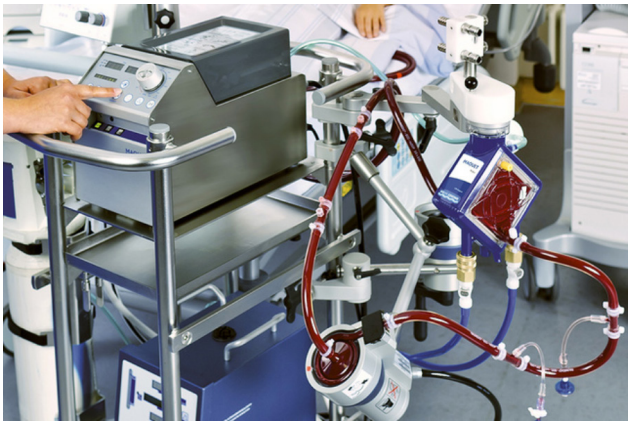


Table 1
Key components of hyperbaric equipment safety assessment and validation

General
Checking for safety of use if modified within and outside the hyperbaric chamber
Checking for safety of use at the maximum chamber pressure
Check for any damage or concerns with maximum rates of pressurisation and depressurisation
Ability to clean parts safely and reprocessing of consumables
Evaluation of air spaces for venting or fluid replacement requirements
A multidisciplinary consultation process between technical staff, biomedical engineering, medical and nursing representation
Oxygen Safety
Identification of energy sources, battery types, electrical connection security
High temperature components
Motor/s configuration
Mechanical and electrical relays
Safety of use within different oxygen concentrations
Testing
Preliminary discussion and define scope
Device documentation and literature review
Internal inspection
Device modifications
Device performance testing
Preliminary tests 1–6
Operational tests 1–4
Additional tests 1–3
Calibration test
Device operational needs
Accept device
Approval and sign off

preliminary infra-red temperature screening, identification of battery chemistry types and run-time and routine preventive maintenance.

General safety

The Rotaflow 1 contains a nickel-cadmium battery which provides a run-time of approximately 45 minutes, which is insufficient for the duration of a standard HBOT session. The battery was therefore removed from within the console.

The ECMO console and circuit was initially subjected to pressures up to 304 kPa (3.0 atmospheres absolute [atm abs]) to test for structural integrity, using several pressurisation and depressurisation profiles as per our equipment testing matrix.

Oxygen safety

Assessment of power and electrical safety (including ignition risk), motor type and configuration, mechanical and electrical relays and temperature testing were performed. Modifications, where performed, are detailed below in the results section.

Circuit flow performance

Testing of the ECMO circuit flow was performed with an independent in-line flow sensor [SEN-HZ21WA (½") PVC] to validate the readings of the console at various chamber pressures. Other than insertion of the in-line flow sensor via two barbed fittings for in-vitro tests, the ECMO PLS set circuit was not modified.

Flow performance was assessed against the manufacturer's specifications with saline in the circuit, to a maximum pressure of 304 kPa and during hyperbaric chamber pressurisation and decompression rates of 180 kPa·min^{-1}.

Bubble assessments

An initial visual inspection was performed to assess for the generation or introduction of macroscopic bubbles in the oxygenator or the circuit during compression, maintenance and decompression phases using an ECMO circuit primed with normal saline and set to 2,290 revolutions·min^{-1} (rpm). The chamber was pressurised and decompressed at 30 kPa·min^{-1} and a plateau pressure of 243 kPa (2.4 atm abs) was used. The set O_2 flow rate to the membrane oxygenator was increased in intervals of 1 L·min^{-1} until a final set flow rate of 7 L·min^{-1} was achieved. ECMO circuit flow rate was also monitored throughout the course of this trial. After completion of O_2 flow rate tests at 243 kPa (2.4 atm abs) the circuit was clamped, revolutions reduced to zero, and the chamber decompressed at 30 kPa·min^{-1}. On return to normobaric conditions, the circuit was connected to a Getinge Rotaflow II (Rotaflow 2) console with an in-line ultrasonic bubble sensor (FBS 3/8" x 3/32" L1.7) to provide an objective assessment of bubble status within the circuit. The pump was set to 1,885 rpm achieving a flow rate of 4.80 L·min^{-1}, to enable the bubble detector to complete its assessment.

CLINICAL TESTING

HBOT was considered for a critically ill patient who had failed to respond to all other treatment options and was on VV ECMO support. Following recommissioning of the legacy Rotaflow 1 device with its modified power supply, hospital biomedical engineering review and approval, ethics committee endorsement and informed consent from the patient's medical treatment decision maker, the device was utilised in an experimental capacity for this patient.[9] A treatment pressure of 140 kPa (gauge) (2.4 atm abs) and compression and decompression rates of 30 kPa·min^{-1} were utilised. Protocolised assessment of device stability, performance, bubble assessment and clinical status occurred during each of the 13 HBOT sessions that followed, and arterial blood gases were taken to ensure target oxygenation was achieved and ventilation maintained.

Results

HYPERBARIC SAFETY ASSESSMENT, MODIFICATIONS AND *IN VITRO* TESTING

General safety

The device was visually inspected and underwent all performance verification tests as per the Getinge service protocol. The device maintained structural integrity and performed according to the manufacturer's specifications up to the testing limit of 304 kPa (3 atm abs). There were no closed gas spaces that required venting, and no activation of console buttons occurred with pressure changes.

Oxygen safety

The console was reconfigured to run on a dedicated and highly redundant 24 V DC medical power supply installed into the hyperbaric chamber. Electrical component temperature testing of the power supply and control boards was performed under load and remained within recommended safe limits (Figures 2 and 3).[10] The Rotaflow drive head has a brushless motor and was not modified. Nitrogen purging was introduced to reduce ignition risk from dust accumulation and/or static production and to improve cooling of the console's electronics.

Circuit flow assessment

Flow performance of the Rotaflow 1 (set at 2,500 rpm, 4.63 L·min^{-1} flow) was maintained to the manufacturer's specifications during saline PLS set circuit testing to 304 kPa (3 atm abs) with pressurisation and decompression rates of 180 kPa·min^{-1}. Preliminary flow tests demonstrated

Figure 2
Temperature data log of the Rotaflow power supply board; TC – thermocouple

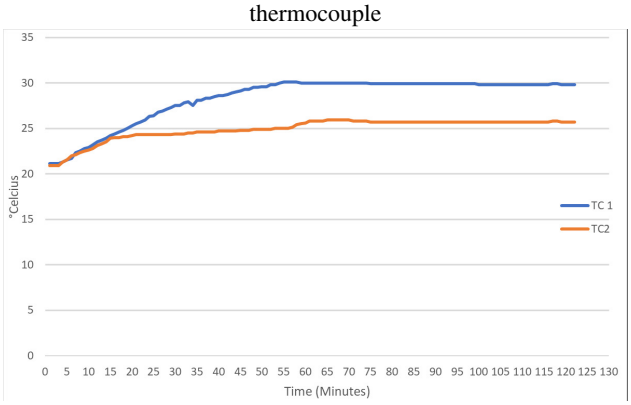
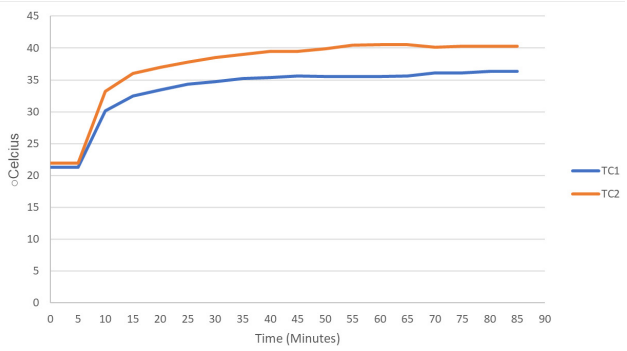


Figure 3
Temperature data log of the Rotaflow control board; TC – thermocouple



sufficient agreement between the displayed rpm and flow on the console to suggest that it would be unlikely that flow variation during pressure changes would have clinically significant impacts on cardiopulmonary support.

The measured flow in the independent in-line flow sensor and displayed on the Rotaflow 1 console demonstrated consistency to within 180 mL·min^{-1}, that is, within 10% variance. There were otherwise no changes noted in displayed flow on the console or in-line flow sensor when compared at 101.3 and 284 kPa (1 and 2.8 atm abs) (Table 2). There were no significant changes in flow or rpm displayed on the Rotaflow 1 console when flow variability was assessed during 30 kPa·min^{-1} pressurisation and decompression phases.

Bubble assessments

To assess for bubble formation, oxygen gas flow to the oxygenator was progressively increased from flowmeter indicated gas flow of 1 L·min^{-1} up to 7 L·min^{-1} at 243 kPa (2.4 atm abs) chamber pressure. Visual assessment throughout all phases did not indicate any bubble formation.

After completion of final O_2 flow rate tests, clamping of the circuit and decompression of the chamber, the circuit was assessed for bubbles by two ECMO physicians using an in-line bubble sensor (FBS 3/8" x 3/32" L1.7) on a Rotaflow 2 console, and no bubbles were detected.

CLINICAL TESTING

Thirteen daily HBOT sessions were completed over 15 days without interruption or dysfunction in the delivery of VV ECMO.[8] Specifically, the structural integrity and performance of the device was maintained. No bubbles were visually or clinically suspected. Invasive mechanical ventilation parameters and ECMO blood and gas flows were titrated by experienced critical care and hyperbaric physicians.[9] The patient remained clinically stable

Table 2
Independent in-line testing of flow rates at 101.3 and 284 kPa (1.0 and 2.8 atmospheres absolute); Rev·min^{-1} – centrifugal pump revolutions per minute

Rev·min^{-1}	Rotaflow display (L·min^{-1})	In-line flow sensor (L·min^{-1})
101.3 kPa (1 atmosphere absolute)		
2000	2.25	2.20
2200	2.42	2.40
2400	2.81	2.80
2600	3.13	3.01
2800	3.45	3.51
3000	3.61	3.70
3200	3.84	3.90
3400	4.51	4.41
3600	4.68	4.79
3800	4.71	4.72
4000	4.98	5.01
284 kPa (2.8 atmospheres absolute)		
2000	2.28	2.10
2200	2.55	2.45
2400	2.82	2.80
2600	3.09	3.01
2800	3.35	3.25
3000	3.62	3.70
3200	3.91	3.90
3400	4.19	4.01
3600	4.45	4.35
3800	4.71	4.72
4000	4.98	4.95

throughout the HBOT sessions without any significant changes to Rotaflow rpm, ventilation parameters or fresh gas flow as measured in actual litres per minute. Reductions in vasopressor support are typically expected during the course of HBOT due to hyperoxia-induced vasoconstriction and increased hydrostatic pressure, and this was demonstrated, as was the need for support to be increased again (although generally not back to pre-treatment levels) during the decompression phase.

Serial arterial blood gases were taken from a peripheral radial arterial line and pre- and post- oxygenator via 3-way connectors, demonstrating that target oxygenation was achieved and ventilation maintained. A standard blood gas machine has an upper reporting limit of ~700–800 mmHg pO_2, and as anticipated on un-diluted samples, the levels of hyperoxia generated were unreportable. The use of an experimental dilutional technique to assess the degree of hyperoxia suggested an oxygen partial pressure of ~1,500 mmHg. This correlated clinically with a loss of colour differentiation between return and access ECMO cannulae.

There were no HBOT related complications. Following his course of HBOT, the patient's clinical condition continued to improve and after a prolonged period of recovery and rehabilitation, he was discharged home with the ability to care for himself independently.[8]

Discussion

DEVICE MODIFICATION PRINCIPLES

In the hyperbaric environment, fire risk is not tolerated. Addressing fire risks related to ignition, fuel and ambient oxygen concentration are key to maintaining a safe hyperbaric environment. When validating equipment for use in the hyperbaric environment, other key principles include:
1. Ensuring structural integrity of the equipment
2. Identifying and venting any possible confined air spaces
3. Ensuring consistent and predictable equipment performance
4. Utilising nitrogen or air purge techniques to cool, deoxygenate and maintain cleanliness of internal equipment parts
5. Regular checking, maintenance and servicing by biomedical engineers and technicians.

The majority of the oxygen entering the gas port of the oxygenator is not transferred into the patient but is vented.[11] This has the potential to increase ambient oxygen levels and underscores the importance of ambient oxygen monitoring and adequate chamber ventilation. The National Fire Protection Agency (NFPA) 99 Standard for hyperbaric facilities in healthcare and Australian Standard 4774.2 are two of a number of useful guidance documents on safe practice.

BUBBLE CONSIDERATIONS

Intravascular gaseous microemboli (GME) occur routinely with ECMO, particularly at times of patient movement, IV fluid infusion and injection.[12] Transcranial Doppler commonly demonstrates cerebral microembolic signals in patients on veno-arterial (VA) ECMO, and to a lesser degree, VV ECMO support.[13,14]

Under normobaric conditions, oxygenators reduce the volume of GME. For example, using similar equipment to the current report (Bioline heparin-coated tubing system, Rotaflow centrifugal pump and Quadrox-i Adult oxygenator without integrated arterial filter), one study found removal of larger bubbles (with effectiveness progressively increasing from GME diameters of 150 μm up) and reduced overall GME volume delivered, at the expense of an increase in the number of smaller bubbles post-oxygenator (perhaps due to fractionation of larger bubbles into smaller ones).[15] Data are unavailable on oxygenator interactions with GME number and volume under hyperbaric conditions, although an *in-vitro* study showed GME removal was more effective under hypobaric conditions.[16]

Although existing GME would shrink upon pressurisation by Boyle's law, GME generated at pressure may expand in volume during decompression. The expected high fraction of oxygen in these bubbles is a mitigating factor in these concerns. Metabolic consumption of oxygen by the surrounding tissues will work to diminish and resolve bubbles. As a result, the risk of a clinically significant obstructive or inflammatory effect of oxygen bubbles should be minimal compared with the more concerning risks of air bubbles.

The Quadrox oxygenator is rated to a maximum blood flow of 7 $L \cdot min^{-1}$ and gas flow of 14 $L \cdot min^{-1}$ at sea level pressure. The practice in our ICU was to limit gas flow to 10 $L \cdot min^{-1}$. Taking into account an increase in gas density at 140 kPa (gauge) (2.4 atm abs), the maximum indicated gas flow rate from the variable cross-sectional area 'ball in tube' flowmeter utilised was restricted to 8 $L \cdot min^{-1}$ to reduce the risk of gas pressure damage to the oxygenator. Within this gas flow limitation during the HBOT sessions we provided, there were no observed issues with bubble entrainment or oxygenator malfunction and carbon dioxide clearance was adequate. In principle, the Quadrox oxygenator can receive gas flow with an oxygen concentration from 21% to 100% from a gas blender or can be supplied with 100% oxygen directly from a medical gas wall outlet through an oxygen flowmeter. Australian medical gas standards require gas supply delivery to flowmeters at 415 to 430 kPa above ambient pressure and flowmeters that can deliver up to 15 $L \cdot min^{-1}$ of flow. This capacity is installed into our hyperbaric chambers.

Given some uncertainty around the potential for bubble formation, a 'safety stop' was introduced at 60 kPa (gauge) (1.6 atm abs) into the compression phase of our HBOT sessions to allow time for the ECMO attendant to check for equipment issues and to investigate for any visible bubble formation. To further reduce the risk of inert gas bubble formation, the modified HBOT treatment table contained no 'air breaks' and the patient ventilation gas was set to 100% oxygen from the time of arrival at the hyperbaric chamber until the time of return to the intensive care unit.

BLOOD FLOW CONSIDERATIONS

To adhere to manufacturer-recommended procedures and implement robust quality control measures, real-time flow monitoring was considered an essential safeguard to minimise risks associated with ECMO equipment in hyperbaric environments and promptly identify any potential complications resulting from the use of the device under hyperbaric conditions.

During HBOT our patient demonstrated preserved blood flow with a predictable non-clinically significant difference in console-displayed flow and the independent flow meter. This suggests that the blood flow in the ECMO circuit in hyperbaric conditions continues to behave similarly to normal saline flow, a Newtonian fluid, where shear rate does not affect viscosity of fluid.[17,18]

LIMITATIONS

The Quadrox oxygenator is reported to reduce the delivered volume of GME by about 70%, and removes nearly all very large bubbles (350 μm and bigger) under normobaric conditions.[15] This process has been demonstrated to be more efficient under *hypobaric* conditions (likely due to the increase in bubble volume that occurs with a reduction in ambient pressure due to Boyle's law), but it is not known if it is less effective under *hyperbaric* conditions, and if so, the clinical impact of this. However, within the limits of the technology available at the time of our testing and clinical use of the Rotaflow 1, no bubbles were visualised under hyperbaric conditions.

FUTURE DEVICE MODIFICATIONS

The Rotaflow 1 console required minimal modification for safe use in the hyperbaric environment and commissioning checks were performed by a Maquet/Getinge company representative after the change from battery to external power supply to facilitate our first HBOT with ECMO. The Rotaflow 1 has, however, been superseded by the Rotaflow 2. Given limited ongoing support for the older device it will be important to validate the new Rotaflow 2 console and components in the future.

As part of an equipment minimisation strategy, the ECMO heater was disconnected during HBOT. Hyperbaric validation of the ECMO heater or addition of an alternate heating mechanism would allow for improved thermal control of the patient in the chamber.

Additionally, assessment for suitability, and validation of other ECMO models would also be useful.

TREATMENT TABLE PRESCRIPTION

We adjusted our usual treatment table for this case and used an oxygen-only table with 85 minutes at 140 kPa (gauge) (2.4 atm abs), and pressurisation and decompression rates of 30 $kPa \cdot min^{-1}$. We added a two-minute compression pause at 60 kPa (gauge) for ECMO equipment and staff safety checks. Considerations in the development of this table included maximising oxygen delivery time at pressure whilst ensuring a no-decompression obligation profile for inside staff so that decompression could be conducted at any time for any ECMO-related emergencies.

Conclusions

Our testing of the Rotaflow 1 with a PLS circuit primed with saline and subsequent clinical use with a veno-venous configuration has demonstrated safety and uncompromised performance of the device up to 304 kPa (3.0 atm abs) and 243 kPa (2.4 atm abs) respectively.

DIRECTIONS FOR FUTURE RESEARCH

Areas for future research include more detailed in-vitro testing of flow and pressure limits of the ECMO circuit using blood with differing levels of haematocrit and viscosity; this would add to our understanding of flow dynamics within the ECMO circuit and the relationship to ambient pressure and would highlight possible variations of device performance in the hyperbaric chamber. The bubble-handling characteristics of the Quadrox oxygenator under hyperbaric conditions should be further clarified, and an assessment made of the potential clinical impact of any potential differences in these characteristics to patients supported by VA compared to VV ECMO. Future trials of patients on a VA ECMO configuration will also help validate the safety of the equipment and configuration in line with the expected vasoconstriction and bradycardia related to HBOT. Finally, the newer model (Rotaflow 2) should be validated for use in the hyperbaric setting as this could significantly improve HBOT delivery protocols and workflows.

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Original articles

Functional outcome and quality of life after iatrogenic cerebral air embolism treated with hyperbaric oxygen: a prospective cohort study

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Keywords
Arterial gas embolism; Health surveys; Hyperbaric medicine; Treatment sequelae

Abstract
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Introduction: Iatrogenic cerebral air embolism (CAE) is an uncommon, potentially fatal condition characterised by accidental introduction of air into the circulation during invasive procedures. Prompt recognition and treatment with hyperbaric oxygen therapy (HBOT) are required. Data on long-term functional outcome and specifically quality of life (QoL) in patients experiencing CAE are limited.

Methods: This prospective, single-centre, observational cohort study examined patients with iatrogenic CAE who were treated with HBOT. Patient characteristics, clinical severity scores and treatment details were recorded. The primary outcomes of the study were the Glasgow Outcome Scale (GOS) score at discharge and six months, and QoL measured by the World Health Organization quality of life brief version at six months.

Results: A total of 22 patients were included, with 14 patients (64%) having arterial CAE, five (23%) retrograde venous CAE, and the remaining three having either both (n = 1) or unknown (n = 2) forms of CAE. Median time-to-HBOT was seven hours [IQR 5–10]. The overall mortality rate was 23% (n = 5), eight of 22 patients achieving full recovery (GOS 5) at six months, and another six patients having moderate disability (GOS 4) at six months. Nine of 17 survivors (53%) reported a decline in QoL compared to their pre-incident status. Outcome in patients with retrograde venous CAE seemed to be better, and outcome in patients with CAE following neuroangiographic procedures for stroke or subarachnoid haemorrhage seemed to be worse, compared to the remainder of patients.

Conclusions: Iatrogenic CAE is associated with substantial morbidity and mortality, with only a third of patients in our cohort achieving good functional recovery. Over half of survivors in this cohort self-reported reduced QoL as compared to their situation before the CAE incident.

Introduction

Inadvertent introduction of gas into the vasculature is a known risk of diving, because reduction of ambient pressure during ascent may lead to pulmonary barotrauma and consequent introduction of gas into the pulmonary veins. The most feared destination of these air bubbles is the cerebral and coronary arteries, requiring urgent application of hyperbaric oxygen therapy (HBOT). While having totally different causes, the iatrogenic introduction of air into the cerebral vasculature bears many similarities with diving related arterial gas embolism.[1] Iatrogenic cerebral air embolism (CAE) can occur during a wide variety of medical interventions, such as cardiac surgery, neurosurgery, vascular catheterisation, and lung biopsies.[2] The outcome of CAE ranges from asymptomatic to fatal.[3] Even though it is a rare condition that affects approximately 2.65/100,000 hospitalised patients, its high morbidity and mortality makes it of critical concern to have a high index of suspicion.[4]

Besides prevention, which is reliant on stringent procedural protocols and awareness during at-risk interventions,[5] rapid diagnosis and intervention are key to reduce morbidity and mortality associated with CAE. Prompt application of an

inspired oxygen concentration as close to 100% as possible, followed by HBOT, is the gold standard treatment.[6] HBOT reduces air bubble size according to Boyle's law and by expediting denitrogenation, thereby aiming to restore blood flow. Additionally it enhances oxygen delivery to affected tissues and has anti-inflammatory properties.[7] Its proven efficacy in mitigating neurological injury has been well documented, and there is an inverse relationship between time to HBOT initiation and the probability of a favourable outcome.[8]

Despite the well-documented risks associated with CAE there is still limited research exploring the long-term impact on patients' functional outcome, and quality of life (QoL) specifically.[4] While acute management focusses on survival and immediate complications, patients may experience long-term deficits.[4] It is not unreasonable to assume that these persistent issues can significantly impact their functional abilities and overall well-being. This study reports on the six-month functional outcome and QoL of prospectively followed patients with CAE who were treated with HBOT.

Methods

STUDY DESIGN AND PATIENTS

This prospective, single-centre, observational cohort study was conducted at the department of Hyperbaric Medicine of the Amsterdam University Medical Center. The local ethics committee reviewed the study and waived the need for formal approval (review number W22_455). Written informed consent was obtained from all patients or their lawful representatives. We included patients from inception of the database in 2020 until the end of 2023 who met the following criteria: (1) at least 18 years of age at the time of diagnosis; (2) diagnosed with CAE; (3) able to communicate in Dutch. Diagnosis of CAE was made clinically (intravascular air entry followed by neurological deterioration) and/or radiologically (presence of intravascular air on computed tomography [CT]).

CLINICAL EVALUATION

Collected data included: general patient characteristics; type and source of the CAE; Glasgow Coma Scale (GCS) score at presentation; neurological, circulatory, and respiratory symptoms; use of high-flow supplemental oxygen before initiation of HBOT; details of HBOT. Type of the CAE (i.e., the cerebral vasculature affected by the air bubbles) was determined to be arterial, retrograde venous, both or unknown, based on a combination of causative procedure, symptomatology and location of intravascular air on imaging. To evaluate illness severity the Acute Physiology and Chronic Health Evaluation II (APACHE-II) and Simplified Acute Physiology Score II (SAPS-II) scoring systems were used.

TREATMENT

All patients were provided with supportive care, including sedation, respiratory management and mechanical ventilation when clinically required. HBOT was initiated as soon as possible after presentation, with the first session adhering to the U.S. Navy Treatment Table 6 (starting at 284 kPa, 2.8 atmospheres absolute [atm abs]) with the possibility of extension if required.[9] Subsequent treatments were conducted daily at 243 or 253 kPa (2.4 or 2.5 atm abs) with a session duration of two hours. Sessions could be repeated to a maximum number of 10 if the patients demonstrated ongoing clinical improvement. In 2022, after consultation with international experts, the protocol was updated to mandate a minimum of three HBOT sessions for all patients, unless the neurological situation was deemed so severe that continuation of HBOT was considered futile.

FOLLOW-UP

Glasgow Outcome Scale (GOS) score was determined at discharge and six months after the event in a telephone call with the patient (or relative, if deceased or unable) by one of the authors (RPW). At six-month follow-up patients were sent the Dutch version of the World Health Organization quality of life brief version WHOQOL-BREF[10] by email or postal mail and requested to fill it out and return it. This questionnaire is a shortened version of the commonly used WHOQOL-100 which investigates QoL on four domains: physical, psychological, social, and environmental. Questions are answered on a 1–5 scale and scores of the four domains are transformed into a 0-100 score according to the official instructions from the WHOQOL user manual (WHO/HIS/HSI Rev.2012.03). In addition to the WHOQOL-BREF questionnaire we asked two supplementary questions regarding self-reported change in QoL between the situation before the CAE and the current situation, and the perceived relationship of this change (if any) with the CAE.

STATISTICS

Data for this study were prospectively entered in Castor EDC (Ciwit BV, Amsterdam, the Netherlands). For patients referred to our institution, prospective data collection started on arrival in our hospital. All efforts were made to retrospectively collect data from the referring hospital. Only descriptive statistics are presented, using GraphPad Prism (version 10.2; GraphPad Software, San Diego, CA, USA), and no inferential statistical analysis was performed.

Results

CLINICAL EVALUATION

Over the four-year period 22 patients were entered into the database, all of whom were eligible for inclusion in this study (Table 1 and Table 2). Most common causative

Table 1
Patient characteristics, severity of illness at admission, and presenting symptoms; for each variable, *n* indicates the number of patients with available data. Continuous data are shown as median (interquartile range). APACHE-II – Acute Physiology and Chronic Health Evaluation II; SAPS-II – Simplified Acute Physiology Score II

Characteristic	*n* = 22
Age (years), median (range)	69 (60–76)
Female sex, *n* (%)	11 (50%)
Referred patient, *n* (%)	16 (73%)
Severity scores at admission, median (range)	
Glasgow Coma Scale score, *n* = 21	10 (8–13)
SAPS-II, *n* = 17	31 (30–42)
APACHE-II, *n* = 18	20 (15–22)
Presenting symptoms, *n* (%)	
Neurological, *n* = 21	21 (100%)
Lateralization, *n* = 20	15 (75%)
Impaired consciousness, *n* = 19	14 (74%)
Confusion / agitation	6 (29%)
Seizure	3 (14%)
Circulatory, *n* = 21	12 (57%)
Cardiopulmonary resuscitation	2 (9.5%)
Hypotension	3 (14%)
Hypertension	4 (19%)
ST-segment abnormalities	4 (19%)
Arrhythmias	2 (9.5%)
Respiratory, *n* = 21	7 (33%)
Respiratory insufficiency	5 (24%)
Haemoptysis	2 (9.5%)

procedures were either radiological or thoracic in nature. In all cases air was the gas involved. Neurological status prior to HBOT was unknown for one patient who was diagnosed intraoperatively based on witnessed air entry into a peripheral vein and air bubbles in the carotid arteries on ultrasound. This patient remained intubated until HBOT. In the remaining 21 patients median GCS score at presentation was 10 [interquartile range (IQR) 8–14]. Fifteen patients (68%) exhibited signs of lateralisation, and three (14%) presented with epileptic seizures. Circulatory symptoms developed in 12 patients (57%), including two who required cardiopulmonary resuscitation. Respiratory symptoms were present in seven patients (33%).

TREATMENT

Nine patients (41%) received full treatment with high flow supplemental oxygen, defined as initiation soon after diagnosis of CAE and continuation until start of HBOT (Table 3). HBOT was initiated a median of seven hours from symptom onset, which includes two outliers who received their initial session at 22 and 23 hours post embolism. All

Table 2
Air embolism characteristics, imaging and causative medical procedures

Air embolism characteristics	*n* (%)
Vascular bed of entry	
Cerebral arterial	5 (23%)
Systemic arterial	2 (9.1%)
Pulmonary venous	6 (27%)
Systemic venous	8 (36%)
Left heart	1 (4.5%)
Type of cerebral air embolism	
Arterial	14 (64%)
Retrograde venous	5 (23%)
Both	1 (4.5%)
Unknown	2 (9.1%)
Air on imaging, *n* = 21	
Yes, intracerebral air	10 (48%)
Yes, air in other vasculature	4 (19%)
No air	7 (33%)
Causative medical procedure	
Interventional radiology	
Neuro angiography	5 (24%)
Cardiac angiography	2 (9.1%)
Thoracic procedures	
Cardiac valve surgery	1 (4.5%)
Bronchoscopy	1 (4.5%)
Lung or pleural biopsy/puncture	4 (18%)
Chest drain manipulation	1 (4.5%)
Other	
Haemodialysis	3 (14%)
Arthroscopy	1 (4.5%)
Endoscopy	1 (4.5%)
Central venous catheter	2 (9.1%)
Peripheral venous catheter	1 (4.5%)

initial sessions adhered to the treatment protocol, and for two patients an extension at 284 kPa (2.8 atm abs) was applied. Both these patients were not intubated, and extension was performed due to persistence of neurological symptoms. A total of 82 HBOT sessions were conducted, with 19 (86%) patients receiving at least two sessions and 12 (55%) patients receiving at least three sessions. The majority of abnormalities (10 of 19) in the hyperbaric chamber occurred during the first session (Table 3).

FOLLOW-UP

The median length of hospital stay was six days [IQR 2–9], with one patient who died on the second day post-embolism after withdrawal of life support because of the extent of

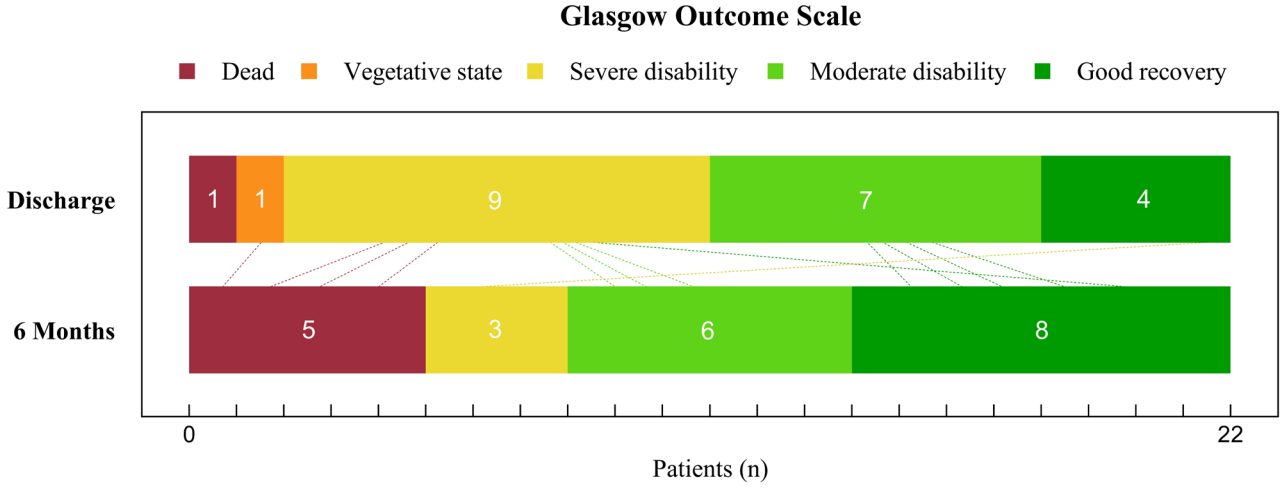
Table 3

Treatment characteristics and abnormalities during hyperbaric oxygen therapy (HBOT); continuous data are shown as median (interquartile range). Regarding high-flow supplemental oxygen prior to HBOT, 'completely' means initiation of (close to) 100% inspired oxygen concentration immediately after diagnosis until HBOT, 'partly' means that supplemental oxygenation was performed, but not from diagnosis until HBOT and/or not with adequately high flow

Treatment characteristics		**Abnormalities during HBOT (*n* = 82 sessions)**	
Time from incident to diagnosis (hours)	1 (0–2)	Psychomotor agitation	6 (7.3%)
Time from incident to HBOT (hours)	7 (5–10)	Seizure without indication for O_2 toxicity	5 (6.1%)
Intubated prior to HBOT	13 (59%)	Respiratory deficits requiring supportive care	3 (3.7%)
Normobaric oxygen prior to HBOT		Vomiting	2 (2.4%)
Completely	9 (41%)	Neurological deterioration	1 (1.2%)
Partly	4 (18%)	Seizure suggestive of O_2 intoxication	1 (1.2%)
None	9 (41%)	Respiratory deficits requiring intubation	1 (1.2%)
HBOT sessions per patient	**3 (2–5)**		
With anaesthetist present	3 (1–4)		

Figure 1

Stacked bar chart illustrating the Glasgow Outcome Scale (GOS) score at hospital discharge and six-month follow-up; each bar is subdivided into GOS scores 1 (left) to 5 (right). Dotted lines between bars show changes in individual patient outcomes from discharge to six-month follow up



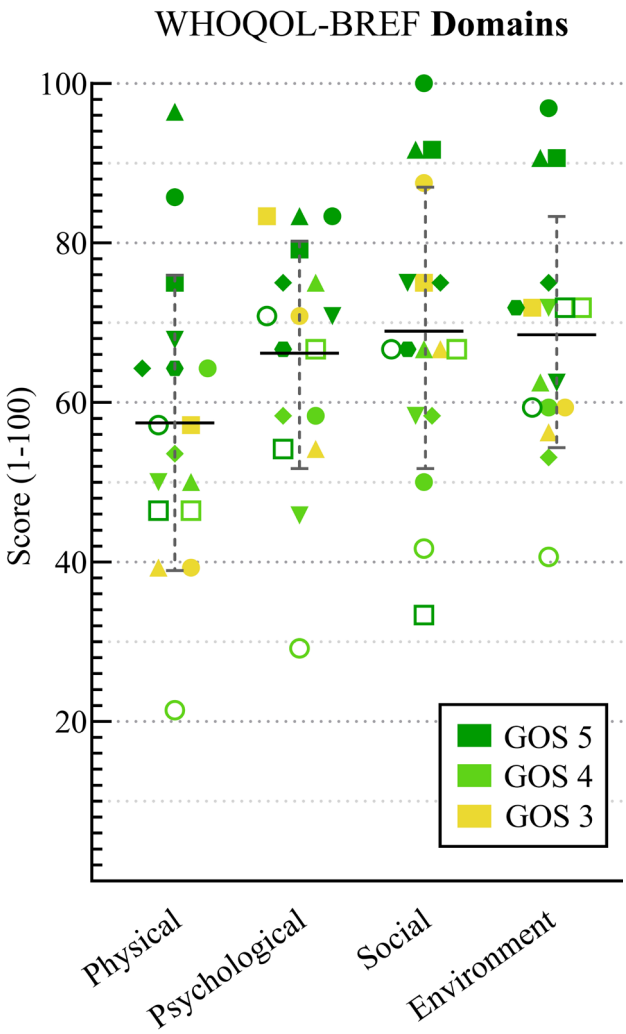
cerebral injury. The overall mortality was 23% (five patients) at six months. GOS data were received from all surviving patients at discharge, GOS and QoL data were received from all surviving patients at six months. At the time of discharge the median GOS score was 4 [IQR 3–4], and at six-month follow-up the median GOS score was 4 [IQR 3–5] (Figure 1).

To further assess QoL after sustaining CAE, QoL was measured using the WHOQOL-BREF questionnaire at six-month follow-up. Median overall perception of QoL and health satisfaction were both 3 out of 5 [IQR 2–4]. The mean (SD) scores for the WHOQOL-BREF in the four domains were as follows: physical health 58 (18), psychological health 66 (15), social relationships 69 (18), and environment 69 (15) (Figure 2).

SUBGROUPS

Retrograde venous CAE has a distinct aetiology that differs from arterial CAE. Likewise, patients with arterial CAE as a complication of a neuroangiographic procedure for stroke or subarachnoid haemorrhage are an interesting subpopulation, because they suffered two serial episodes of cerebral injury. Although no formal statistical testing was performed, it seems from Table 4 that patients with retrograde venous CAE had better outcomes than the remainder of patients. All five patients survived and reported no deterioration of their health as compared to before the CAE. However, they were also younger and had shorter time from incident until start of HBOT than the remainder of patients. Conversely, only two of five patients with CAE after neuroangiographic procedures survived, and both survivors self-reported

Figure 2
World Health Organsiation quality of life brief version (WHOQOL-BREF) domain scores at six month follow-up in the 17 survivors, color-coded by Glasgow Outcome Scale (GOS) score; symbols represent individual patients and the black and grey lines show the mean (standard deviation) for each domain



deterioration of their health compared to before the event. They were, however, older and more likely to be intubated before HBOT.

Discussion

In this prospective cohort study we evaluated the outcome and QoL of 22 patients who suffered iatrogenic CAE and were treated with HBOT. To our knowledge, this is only the second prospective study focusing on iatrogenic CAE. Crude mortality in this study was 23%, which underscores the severity of this condition. At six-month follow-up, eight of 22 patients achieved complete recovery (GOS 5) and another six of 22 patients had moderate disability (GOS 4). Despite HBOT and supportive care, the remaining eight of 22 patients either died (n = 5, GOS 1) or were left with severe disabilities (n = 3, GOS 3).

In our cohort, neurological dysfunction most frequently manifested as signs of lateralisation (75%) and/or decreased consciousness (74%), with circulatory (57%) and respiratory (33%) disabilities also frequently observed. In general, the severity of illness was somewhat less pronounced than that reported by the only previously published prospective cohort study, in which median GCS score was eight [IQR 3–15] and SAPS-II was 33 [IQR 21–55], in contrast to our median GCS score of 10 [IQR 8–14] and SAPS-II of 31 [IQR 30–42].[4] Furthermore, more patients in the previously published cohort experienced cardiac arrest and shock compared to our study.

The profile of causative procedures appears to be different in our study than in previously reported series. In earlier studies, cardiac surgery and central venous catheterisation were identified as the primary cause, in contrast to our cohort with predominantly radiological and lung procedures.[4,11,12] One explanation may be the general increase in the use of interventional radiology, for instance for mechanical thrombectomy in acute stroke, which by its nature carries a high risk of CAE.[13] Similarly, the inherent nature of lung surgery and lung biopsies continues to pose a procedural risk.[14]

Interestingly, although CAE is regarded a primarily clinical diagnosis, CT imaging was available in all but one of our cases, showing intravascular air in 67% of them. In diving medicine, a diver who surfaces with acute neurological injury may be treated with HBOT without prior cerebral imaging. The fact that in our clinical cases almost all patients undergo CT imaging is probably a result of the clinical reality in which the incident occurs. The rarity of iatrogenic CAE, suboptimal clinical awareness among non-hyperbaric physicians and the broad differential diagnosis of acute neurological symptoms in the hospitalised population most likely explain why most patients in our series underwent CT imaging. It must be reiterated, however, that intravascular air is not a prerequisite for the diagnosis of CAE, although its presence does support the diagnosis.

Previous CAE studies have largely focused on neurological sequelae and functional outcomes (e.g., GOS), rather than patient-reported QoL. We applied the WHOQOL-BREF at six months, providing new insights into self-perceived well-being. It must be noted that all our patients were, by definition, already exposed to a medical procedure before they suffered CAE. Compared with previously hospitalised elderly patients (mean age 80 years) who reported QoL six months after hospital admission, our CAE survivors reported comparable scores in the physical (58 vs. 58), psychological (66 vs. 67) and social (69 vs. 65) domains, and better scores in the environmental (69 vs. 55) domain.[15]

Table 4

Outcome for all patients, patients with retrograde venous cerebral arterial embolism (CAE), patients with CAE that occurred during neuroangiography, and all patients with exception of these two categories; continuous data are shown as median (interquartile range). *as only two observations were made the unique values of both patients are shown; CAE – cerebral air embolism; GCS – Glasgow Coma Scale; HBOT – hyperbaric oxygen therapy

Characteristic	All patients *n* = 22	Retrograde venous CAE *n* = 5	CAE during neuroangiography *n* = 5	Remainder
General				
Age	69 (60–76)	59 (50–73)	75 (69–77)	68 (61–76)
Female sex	11 (50%)	2 (40%)	2 (40%)	7 (58%)
GCS score at admission	10 (8–13) (*n* = 21)	12 (9–15)	9 (8–9)	11 (8–14) (*n* = 11)
Intubated prior to HBOT	13 (59%)	2 (40%)	4 (80%)	7 (58%)
Time from incident to HBOT	7 (5–10)	5 (5–5)	6 (3–11)	8 (5–11)
Overall mortality	5 (23%)	0 (0%)	3 (60%)	2 (17%)
Discharge				
n survivors	21	5	4	12
Glasgow outcome score	4 (3–4)	5 (4–5)	3 (3–3)	4 (3–4)
Six months				
n survivors	17	5	2	10
Glasgow outcome score	4 (3–5)	5 (3–5)	1 (1–4)	4 (4–5)
Quality of life	3 (2–4)	3 (2–4)	3 and 4*	4 (2–4)
Health satisfaction	3 (2-4)	3 (2–4)	3 and 4*	4 (2–4)
Health deterioration	9 (53%)	0 (0%)	2 (100%)	7 (70%)

When comparing functional recovery in our cohort to previous CAE research, our outcomes appear less favourable. The prospective study by Bessereau et al. notes that 75% of patients achieved a good recovery by six months, while only 33% of our patients reached this level of functional independence.[4] It must be noted, however, that Bessereau et al. included not only CAE, but also cases of gas embolism with only circulatory abnormalities. It cannot be calculated from their data how many patients actually had CAE and how many had gas embolism without cerebral involvement. In this regard, the retrospective study by Blanc et al. is more informative, since they only included patients experiencing cerebral gas embolism.[12] Although GCS is not reported, their 70% incidence of impaired consciousness at presentation is comparable to our data. They report that 58% of their patients had full recovery on discharge, compared to 14% in our data (increasing to 36% at six months). Another retrospective study, by Beevor and Frawley, that only included patients with cerebral gas embolism, reported a mean moderate disability at discharge (6.5 on the extended GOS that ranges from 1 to 8),[11] whereas our median GOS score at discharge was only three, reflecting severe disability. However, median GOS score in our cohort increased to four (moderate disability) at six-month follow up, and these data are not known for the Beevor and Frawley cohort.[11] The comparatively worse condition of our patients at discharge may be partly explained by the fact that the percentage of patients with hemiplegia was much higher in our cohort (75% as opposed to 20% in the Beevor and Frawley study) and the fact that hemiplegia was associated with poor outcome in the Beevor and Frawley study.

An important point to make when comparing studies, is that distinction between cerebral arterial and cerebral retrograde venous air embolism is usually not made. Although the abovementioned studies do report on arterial versus venous embolism, from the text it can be deduced that this refers to the type of vessel where the air was introduced, not to the cerebral vasculature in which the bubbles lodged. Despite absence of studies investigating the clinical outcome of arterial versus retrograde venous CAE, mechanistically it can be hypothesised that retrograde venous embolism has a more benign course, given the fact that these bubbles have ascended retrogradely to the cerebral veins and therefore did not obstruct arterial flow. The small size of our cohort precludes meaningful quantitative analysis, but it is interesting to note that the patients with retrograde venous CAE seemed to have higher GOS score at six-month follow up and none of them self-reported a deterioration in their health compared to baseline. On the other hand, however, these patients were younger and their median time to HBOT was shorter than in the remainder of the study population, which may confound the suggested improved outcome.

Another relevant subgroup in our cohort is formed by the five patients who suffered CAE as a complication of either mechanical thrombectomy for stroke (n = 4) or coiling after subarachnoid haemorrhage (n = 1). These patients suffered two serial episodes of cerebral injury. Only two of these patients survived and both reported reduced QoL compared to before the events. The increasing use of neuro-interventional procedures warrants further study into the incidence of CAE and the role of HBOT in these patients.

Strengths of our study are its prospective nature and complete availability of outcome data. Several limitations should however be acknowledged. Firstly, the relatively small size of our cohort, reflective of the rarity of CAE, constrains the generalisability of our findings and precludes statistical analysis to identify prognostic factors. This is compounded by the heterogenic background of patients suffering CAE, which is indicative of the wide variety of medical procedures that can cause this complication. Secondly, the functional status of our patients before the CAE incident is unknown. All patients at least required a medical intervention that caused the CAE. It cannot be determined with certainty to what extent the functional outcome as determined at six months is attributable to the CAE, or to any underlying disease. Specifically, when CAE occurs as a complication of a neurological intervention such as thrombectomy after stroke, analysis of outcome is troubled by the fact that patients already suffered from neurological injury before they sustained CAE. Lastly, the follow-up period was limited to six months, which in neurological disorders may not adequately reflect final outcome. Nevertheless, evidence from stroke literature suggests that the six-month timepoint can be predictive of longer-term results.[16]

Conclusions

We have shown that in our cohort of 22 patients who suffered iatrogenic CAE, six-month mortality was 23%, while 36% obtained good functional outcome. The fact that only 41% of our patients received continuous high-flow supplemental oxygen prior to HBOT indicates a potential for improving care, by continuing emphasis on this important initial treatment. Also, the median time until initiation of HBOT of seven hours, despite only a median of one hour between onset of symptoms and diagnosis, suggests that familiarity with HBOT and logistical factors such as referral and transportation can still be optimised. Given the recently reported[8] clear inverse relationship between time-to-HBOT and probability of favourable outcome, all efforts should be pursued to initiate HBOT as early as possible. The suggestion of improved outcome in retrograde venous CAE as compared to arterial CAE warrants further investigation in future studies. The specific subpopulation of CAE after neuroangiographic procedures also requires additional study.

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The incidence of cardiac arrest requiring defibrillation and defibrillation protocols in Australasian hyperbaric units

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Cardiovascular; Diving medicine; Fire or explosion; Hyperbaric medicine; Hyperbaric facilities; Pressure chambers; Safety

Abstract
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Introduction: Cardiac arrest (CA) during hyperbaric oxygen treatment (HBOT) is exceedingly rare with only a few cases reported. It is unknown if in-chamber defibrillation of a patient has been performed in Australasia. In-chamber defibrillation is potentially dangerous with the risk of fire in an oxygen-rich environment. Australasian Standards prohibit the use of currently available defibrillators licensed for in-chamber use, as they contain lithium batteries. This study aimed to investigate how CA is managed in Australasian hyperbaric medicine units (HMUs) and to establish if there is a need to develop standardised protocols.

Methods: A 10-part SurveyMonkey® questionnaire sent to all 15 Australasian HMUs. Questions aimed to ascertain if there were cases where defibrillation during HBOT was indicated and if it was performed. We asked about emergency treatment protocols, defibrillation capabilities and if regular training drills were conducted. We asked if colleagues felt the need to have a uniform treatment protocol across Australasia and invited them to share their emergency protocols.

Results: Fourteen responses (93.3%) were received. No clinical cases of in-chamber CA or defibrillation were reported. Examples of emergency treatment protocols were provided by two respondents. Six respondents (43%) stated that regular emergency training drills for CA are performed in their HMU. Eleven respondents (79%) favoured standardised treatment protocols; however, comments suggested that this might be unachievable.

Conclusions: CA requiring defibrillation in the hyperbaric medicine context is rare and has not been performed in Australasia. Most HMUs have protocols in place, but they are not universally practiced regularly.

Introduction

Cardiac arrest (CA) in the context of hyperbaric oxygen treatment (HBOT) is a rare, yet critical event requiring special considerations. The incidence of defibrillation during HBOT in Australasia is unknown and there are only a few reported cases worldwide.[1–7] The purpose of this study was to ascertain the incidence of CA requiring defibrillation from all hyperbaric medicine units (HMUs) in Australasia.

Patient cohorts in HMUs are often elderly, at risk of coronary artery disease and its complications (including CA), and present with multiple co-morbidities.

When a cardiac arrest occurs during HBOT, the aetiology is of significance to determine the most suitable management option whilst aiming to minimise the risk of harm to other chamber occupants (other patients and medical attendants).

Hyperoxygenation during HBOT seems to confer a protective effect and may prevent cardiac insults to a certain degree, as well as providing a 'grace period' during which cardiopulmonary resuscitation (CPR) can be delayed. The high dissolved blood oxygen concentration is assumed to delay the onset of tissue hypoxia, buying time to decompress and provide out-of-chamber defibrillation.[4,6]

CA with ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) is best managed with early defibrillation. There is overwhelming evidence that the timely delivery of a direct current (DC) countershock in such cases is the most significant determinant of survival.[1,2] The Australian and New Zealand Committee on Resuscitation recommends that a shock is delivered as soon as a defibrillator is available and that pads are placed on the exposed chest in the antero-lateral or anterior-posterior position (in patients with an implantable cardioverter-defibrillator or a permanent pacemaker at least 8 cm from the generator position). Biphasic waveforms should be used with a default energy level of 200 joules (J) for all shocks; if not successful it is reasonable to increase the energy for subsequent shocks.[3] A single shock protocol should be followed with an emphasis on delivering

uninterrupted good quality chest compressions to create ideal conditions for successful shock delivery.[3]

Under 'Precautions' (9) and 'Oxygen and Fire Risk' (9.1), attention is drawn to electrical hazards in the presence of water, metal fixtures, oxygen (O_2) and flammable substances, but there is no specific mention of the special circumstance of defibrillation under hyperbaric conditions.[3] Four case reports involving adults and one involving a neonate have resulted in fires caused by sparks generated during defibrillation attempts when paddles were used in the vicinity of high flow O_2 (at ambient atmospheric pressure).[1] However, paddles have now become widely obsolete. There have been no reports of fires caused by sparking when shocks were delivered using adhesive pads.[2] The recommended technique advises taking precautions to minimise sparking by ensuring good pad contact with the chest wall, correct pad placement and that rescuers should try to ensure that defibrillation is not attempted in an O_2-rich environment (i.e., high-flow O_2 directed across the chest).

The Fiona Stanley Hospital HMU has emergency procedures to follow in case of CA requiring defibrillation in monoplace and multiplace chambers (*Appendix 1). All include decompressing the chamber to surface pressure and shock delivery outside the chamber. We aimed to establish the incidence of cardiac arrest requiring defibrillation in Australasian HMUs and to survey their related emergency procedures.

Methods

Written approval was obtained for data review and extraction from Governance, Evidence, Knowledge and Outcomes (GEKO) at Fiona Stanley Hospital, Perth, Western Australia (Approval Number 52502).

A questionnaire was sent to all HMUs in Australasia via SurveyMonkey (SurveyMonkey Inc.) to ascertain the incidence of in-chamber CA requiring defibrillation. The survey was anonymous, with the option to self-identify and provide further details if deemed appropriate. The survey questionnaire (*Appendix 2) also aimed to establish if HMUs had related emergency procedures in place should a CA occur and if they differed with respect to monoplace or multiplace chambers and between different treatment tables. If a HMU had such cases occur, they were invited to provide more detail on how these were managed.

Results

The survey questionnaire and respondents' replies can be viewed in *Appendix 2.

The response rate to the survey was 93.3% (14/15 HMUs). There were no clinical cases of CA, but one historical experiment of multiple in-chamber defibrillations of a large piece of meat while under pressure was reported.

Two respondents provided examples of emergency procedures and 11 (79%) favoured these to be standardised across all HMUs. However, comments suggest that this might be impractical or unachievable.

Discussion

Defibrillation forms an essential part of modern resuscitation algorithms by delivering an electric shock to a patient's chest with the aim of converting a shockable rhythm (ventricular fibrillation or tachycardia) causing cardiac arrest to sinus rhythm.[1–3] This is achieved by storing an electrical charge in a capacitor and then subsequent discharging it to the patient's chest via electrodes (pads or paddles).

Most defibrillators currently in use are biphasic, delivering lower energy shocks via adhesive pads with supposedly less risk of sparking and arcing compared to monophasic shocks delivered with paddles.[1] However, several reports to the US Food and Drug Administration (FDA) database MAUDE (Manufacturer and User Facility Device Experience) indicate sparking even with adhesive pads and there is one report of a patient receiving burns during defibrillation in an ambulance.[4] Sparking, arcing and risk of burns can be minimised by applying a meticulous technique with either paddles or pads. The Australia and New Zealand Committee on Resuscitation recommends to avoid charging paddles unless on the patient's chest, ensuring good contact and no gap between paddles/pads and the chest wall, avoiding placement over ECG electrodes, leads, medication patches, implanted devices or central line insertion sites. The patient should not be in contact with metal fixtures or in a high O_2 environment.[3]

In-chamber defibrillation is potentially dangerous with the most significant risk being fire from elevated partial pressures of O_2 and voltaic arcing providing an ignition source. Performing in-chamber defibrillation with cables being fed out of the chamber via a penetrator and the defibrillator and operator placed outside the chamber may reduce the risk of fire. This system is currently mostly applied in HMUs in Europe and less commonly in North America, Asia and Australasia. None of the Australia-New Zealand respondents were using this capability at the time of our survey.

The results of our survey revealed that there is no actual clinical experience with CA in the diving and hyperbaric medicine context in Australasia. The only reported experience with defibrillation under pressure in a

*** Footnote:** Supplementary Appendix 1 and 2 are available to view at https://www.dhmjournal.com/index.php/journals?id=374

Figure 1
Simulation model for informal in-chamber defibrillation trial Fremantle Hospital 1994

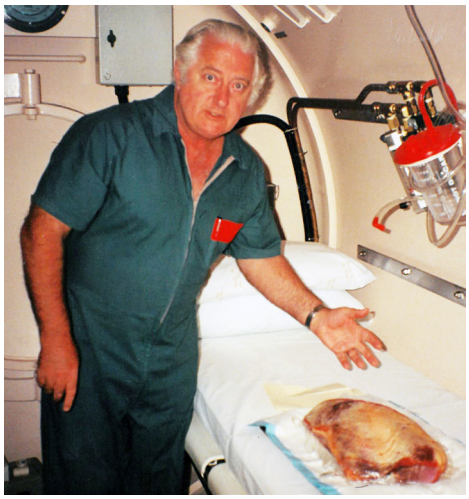
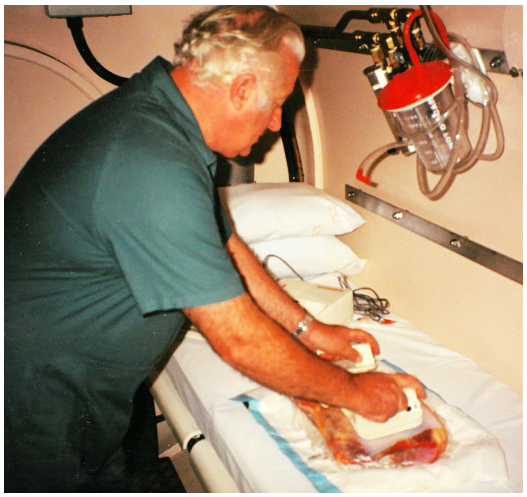


Figure 2
In-chamber defibrillation model in use Fremantle Hospital 1994



hyperbaric chamber was from the Fremantle Hospital HMU, Western Australia, where experiments of defibrillation of a large piece of meat were conducted in 1994 (Figures 1 and 2). Defibrillation was performed with a LifePak 5® defibrillator using paddles (pre-adhesive pad era) with an energy of 360 J, monophasic shock energy delivery, at ambient pressure and at 203, 284, 304 kPa (2.0, 2.8 and 3.0 atmospheres absolute) in a six atmospheres capable cylindrical multiplace chamber. No sparking or other adverse effects were observed. (Personal Communication H Oxer, N Banham, A Waring 2023). The LifePak 5® defibrillator functioned normally at pressure and subsequently.

Griffiths found in his 2007 survey of HMUs worldwide that in-chamber CA was rare.[8] Ten cases were reported by 51 facilities over a five-year period. Three out of the 10 cases received in-chamber defibrillation without adverse effects or reported safety incidents.

Of the five cases of CA that occurred in facilities without the capability to defibrillate at pressure, only one had a shockable rhythm. The condition treated was cerebral arterial gas embolism, the patient and inside attendant the only occupants in a three-person square chamber. CA occurred at treatment pressure (not further detailed), with the time for decompression from arrest to surface reported as 1–3 minutes (mins). Defibrillation was performed outside the chamber and the patient survived.

Of the 12 HMUs with the capability for in chamber defibrillation under pressure, eight were in Europe, one each in the US and Canada and the remainder in Asia and Australasia. Interestingly, two of these units indicated that, despite the capability being present, in-chamber defibrillation was not permitted. The reasoning behind this was not specifically mentioned, though safety concerns (such as unintentional electric shock to staff or patients, fire hazard, complicated procedures leading to mistakes, limited space to carry out safe defibrillation and patient clinical safety issues) amongst the questionnaire respondents were mentioned. These were reported as percentages of responses, not the concerns of the individual facilities.

Patient survival rates for in-chamber cardiac arrest mirrored those of in-hospital cardiac arrest, regardless of being defibrillated inside or outside the chamber.

Griffiths identified the need for more research and covering a longer observation period.[8]

Schmitz et al., reviewed cardiopulmonary resuscitation (CPR) during HBOT in 2023, making recommendations for practice.[7] The extensive literature review concluded that CPR in the context of HBOT is a rare, but a critical event requiring special considerations. The review re-visited known safety issues, including the risks that the presence and use of a defibrillator in the hyperbaric environment poses, i.e., fire risk, implosion of vacuum filled cathode ray tube monitors, device malfunction under pressure and operator error from nitrogen narcosis. Recommendations were that the defibrillator should be stored outside the chamber and, if the capability is present, adhesive pads attached to the patient and fed through a penetrator in the chamber hull connecting to the defibrillator outside. With this setup, unexpected cases requiring defibrillation could have a first shock delivered within a reasonable two-minute period after recognition of CA. If defibrillation is necessary at pressure, an FiO_2 of 21.5% should not be exceeded at the moment of shock delivery. This is even stricter than the 23.5% maximal allowable concentration in the Australasian Standard.[9] In the absence of a defibrillator, some authors recommend a precordial thump in case of witnessed VT or VF, but this remains controversial.[7]

Stricter safety requirements are necessary in clinical hyperbaric chambers for two reasons. There is an increased risk of a fire starting due to the O_2-rich environment and this spreading rapidly or being explosive, and the chamber environment represents a pressurised and sealed space which does not allow immediate access or exit in the event of an emergency, therefore posing a safety risk to both patients and attendants.

The amount of electrical energy discharged by a defibrillator is more than enough to start a fire. For example, an energy of 150 J is still approximately 80 times greater than the minimum ignition energy (MIE) requirement for cotton, a common material used in the hyperbaric setting (MIE 1,950 mJ).[8]

Sustaining a CA during HBOT potentially puts the patient at a distinct disadvantage. Delay to diagnosis, the requirement to decompress, space constraints, limitations to staff access and other measures to reduce fire risk conflict with the patient's need for timely diagnosis and effective resuscitation. All commonly used treatment tables used in Australasia allow rapid depressurisation of a chamber to surface pressure, although the decompression obligation of the inside attendant may need managing. Rapid depressurisation and moving out of the chamber can offset most of the above- mentioned disadvantages. Doing so will facilitate easier management of CA and, given the overall low incidence of shockable ryhthms, not impact on overall survival from CA.

In Australasia, the use of defibrillators licensed for in-chamber use in Europe (Physio-Control LifePak 1000® and GS Elektromed Corpuls3®) has not been permitted according to Standards (AS/NZS 4774.2), as they contain lithium-ion batteries.[9]

In case of cardiac arrest in any HMU in Australasia, because the defibrillator cannot be present inside the chamber when pressurised, chamber decompression must occur if the decision is made to defibrillate. Only one of the responding HMUs in Australasia has the capability for the defibrillator leads to be fed through the chamber hull via a penetrator and is currently evaluating its use with the Corpuls3 defibrillator.

Twelve out of 14 responding HMUs surveyed have emergency procedures in place, and all of these mandate that defibrillation is performed only after the chamber is depressurised. The emergency procedures at Fiona Stanley Hospital states that in case of CA, a monoplace chamber is to be depressurised immediately, the patient slid onto the gurney, and CPR commenced as directed by medical staff. In case of CA in multiplace chambers, there is a distinction between treatment pressure and duration at that pressure which determines if the inside attendant can bring the patient to surface whilst performing chest compressions or if the attendant has to surface in a separate lock to complete their required decompression obligation and a doctor locked into the chamber to surface with the patient, performing chest compressions. This time limit is 60 mins at 140 kPa gauge pressure (14 metres of seawater (msw) equivalent / ~243 kPa absolute pressure), or 45 mins at 180 kPa gauge pressure (18 msw / ~284 kPa absolute pressure), respectively. These emergency procedures are clear, simple, very easy to follow and are on one printed page each. (* Appendix 1). These points are also depicted on the respective treatment tables used.

The emergency procedures at Christchurch Hyperbaric Medicine Unit in New Zealand are much more complex and detailed. The nine-page document distinguishes between multiple treatment tables and incorporates in-hospital adult life support algorithms.

Neither the Fiona Stanley nor Christchurch emergency procedures stipulate the exact timing and location of defibrillation, but the Fiona Stanley procedure does state that the patient is to be removed from the chamber immediately after emergency decompression. An important component of the Fiona Stanley procedure is immediate activation of the hospital's 'Code Blue' resuscitation team by the outside attendant.

Preparedness of all staff in hyperbaric facilities to respond rapidly to an emergency can be maintained and improved by performing regular emergency drills, where emergency procedures and their performance by staff are tested. However, less than half of respondents to our survey reported such drills occur regularly. Incorporating simulation training methods into the environment of HMUs is increasingly being encouraged to improve fidelity and quality of emergency response training. The Master's degree in Hyperbaric and Diving Medicine of the University of Padova in Italy, implemented by Paganini, is an example of how in-situ simulation can be used in this context.[10] A recent Delphi study identified cardiac arrest as one of five scenarios that merit simulation training in HMUs.[11]

Meunier et al., looked at the optimal timing and positioning for safe defibrillation after emergency decompression and opening of an O_2-filled monoplace chamber.[12] They found that it took two to four mins for the O_2-concentration to fall below 23.5% after emergency decompression. The position at which < 23.5% was achieved fastest was on the gurney, outside the chamber.

There appears to be no great desire to change the current response to an in-chamber CA in the Australasian facilities surveyed. The opinion of some practitioners that a uniform approach across all units might be beneficial in managing this rare event is questioned by more experienced colleagues, who advise that this might be difficult, as it does not take individual operational circumstances into account and might in fact be unachievable. The consensus was to keep with current regimes and emergency procedures as per individual units.

Recent technical developments, such as defibrillators with non-lithium-ion batteries or penetrators for defibrillator leads should encourage the hyperbaric community in Australasia to consider moving to an in-chamber defibrillation policy. New technology and understanding would benefit the patients and might lead to progressive risk reduction.[13] The reluctance to change the status quo might have to do with the rarity of the event and the fact that equipment upgrades in hyperbaric facilities are expensive, might lag behind new technological developments and are subject to extensive regulatory processes. A literature review of this issue in the current issue of Diving and Hyperbaric Medicine Journal drew conclusions that are thematically aligned with those arising from this survey.[14]

Conclusions

Defibrillation under hyperbaric conditions is a very rare event, with no clinical cases reported in Australasia and the only in-chamber defibrillation having taken place in an experimental setting. The consensus from this survey is to continue with current protocols and defibrillate only after decompression of the hyperbaric chamber and applying emergency procedures as per individual facilities. Improving preparedness may be improved by performing regular emergency drills, with the addition of *in-situ* simulation techniques to improve fidelity.

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Hyperbaric oxygen treatment and pulmonary air-containing lesions

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Keywords
Bullae; Lungs; Hyperbaric oxygen treatment; Pulmonary barotrauma

Abstract
(Türkmen O, Özkan R, Özgök Kangal K, Dur İnce M, ArslanY. Hyperbaric oxygen treatment and pulmonary air-containing lesions. Diving and Hyperbaric Medicine. 2025 December 20;55(4):343–351. doi: 10.28920/dhm55.4.343-351. PMID: 41364858.)
Introduction: Pulmonary barotrauma is a rare but serious complication of hyperbaric oxygen treatment. Pulmonary air-containing lesions may predispose pulmonary barotrauma by causing air trapping during changes in environmental pressure. This study aimed to investigate whether pulmonary air-containing lesions cause pulmonary barotrauma during hyperbaric oxygen treatment.
Methods: This study retrospectively analysed data from individuals who presented to the Undersea and Hyperbaric Medicine Clinic at Gülhane Training and Research Hospital, Health Sciences University, between 2017 and 2022. The relationship between presence of pulmonary air-containing lesions on computed tomography and pulmonary barotrauma was evaluated.
Results: Gas containing lesions were not associated with an increased risk of pulmonary barotrauma. The incidence of pneumothorax during hyperbaric oxygen was extremely low (0.0059% per session, 0.15% per patient).
Conclusions: Pulmonary air-containing lesions, including bullae, were not associated with an increased risk under standard hyperbaric oxygen treatment protocols. Routine chest computed tomography screening is not warranted due to the low complication rate. Nevertheless, clinical evaluation and informed consent are essential, particularly for patients with underlying lung disease. Further studies are needed to improve risk assessment.

Introduction

Pressure and volume are inversely proportional according to Boyle's law. As the ambient pressure decreases, the volume of gas increases. Consequently, anatomical gas-containing spaces that do not freely equalise with ambient pressure may contract and expand during hyperbaric oxygen (HBO) treatment, depending on the phase of treatment (i.e., compression or decompression). Pulmonary overinflation can lead to disruption of the lung parenchyma, allowing air to enter the pulmonary interstitial spaces. Additionally, damage to the alveolar capillary vasculature may permit air to enter the bloodstream. This may manifest as local pulmonary injury, pneumomediastinum, subcutaneous emphysema, pneumothorax, or life-threatening arterial gas embolism.[1] Such pulmonary barotrauma (PBt) is a rare but serious complication of HBO treatment. Nevertheless, HBO treatment candidates should be screened for PBt risk factors, including chronic obstructive pulmonary disease (COPD), asthma, pulmonary blebs or bullae, and other pulmonary lesions that could impair gas exchange. Screening involves medical history, physical examination, and pulmonary imaging if necessary. HBO treatment should be tailored on a case-by-case basis, considering the individual patient's risk factors.[1]

There are various lesions that may cause air trapping in the lungs. Pulmonary air-containing lesions (PACL) can appear in various forms in the lungs, including cysts, cavities, bullae, blebs, emphysema, pneumatocele, honeycombing and bronchiectasis.[2,3] In addition, the mosaic attenuation pattern, which may indicate obliterative small airways disease rather than a cystic pattern, should also be considered.[2] These pathologies may be a predisposing factor for PBt. In this respect, pre-HBO examination of respiratory system is an important step for hyperbaric physicians. Detailed respiratory medical history, auscultation and chest X-ray has been frequently used for respiratory system screening in patients/divers. In our department, we refer patients who have COPD, restrictive pulmonary disease or suspicious

air-trapping lesion on chest X-ray, to the pulmonology department for further evaluation. Informed consent is obtained from all patients scheduled to receive treatment.

The primary aim of our study was to evaluate whether PACL leads to PBt in HBO treatment. Additionally, we aimed to evaluate the incidence of PBt, determine the sensitivity and specificity of chest radiographs for pulmonary PACL, and examine the relationship between PBt and diseases affecting the lungs.

Methods

The present study was approved by the Scientific Research Ethics Committee of Health Sciences University Gülhane Faculty of Medicine with project/decision number 2022-101, dated 17 March 2022. Additionally, the study was approved by the Scientific Research Platform of the Republic of Turkey Ministry of Health under application number 2022-01-18T14_31_33.

This study was carried out retrospectively at the Department of Undersea and Hyperbaric Medicine and The Department of Pulmonology in Gülhane Training and Research Hospital, Health Sciences University between 1 January 2017 and 31 December 2022. All patients screened before HBO treatment and who underwent HBO treatment in the Department of Undersea and Hyperbaric Medicine were included in this study. The inclusion criteria included having both chest X-ray and thoracic computed tomography (CT) images prior to HBO treatment. No chest CT scans were performed specifically for this study; instead, existing chest CT scans were reviewed.

We recorded age, gender, smoking history, smoking exposure (pack-years), smoking cessation history (years), history of lung disease, history of pneumothorax, chest X-ray findings, chest CT findings, symptoms and findings of PBt during HBO treatment, number of HBO sessions and need for mechanical ventilation during HBO sessions.

Chest X-ray and chest CT images were accessed via our hospital's picture archiving and communication systems. For patients with multiple chest CT scans, the scan closest to the date of admission was evaluated. Chest CT scans with artifacts or a slice thickness greater than 5 mm were excluded. To ensure that potential lesions were not overlooked, CT neck and CT abdomen scans containing lung sections, if available, were also evaluated.

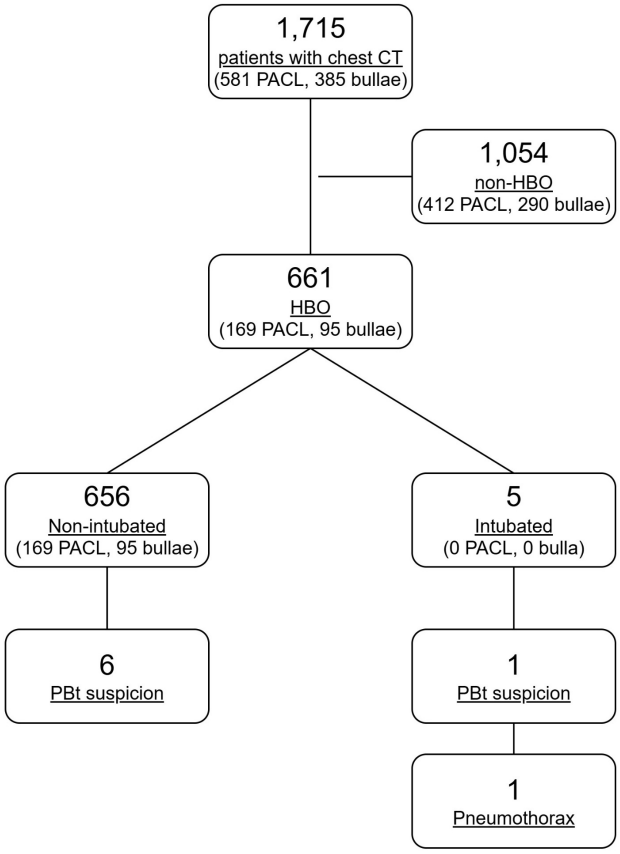
The patients' chest X-ray images and chest CT scans were evaluated for PACL by author OT under the supervision of author YA (Pulmonology Specialist), and the radiology reports of the CT scans were reviewed. In addition, the CT scans of 100 randomly selected patients were independently evaluated for PACL by a blinded radiologist (author MD). Thus, two independent raters (OT/YA and MD) assessed the presence or absence of PACL on CT scans, and inter-rater reliability was calculated to evaluate the consistency between their assessments.

Different materials have characteristic and reproducible CT attenuation values, expressed in Hounsfield units (HU). CT scanners are calibrated to assign water an attenuation value of 0 HU. Materials with higher attenuation, such as soft tissue and bone, yield positive HU values, while materials with lower attenuation, such as air, correspond to negative HU values. Approximate HU values of different tissues on CT imaging are: -1,000 in air, -90 in fat, 0 in water, 30 in white matter, 40 in gray matter, 50 in muscle, and over 1,000 in cortical bone.[4] Normal lung tissue during inspiration exhibits relatively homogeneous low attenuation, with average HU values ranging from approximately -700 to -900.[5] Emphysema is characterised by low attenuation areas, defined using a HU threshold of -950.[6] In this study, for PACL, a threshold value of -950 Hounsfield units (HU) and below was considered for voxels. The type of PACL, their peripheral or central location, the affected lobe, and the maximum diameter of the largest lesion were recorded. Small pure hyperlucency areas were defined as PACL. Mosaic attenuation patterns were also documented. The locations of the lesions were categorised by lobes as the right upper, right middle, right lower, left upper, and left lower lobes.

The following symptoms and signs occurring during or after HBO were classsified as PBt signs and symptoms: chest pain, cough, haemoptysis, substernal pressure, dyspnoea, tachypnoea, asymmetric breathing, and decreased breath sounds. The term 'PBt suspicion' refers to the physician's initial clinical assessment based on the patient's history, presenting symptoms, and physical examination findings. In most cases, the suspicion was based on respiratory symptoms, along with auscultation findings or other clinical clues. This term does not represent a confirmed diagnosis but rather reflects cases in which the physician considered the possibility of PBt.

The statistical analyses were performed using Jamovi version 2.3.28 (The Jamovi Project, Australia). Descriptive statistics derived from the study data included frequencies and percentages for categorical variables, while continuous variables were expressed as mean (standard deviation [SD]) for normally distributed data and as median with interquartile range (IQR) for non-normally distributed data. The normality of continuous variables was assessed with the Shapiro-Wilk test and histograms. For group comparisons, categorical variables were compared with Pearson's chi-square test or Fisher's exact test. The Mann-Whitney U test was used for non-normally distributed continuous variables. For normally distributed ordinal variables, homogeneity of variances was evaluated. If variances were homogeneous, the Student's *t*-test was used, while the Welch test was applied in cases of unequal variances. A P-value of < 0.05 was considered statistically significant.

Figure 1
Flowchart displaying patient selection and outcomes of investigation



Results

Between 1 January 2017, and 31 December 2022, a total of 1,715 individuals met the inclusion criteria. Of these, 661 (38.5%) received HBO treatment in our clinic. Among the remaining individuals, 29 (1.6%) were divers, six (0.3%) were inside tenders, and 1,019 (59.4%) did not receive HBO treatment in our clinic due to contraindications, refusal of treatment, or opting to begin treatment at another HBO centre. Although the original dataset consisted of two groups, the lesion characteristics of those who did not receive HBO treatment (non-HBO group) do not directly align with the core hypothesis or objectives of this study. Therefore, the analysis was focused solely on the group that received HBO treatment (HBO group). This approach was chosen to better understand the potential effects of HBO treatment on PACL and to make original contributions to the relevant literature. The non-HBO group was used solely to calculate the sensitivity of chest X-ray, to determine the etiology of PACL, and to estimate the prevalence of PACL. (Figure 1)

Only 238 (15.1%) of the chest CT scans were performed specifically for the HBO/diving examination. The remaining 1,477 scans (84.9%) were performed during follow-up evaluations in other departments (e.g., COPD screening in

Table 1
Descriptive statistical findings of the patients included in the study; COPD – chronic obstructive pulmonary disease; PACL – pulmonary air containing lesions; PTE – pulmonary thromboembolism

Parameter	Data
Age, median (IQR)	56 (26)
Sessions, median (IQR)	30 (15)
Intubated sessions, mean (SD)	4.8 (2.9)
Sex	
Male	526 (79.6)
Female	135 (20.4)
Smoking	
Smokers, *n* (%)	150 (22.7)
Non-smokers, *n* (%)	511 (77.3)
Current Smokers, *n* (%)	93 (62.0)
Pack years, median (IQR)	30 (23.8)
Quit Smoking, *n* (%)	57 (38)
Years since cessation, median (IQR)	14 (15.0)
Comorbidities (*n* %)	
Asthma	25 (3.8)
COPD	22 (3.3)
History of Covid19 pneumonia	10 (1.5)
Collagen Vascular disease	13 (2.0)
Buerger's disease	10 (1.5)
History of pneumothorax	9 (1.4)
History of tuberculosis	6 (0.9)
History of PTE	4 (0.6)
Sarcoidosis	4 (0.6)
PACL presence and location, *n* (%)	
PACL present	169 (25.5)
PACL absent	492 (74.4)
Right upper lobe	84 (49.7)
Right Middle lobe	14 (8.2)
Right Lower lobe	25 (14.7)
Left Upper lobe	22 (13)
Left Lower lobe	23 (13.6)
Paratracheal	1 (0.5)
PACL classification, *n* (%)	
Bulla, bleb	95 (56.2)
Cyst, emphysema-like changes	56 (33.1)
Bronchiectasis	16 (9.4)
Cavity	1 (0.5)
Paratracheal diverticulum	1 (0.5)

the pulmonology department one year after HBO treatment, or for suspected pulmonary embolism in the emergency department six months prior to HBO treatment).

The median age of the HBO group was 56 years (IQR 26), and 79.6% of the patients were male (Table 1). A total of 16,710 HBO sessions were administered to 661 patients. Among these, 169 patients (25.6%) were diagnosed with PACL and collectively underwent 4,502 HBO treatment sessions. The chest CT sections of some of these patients are shown in Figure 2. Among the HBO group, five patients received a total of 24 sessions while intubated during treatment. Four of these patients were treated for anoxic brain injury and one for carbon monoxide poisoning. Mechanical ventilation was provided using the Draeger Oxylog 1000 (Drägerwerk, Germany) pneumatic ventilator.

Among these patients, the largest PACL diameter was eight cm (Figure 3). This patient had tuberculosis history and COPD diagnosis who completed 29 HBO treatment sessions without any complication. Chest X-ray before HBO treatment was evaluated and the patient was taken into treatment. The patient's bulla was detected on a chest CT scan performed by the pulmonology department one year after HBO treatment.

PATIENTS SUSPECTED OF PULMONARY BAROTRAUMA

Only seven of the 661 patients who received HBO treatment (Table 2) were evaluated for PBt due to the reported possible symptoms and findings. The symptoms were chest pain (n = 1, 14%), dyspnoea (n = 2, 28.5%), haemoptysis (n = 1, 14%), and low oxygen saturation observed at the end of HBO sessions. Regarding the findings, radiological examinations revealed pneumothorax confirmed by chest radiograph in one patient, and localised pulmonary damage detected by high resolution CT (HRCT) in two patients. While pneumothorax is a definitive diagnostic finding, the parenchymal changes observed on CT are non-specific and were therefore categorised as possible PBt findings. The incidence of pneumothorax during HBO was 0.0059% per session and 0.15% per patient. No signs or symptoms consistent with arterial gas embolism were observed in any patients.

Patient 116 reported chest pain after the third session and was assessed with chest CT. This revealed localised pulmonary damage or consolidation in the left upper lobe, likely secondary to PBt (Figure 4). The patient subsequently continued HBO and completed treatment without complications. Pre-HBO spirometry and chest CT evaluations were normal.

Patient 306 reported haemoptysis after the 19th session and was referred to the Pulmonology Department. Chest CT revealed a lesion in the left upper lobe, suggesting localised pulmonary damage around emphysema-like changes (Figure 5). Previous spirometry had shown a restrictive pattern.

Patient 31 who had carbon monoxide intoxication required mechanical ventilation during HBO treatment. This patient developed hypoxaemia in the intensive care unit after the fourth HBO session. A right-sided pneumothorax was detected on the chest X-ray performed following the onset of hypoxaemia, and a chest tube was inserted. Due to the patient's unstable condition, HBO treatment was discontinued. Importantly, the chest CT performed prior to the initiation of HBO treatment was normal.

Table 2

List of patients suspected of pulmonary barotrauma; [a] linear opacity increase surrounding the emphysema-like changes in the left upper lobe (possible haemorrhage?); [b] consolidation in the posterior left upper lobe; CT – computed tomography; Echo – echocardiography; HRCT – high resolution computed tomography; PACL – pulmonary air containing lesions; PBt – pulmonary barotrauma

Patient	Comorbidity	Signs and symptoms	Evaluation of PBt	PBt evaluation result	Incidental PACL
6	–	Chest pain, dyspnoea	Chest radiograph	No pathological findings	Not detected
31	–	Desaturation	Chest radiograph	Pneumothorax	Not detected
116	–	Chest pain	HRCT	Localised pulmonary injury[a]	Not detected
306	Restrictive spirometry	Haemoptysis	HRCT	Localised pulmonary injury[b]	Detected
401	–	Chest pain, dyspnoea	Echo, CT angiography	No pathological findings	Not detected
476	–	Dyspnoea	Chest radiograph	No pathological findings	Not detected
613	Sarcoidosis	Dyspnoea	Physical examination	No pathological findings	Detected

Figure 2
Examples of pulmonary air-containing lesions marked with yellow arrows on CT slices from patients in the present study population

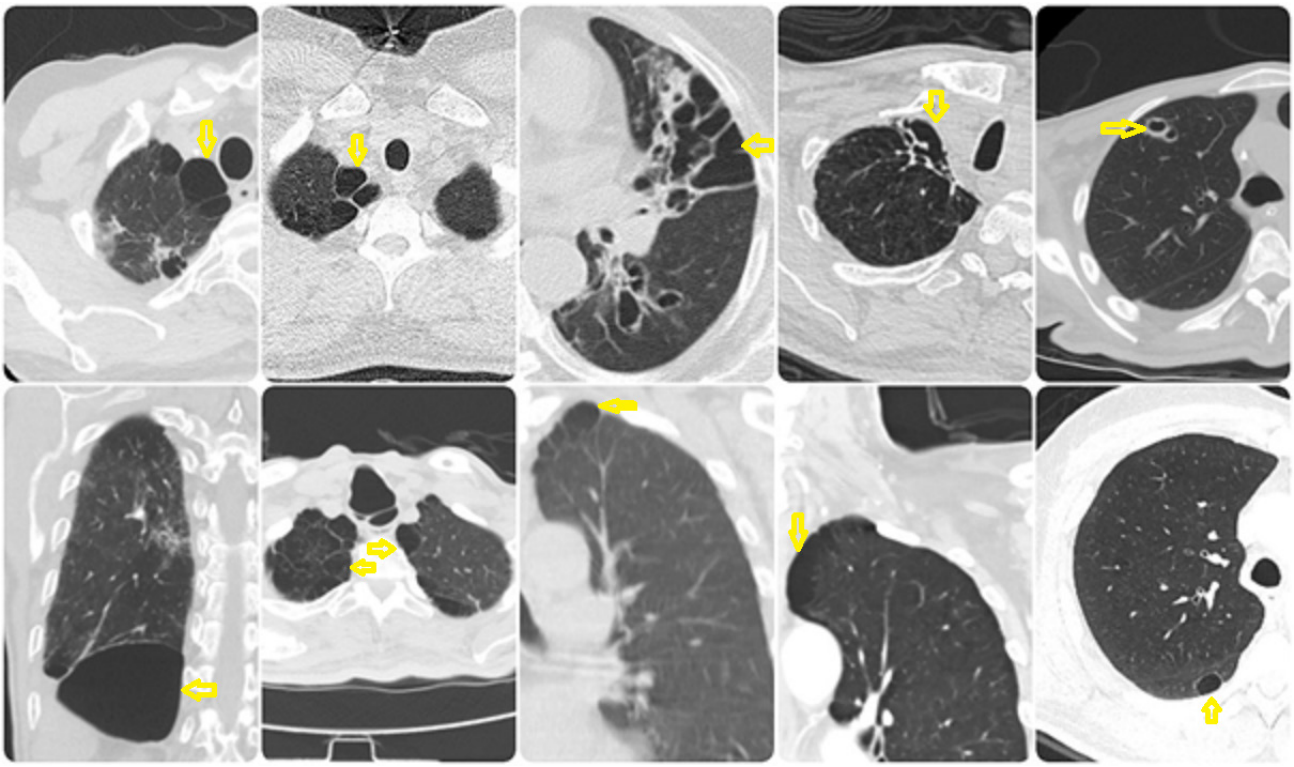
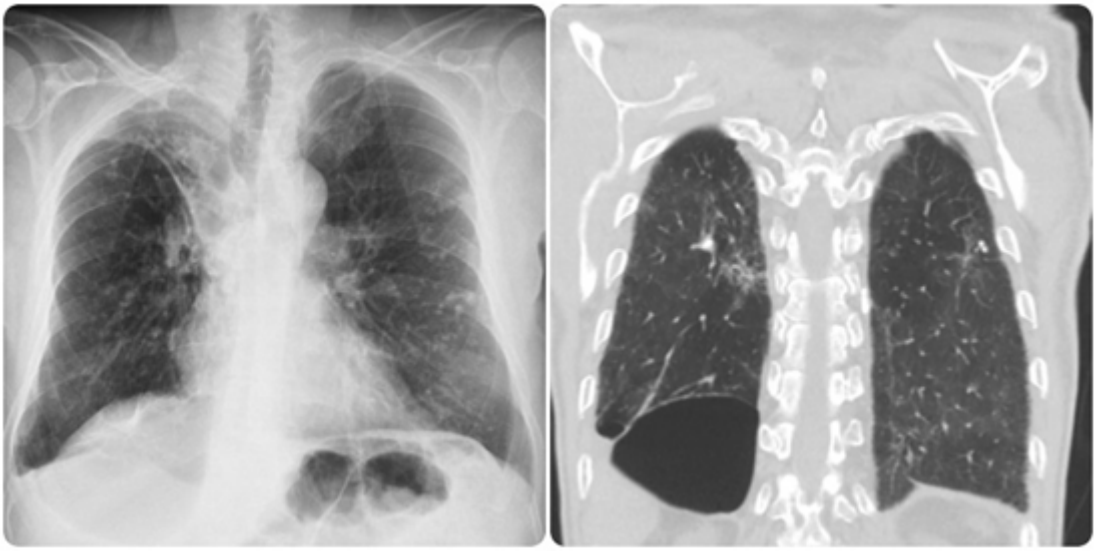


Figure 3
The imaging of a patient who had the largest pulmonary air containing lesion; there is a posteroanterior chest X-ray on the left and a corresponding CT slice on the right



PULMONARY BAROTRAUMA RISK FACTORS

Among the five patients who underwent HBO treatment while intubated, one developed pneumothorax. Of the four patients with sarcoidosis who received treatment, one experienced dyspnoea. In a patient with restrictive spirometry findings, haemoptysis occurred. Apart from these cases, no PBt-related symptoms or signs were observed in 169 patients with PACL. Similarly, among nine patients with a history of pneumothorax, no PBt-related symptoms or signs were detected.

Among these, one patient continued HBO treatment while their pneumothorax was still present, with an indwelling chest tube. Another patient underwent HBO treatment after chest tube removal. The chest tube of this patient

Figure 4
The CT slice of patient 116 with suspected pulmonary barotrauma, where the yellow arrow indicates the consolidated area

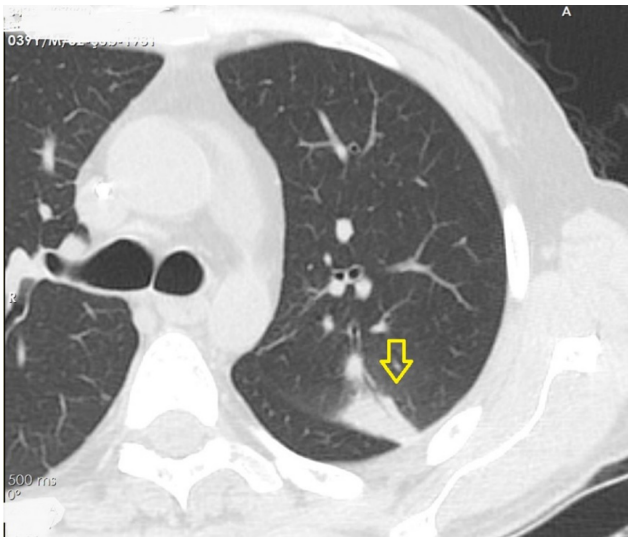
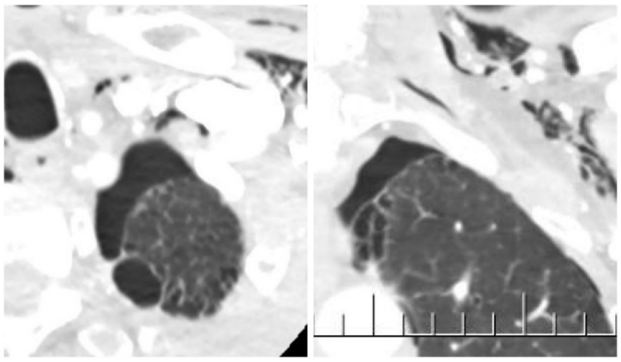
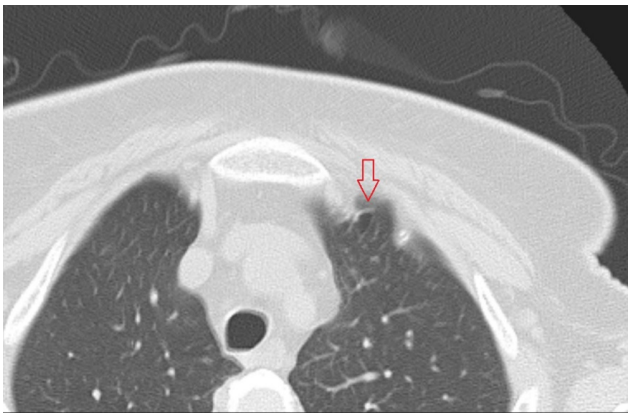


Figure 6
The CT slices of patient with residual pleural air after cardiac surgery who was treated with HBO with the air present



was removed on postoperative day two following coronary artery bypass graft surgery. On postoperative day three, the patient developed central retinal artery occlusion, and HBO was initiated on postoperative day five. A CT angiogram of the head and neck performed two days prior to HBO treatment (Figure 6) revealed a persistent pneumothorax. The patient successfully completed 17 HBO sessions without complications. If the patient's small pneumothorax did not resolve spontaneously within two days, then the patient received HBO treatment while the pneumothorax was still present. The reason it was not identified before HBO treatment was that a posteroanterior chest X-ray was obtained instead of an apicolordotic view, and it was not considered that the chest might be partially visualised in the head and neck CT angiography. The likely reason the pneumothorax did not worsen during treatment is that it was related to residual air following surgery, rather than air leaking from the pleura.

Figure 5
The CT slice of patient 613 with suspected pulmonary barotrauma, where the red arrow indicates a linear opacity increase surrounding the emphysema-like changes



SENSITIVITY AND SPECIFITY

The sensitivity of detailed pulmonary medical history, auscultation, and chest X-ray for PACL was determined to be 34%, while the specificity was 79%.

PACL ETIOLOGY

We analysed the relationship between PACL and conditions such as COPD, asthma, tuberculosis, collagen vascular diseases, sarcoidosis, pulmonary tromboembolism, interstitial lung disease, lung cancer and COVID-19 pneumonia. A significant association was identified between PACL and COPD ($P < 0.001$), tuberculosis ($P < 0.001$), lung cancer ($P < 0.001$), collagen vascular diseaes ($P = 0.012$) and interstitial lung disease ($P = 0.016$). When examining the relationship between PACL and smoking, we found that patients with a history of smoking more than 20 pack-years, current smokers, or those who had quit smoking less than 15 years ago had a significantly higher prevalence of PACL ($P < 0.001$). In the study population, the prevalence of bullae and blebs was 22.4%; this rate was higher among smokers (42%) compared to non-smokers (16.6%).

INTERRATER RELIABILITY

In the analysis conducted between the two rating teams (OT/YA and MD), the agreement rate was 87%, the Cohen's Kappa value was 0.72, and the *P*-value was < 0.0001.

Discussion

PBt during HBO is an extremely rare but serious complication. Although PACLs are theoretically considered to pose a risk for air trapping during changes in ambient pressure, our study demonstrated that these lesions were not observed to be associated with a measurable increased risk of PBt. The incidence of pneumothorax during HBO was found to be

extremely low (0.0059% per session and 0.15% per patient). The sensitivity of detailed pulmonary medical history, auscultation, and chest X-ray for PACL was determined to be 34%, while the specificity was 79%. We did not identify any conditions associated with PBt other than sarcoidosis and intubation.

Expanding alveolar gas can cause localised pulmonary injury and capillary haemorrhage without obvious signs of barotrauma such as pneumomediastinum or arterial gas embolism. Symptoms may include chest pain, cough, and haemoptysis, with supportive care usually being sufficient. However, a detailed neurological examination is essential to rule out subtle cerebral injury from a less apparent arterial gas embolism.[7] We therefore examined suspected PBt cases in detail, as their relatively mild clinical presentations may still conceal serious risks.

The main contributing factor to PBt is environmental pressure change (diving, HBO, flights). Normally, alveolar gas is expelled passively, but any obstruction to airflow can precipitate PBt. In diving PBt most commonly occurs at shallow depths because the proportional change in volume with pressure change is greatest in the shallow depth range.[8]

Another factor is mechanical ventilation and regulator systems, which can directly affect airway pressure. PBt can occur even in the absence of underlying air-trapping lesions in the lungs if the transpulmonary pressure exceeds the sustainable tissue pressure .[9] Transpulmonary pressures of approximately 9.47 to 10.93 kPa can cause alveolar rupture.[10] In a study by Örmeci et al., that measured regulator pressures, it was shown that during expiration, the air pressure inside an HBO mask could increase to 2.24 kPa, and up to 4.63 kPa during forced expiration.[11] Although this pressure increase is not high enough to directly cause alveolar rupture, it may lower the threshold for PBt. In hyperbaric environments, patients undergoing ventilation, especially during the decompression phase, face a risk of PBt if positive pressure exceeds 3.92 kPa;[12] in mechanically ventilated HBO, both airway pressure and ambient pressure vary. Toklu et al., reported PBt in two intubated patients among 98 HBO centres.[8] Bessereau et al., in their study involving 150 intubated patients undergoing HBO with a pneumatic ventilator, reported no pulmonary side effects other than patient-ventilator asynchrony.[13] Stahl et al., reported that airway pressure delivered by the ventilator decreases with increasing ambient pressure.[14] In cases where mechanical ventilation is applied in a normobaric environment, barotrauma is observed with a frequency of 4–15%.[15] In our study, no signs or symptoms were observed in the intubated patient who developed pneumothorax during the decompression phase, or after the HBO session. The onset of symptoms following the patient's transfer to the intensive care unit suggests that the pneumothorax may be related to manual ventilation (ambu-bagging) during transfer or to the mechanical ventilator used in the ICU.

Another factor is lesions that can cause air trapping in the lungs. Whether PACL lead to PBt depends on whether air can freely enter and exit these lesions. In bronchiectasis, the cysts communicate freely with the airways, whereas this is not necessarily the case for emphysema-like changes.[16] In a study by Morgan et al., only one out of 23 bullae contributed more than 10% to ventilation. The remaining 22 bullae either contributed minimally or, in some cases, not at all. Additionally, the volume of air within the bullae showed very little change during respiration.[17] In a study conducted by Pride et al., 13 out of 14 bullae (92%) contributed minimally or not at all to ventilation.[18] Sources on pathology state that bullae are in free communication with the underlying lung parenchyma; however, it is important to note that these tests were conducted *in vivo*.[19] Most bullae are poorly ventilated, a minority are either well ventilated or not ventilated at all.

These three issues should be discussed together. When air exchange in PACL is weak, the bulla will take in air to equalise its internal pressure with the surrounding pressure as ambient pressure increases. Conversely, as ambient pressure decreases, the bulla will release air to balance the increased internal pressure. If the pressure change occurs too quickly for the air to exit, the air inside the bulla will expand, and PBt will occur once the pressure exceeds the sustainable tissue pressure.

When PACL are well ventilated, the risk of PBt will be low due to effective air exchange. If there is no air exchange in the PACL, the size of the lesions will decrease with pressure increases (HBO compression phase) and return to their original size as pressure decreases (HBO decompression phase), in accordance with Boyle's Law. In this case, PACL will not pose a high risk for PBt, except in hypobaric environments such as air travel.[20,21]

In our study, among the 169 patients with PACL, 1.1% ($n = 2$) were suspected of having PBt, but no pneumothorax was observed. Although these lesions were not observed to be associated with an increased risk of PBT, it is important to acknowledge existing case reports and case series suggesting an elevated risk, warranting a careful, informed consent-based discussion with patients who have pulmonary pathology.[22,23] Although the aeration in emphysema-like changes was weak, it was considered sufficient to accommodate changes in transpulmonary pressure. In other words, the transpulmonary pressure generated allowed air to escape without exceeding sustainable tissue pressure. Although the high altitude of our department (938 m, Ankara) theoretically increases the risk of PBt, the fact that many patients with PACL have been successfully treated without complications suggests that the disease carries a lower risk of PBt at altitude than previously thought. Indeed, in the survey conducted by Toklu et al., it was reported that 66% of HBO centres have admitted patients with bullous lesions for treatment,[8] and our study has supported the approach of these centers.

Brenna et al., reported one case of PBt in their single-centre experience involving 2,250 patients and 62,040 HBO sessions. They documented a PBt incidence of 0.0016% per session or 0.044% per patient.[24] Similarly, in a survey study by Toklu et al., encompassing approximately two million sessions across 98 centers, nine cases of PBt were reported in seven centres, with a session-based PBt incidence of 0.00045%.[8] In our study, the PBt incidence was 0.0059% per session and 0.15% per patient. Assuming that chest CT imaging was also performed to detect lung diseases, we can say that the higher incidence in our study compared to other studies is due to our focus on the patient group with chest CT imaging.

There are various concerns regarding the sensitivity of chest radiography in detecting PACL. The sensitivity of chest radiography for moderate to severe emphysema is 41% .[25] In the study by Wingelaar et al., the sensitivity of chest radiography for bullous lesions was 0%, and the specificity was 90%.[26] In emphysema-like changes, lung CT has a sensitivity of 84% and a specificity of 100%.[27] In our study, the sensitivity of a detailed pulmonary medical history, auscultation, and chest X-ray for detecting PACL was found to be 34%, while the specificity was 79%. This highlights the use of chest CT in respiratory system screenings prior to HBO treatment or diving. However, due to radiation exposure, cost, limited availability, and potential for overdiagnosis, CT is not always feasible for routine screening. This situation raises questions about the adequacy of screening patients using only auscultation and chest radiography, particularly for identifying air-trapping lesions that may lead to PBt. While evidence remains insufficient regarding the effectiveness of chest radiographs in detecting such lesions, the role of advanced imaging techniques like CT has become more prominent in recent debates about PACL screening during fitness-to-dive and pre-HBO treatment examinations.

When reviewing studies on the frequency of bullous lesions, the prevalence in the general population ranges from 4.7% to 33.8%.[28–30] In the diver population, it ranges from 1.9% to 7.8%.[26,31,32] In our study, the prevalence of bullous lesions was 22.4%, which was higher than in five of the six studies mentioned above. This may be explained by the retrospective nature of our study, in which the chest CT scans were performed on selected risk groups for pulmonary disease screening.

Brenna et al., identified some low-risk factors for PBt, including the absence of findings, symptoms, or history of conditions such as COPD, asthma, pulmonary fibrosis, sarcoidosis, pneumothorax, and acute respiratory distress syndrome.[24] In our study, apart from intubation and sarcoidosis, we did not identify any other associated conditions. We also could not determine any relationship between PBt and PACL or a history of pneumothorax.

The first limitation of our study is that, for some patients, follow-up CT scans were not performed in close temporal proximity to their HBO treatment. The second limitation is the retrospective design of the study, which may have resulted in incomplete data collection. Additionally, only patients treated at our centre were included; therefore, the generalisability of our findings is limited. However, the inclusion of a large cohort from a single center can also be considered a strength of the study.

Conclusions

PBt during HBO treatment remains an extremely rare complication. Our findings suggest that PACL, including bullae, were not observed to be associated with an increased risk of PBt under standard HBO protocols. The slow and controlled decompression profile of HBO likely mitigates the risk, even in the presence of PACL. Although chest CT remains the most sensitive tool for identifying such lesions, its routine use in screening may not be justified given the low incidence of complications. Careful clinical evaluation, including detailed history and physical examination, remains essential. Patients with underlying pulmonary disease should be appropriately counselled, and informed consent obtained. Further prospective studies are warranted to refine risk stratification and screening strategies for HBO candidates.

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Quality of reporting in hyperbaric medicine clinical trials: a cross-sectional study

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Keywords
CONSORT; EQUATOR; Evidence-based medicine; Hyperbaric oxygenation; Reporting quality; STROBE

Abstract
(Yoo Y, Cléroux A, Pollock NW, Boet S. Quality of reporting in hyperbaric medicine clinical trials: a cross-sectional study. Diving and Hyperbaric Medicine. 2025 December 20;55(4):352–368. doi: 10.28920/dhm55.4.352-368. PMID: 41364859.)
Introduction: Research in hyperbaric oxygen (HBO) medicine is growing, but the quality of HBO studies is variable. Low study quality may compromise evidence-based decision-making and clinical translation.
Methods: This cross-sectional study examined the adherence of 50 randomly selected HBO clinical trials (25 randomised controlled trials [RCTs] and 25 observational studies) to relevant core reporting guidelines: consolidated standards of reporting trials (CONSORT), non-pharmacologic treatments (NPT), and strengthening the reporting of observational studies in epidemiology (STROBE). Studies published in peer-reviewed journals between January 2018 and May 2023 and indexed on PubMed were analysed. Reporting quality was classified as 'excellent' (> 85% of guideline items adequately reported), 'good' (50–85%), or 'poor' (< 50%).
Results: The sample represented 29% of RCTs and 16% of observational studies for the timeframe assessed. No study was rated as 'excellent' for completeness, 28 (56%) were rated as 'good', and 22 (44%) as 'poor'. In RCTs, only one study (4%) adequately reported protocol adherence and eight studies (32%) reported blinding procedures. The NPT checklist showed that key items, including care provider adherence (0 studies) and participant adherence (one study; 4%), were frequently not reported. For observational studies, basic design elements were adequately reported, but with significant gaps in bias management (nine studies; 36%) and missing data handling (13 studies; 52%). Only six studies (12%) mentioned the use of reporting guidelines.
Conclusions: Our results showed that quality of reporting of HBO studies is suboptimal. These findings highlight the need for increased awareness and implementation of reporting guidelines, as well as the potential development of HBO-specific guidelines.

Introduction

Clinical decision-making and public health policy should rely on scientific evidence.[1] Randomised controlled trials (RCTs) are considered to be the most robust study design to inform clinical decisions, but other designs such as cohort, case-control, or cross-sectional also provide evidence.[2] Even when well-designed clinical trials are conducted, evidence can only be translated into practice when reporting of study methods and results is appropriate.[3] Unfortunately, poor reporting is common across many fields.[3–6] Poor quality reporting can lead to bias and research waste, and can impair critical appraisal and appropriate replication of studies, compromise future evidence, and ultimately decrease the value and impact of research.[7]

Since the mid-2000s, an international initiative - the enhancing the quality and transparency of health research (EQUATOR) network – has worked to improve the reporting of healthcare research and aid in research optimisation.[8] More than 550 reporting guidelines have been developed to guide authors, editors, and peer-reviewers to improve reporting quality.[9] Some of the guidelines are dedicated to specific study types, such as consolidated standards of reporting trials (CONSORT) for RCTs,[10] preferred reporting items for systematic reviews and meta-analyses (PRISMA)

for systematic reviews,[11] and strengthening the reporting of observational studies in epidemiology (STROBE) for observational studies.[12] Others are specific to a clinical area with specialised considerations, such as surgery,[13] interventional radiology,[14] or medical education.[12,15] Recent evidence demonstrates that reporting guidelines associated with adoption from journals may improve the reporting quality.[16,17]

Hyperbaric oxygen therapy (HBO) has been used for a number of elective and urgent indications for decades, and there has been a growing number of publications in the field.[18] HBO is a complex intervention with special considerations to report, such as pressure levels, session durations, frequencies, types of healthcare professionals in charge, and equipment type (e.g., monoplace or multiplace chambers, masks or head tents). Poor reporting of clinical HBO trials has been a point of concern in a number of previous systematic reviews.[19–22] However, most of the included studies of those systematic reviews considered older trials, some published before the development and implementation of current best practice reporting guidelines. There is a need to analyse the quality of reporting in recent HBO trials. In addition, no reporting guidelines specific to hyperbaric medicine have been incorporated as part of the EQUATOR Network. Given the evidence of poor reporting across healthcare disciplines, the implications for patient care, and the existence of solutions to improve reporting, we elected to explore the comtemporary quality of reporting in hyperbaric medicine trials.

Methods

This study was reported in accordance with the STROBE guidelines for cross-sectional studies.

Ethics review was unnecessary given that our study only involved publicly available data. We addressed the following research question: '*How do hyperbaric medicine clinical studies published in peer-reviewed journals currently adhere to core reporting standards*?' Our primary outcome was completeness of reporting as assessed by the core set of reporting standards suggested by the EQUATOR Network, i.e., CONSORT and non-pharmacologic treatments (NPT) or STROBE checklists, as appropriate. We did not assign weights to perceived deficiencies.

We included a sample of clinical trials in which HBO was the primary intervention investigated. Inclusion and exclusion criteria are summarised in Table 1. We elected to search references after 2018 because we were interested in recent adherence to reporting guidelines relevant to the field of HBO published in 2017.[23]

The search strategy was designed by the research team with the help from an information specialist (VL). We chose to focus only on PubMed since it is free and readily accessible to most clinicians and researchers around the world, which means that the quality of reporting of these studies is most prone to impact practice. The search strategy was peer-reviewed by a second information specialist using the peer review of electronic search strategies (PRESS) tool.[24] We conducted the search on 19 May 2023. We used observational wording adapted from the SIGN Observational Studies filter[25] and the RCT wording adapted from Cochrane search strategies for identifying randomised trials in PubMed: sensitivity- and precision-maximising version.[26] For logistical reasons and ease, we chose to conduct two separate searches, one for RCT and another for observational studies, and then import all references into a single distiller project for screening.

For RCT: ((((“*hyperbaric oxygenation*”[MeSH Terms] OR (“*hyperbaric**”[Title/Abstract] OR “*hyper baric**”[Title/Abstract])) AND (“*randomized controlled trial*”[Publication Type] OR “*controlled clinical trial*”[Publication Type] OR “*clinical trials as topic*”[MeSH Terms:noexp] OR (“*Randomized*”[Title/Abstract] OR “*randomised*”[Title/Abstract] OR “*randomly*”[Title/Abstract] OR “*placebo*”[Title/Abstract] OR “*Trial*”[Title]))) NOT

Table 1
Inclusion and exclusion criteria; HBO – hyperbaric oxygen treatment; RCT – randomised controlled trial

Parameter	Inclusion	Exclusion
Population	Human	Animal, cell, healthy volunteer, simulated human
Intervention	HBO as the main variable investigated	HBO as part of the intervention but not the main variable tested
Design	Original research: RCTs, observational studies (cohort, case-control studies, cross-sectional studies)	Editorials, commentary, letters to the editor, reviews, conference abstracts, preclinical studies, and pre-print publications
Recency of publication	Appearing in print or final form e-publications from 1 Jan 2018 through 19 May 2023	Publications appearing before 1 Jan 2018 or after 19 May 2023
Language	English	Non-English

("*animals*"[MeSH Terms] NOT "*humans*"[MeSH Terms])) AND 2018/01/01:3000/12/12[Date – Publication]) AND (english[Filter])

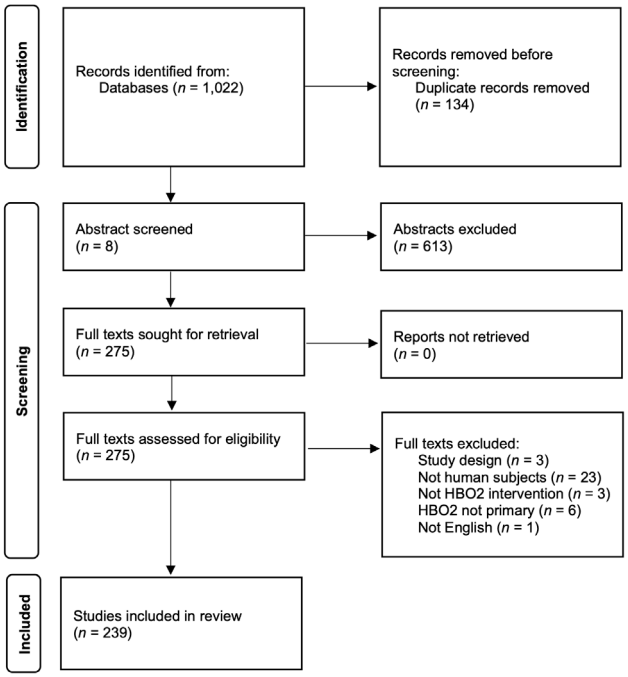
For observational studies: ((("*hyperbaric oxygenation*"[MeSH Terms] OR "*hyperbaric**"[Title/Abstract] OR "*hyper baric**"[Title/Abstract]) AND ("*cohort studies*"[MeSH Terms] OR "*cross sectional studies*"[MeSH Terms] OR "*case control studies*"[MeSH Terms] OR "*epidemiologic studies*"[MeSH Terms:noexp] OR "*case control*"[Title/Abstract] OR "*cohort study*"[Title/Abstract] OR "*cohort studies*"[Title/Abstract] OR "*cohort analy**"[Title/Abstract] OR "*follow up study*"[Title/Abstract] OR "*follow up studies*"[Title/Abstract] OR "*observational study*"[Title/Abstract] OR "*observational studies*"[Title/Abstract] OR "*longitudinal*"[Title/Abstract] OR "*retrospective*"[Title/Abstract] OR "*cross sectional*"[Title/Abstract])) NOT ("*animals*"[MeSH Terms] NOT "*humans*"[MeSH Terms])) AND ((2018/1/1:3000/12/12[pdat]) AND (english[Filter])

Identified articles were uploaded to DistillerSR (Evidence Partners, Ottawa, Canada), and eligible studies were identified after a two-step screening process, completed in duplicate by independent reviewers. Disagreements were resolved by consensus, or a third party as needed. To ensure feasibility, we elected to analyse a random sample of relevant studies, 25 RCTs (CONSORT) and 25 observational studies (i.e., cohort, case-control studies, or cross-sectional) (STROBE). The number was chosen as a convenient and manageable sample that was expected to capture a range of clinical indications, countries, and journals. We proceeded to the random selection of the sample from the eligible references identified after the two-step screening process described above. We exported the list of eligible studies from Distiller to Microsoft Excel (version 16.36, Microsoft Corporation, Redmond, Washington, USA) and then associated a random number (using the function "= *RAND()*") to each. The records were sorted by ascending random numbers and the first 25 for each included design (i.e., RCT or observational) were selected.

Data extraction of the sample set was conducted in duplicate by a pair of independent reviewers. Any disagreements between the two reviewers were resolved by discussion and with a third reviewer as necessary. We developed a coding manual to calibrate agreement between reviewers. To optimise quality, we initially pilot-tested the data extraction form and the coding manual on a set of articles. This pilot enabled refinement of the data extraction form and coding manual to enhance inter-assessor consistency and reliability. Subsequently, the reviewers independently performed the remaining data extraction in duplicate.

Data to be collected included: publication details (e.g., first author, year, country of data collection, journal), study design, and use of reporting guidelines. Each item of CONSORT and NPT or STROBE checklists was rated as 'adequately reported,' 'incompletely reported,' 'not reported,' or 'not applicable,' with descriptions provided by reviewers when 'incompletely reported.' We identified high-income countries (defined by the World Bank as a country with high gross national income per capita) as part of the data extraction.

Figure 1
Study selection process chart;[11] this diagram outlines the steps taken to identify, screen, and select studies for this review on HBO



Completeness and transparency of reporting in HBO for RCTs and observational studies were measured with the CONSORT, NPT, or STROBE guidelines. We calculated for each study the completeness of reporting according to the following: 'excellent' (> 85% of items rated as 'adequately reported'), 'good' (50–85% of items rated as 'adequately reported'), and 'poor' (< 50% rated as 'adequately reported'). This approach aligned with previous research that assessed the reporting quality of randomised controlled trials of massage using a similar descriptive categorisation method.[27] We also categorised and reported the frequency and nature of descriptions for items rated 'incompletely reported' or 'not reported'. Results were descriptively summarised using Excel (version 16.36, Microsoft Corporation, Redmond Washington, USA).

Results

The literature search yielded 1,022 reports from which 134 duplicates were removed. After assessment for eligibility, 239 references met our eligibility criteria for inclusion

(Figure 1). The selected sample of 50 papers[28–77] represented 29% of the RCTs and 16% of the observational studies published in the five-year timeframe assessed.

Tables 2 and 3 present the characteristics of the 50 studies, including 25 RCTs and 25 observational studies, with 27 studies (54%) from high-income countries. The studies covered a wide range of clinical indications, with sudden sensorineural hearing loss being the most frequently investigated condition. The studies were grouped by study design, with RCTs listed first, followed by observational studies. Within each study design category, studies were further ordered alphabetically by the last name of the first author, and then by year of publication for studies with the same first author. Among the 25 RCTs, only three studies explicitly mentioned using CONSORT, one study reported using another guideline, and 21 studies did not mention any structured reporting guideline. Similarly, in the 25 observational studies, one study reported using STROBE, and one study used a different guideline. There was no clear relationship between NPT adherence and the year of publication, indicating that even recent studies did not consistently follow NPT guidelines.

Overall, we found that reporting of clinical studies in HBO was suboptimal. Reporting guidelines were mentioned only in 14% of the analysed studies. None of the sampled studies correctly reported all the items recommended.

For the 50 studies assessed across all reporting items (CONSORT, NPT, or STROBE), none were rated as 'excellent', 28 (56%) were rated as 'good', and 22 (44%) were rated as 'poor'. Among the assessed studies, the completeness of reporting ranged from 35% (i.e., only 35% of the required items were adequately reported) to 82%.

Tables 4 and 5 present an aggregated view of reporting quality, showing the number and percentage of studies that adequately, incompletely, not applicable, or did not report specific checklist items across all assessed randomised controlled trials and observational studies, respectively.

Figures 2 and 3 illustrate the completeness of reporting for each individual study by showing the percentage of items adequately, incompletely, not applicable, or not reported for every single randomised controlled trial and observational study in the sample.

When analysed with CONSORT, 15 RCT studies (60%) were rated as 'good' and 10 studies (40%) as 'poor'. For each study assessed, completeness ranged from 32% to 84%. The basic study elements like structured abstracts (24 studies; 96%), scientific background (23 studies; 92%) and objectives were generally complete, but key methodological details were often incomplete or missing. For example, only one study (4%) reported changes after trial commencement. Blinding information was poorly reported, with only eight studies (32%) adequately describing who was blinded and how. Interim analyses and protocol changes were documented in two studies (8%) and one study (4%), respectively.

When analysed with NPT, the completeness of reporting of RCTs was mostly poor. For each study assessed, completeness ranged from 11% to 33%. The most adequately reported item was detailed intervention descriptions (item 5a NPT, 5b NPT, 5c NPT), but other important NPT items were often missing. Care provider adherence to protocols was not reported in any studies (0%), and participant adherence was reported in one study (4%). Additionally, clustering by care providers (Items 7a NPT and 12a NPT) and care provider descriptions (Item 15 NPT) were often marked as 'not applicable'.

No observational study were rated as 'excellent' (> 85%). Most studies were rated as 'good' (19 studies; 76%), with 6 (24%) rated as 'poor'. Completeness ranged from 41% to 82% depending on the considered item. Similar to patterns observed with CONSORT and NPT items, basic study descriptions were generally adequately reported, while methodological details were often missing. The most adequately reported items were objectives, study design, and item data sources/measurement (items 3, 4, and 8), each with 100% completeness. Critical gaps were identified in bias management (Item 9; nine studies, 36%) and confounding control (Item 12a; 12 studies, 48%). Sensitivity analyses (Item 12e) were rarely addressed, with only one study (4%) reporting them. Key variables and confounders (Item 7) were defined in five studies (20%), and flow diagrams (Item 13c) were used in seven studies (28%).

Discussion

Based on the relevant reporting guidelines (CONSORT, NPT, or STROBE), our study indicates an overall moderate quality of reporting in both RCTs and observational studies investigating HBO. In addition, we identified significant variability in reporting quality across studies assessed from both types. No study reached 'excellent' reporting completeness, just over half fell within the 'good' category, and almost half were rated as 'poor'.

Reporting the income level of countries where patients are recruited was included with the intention to help assess generalisability, equity, and applicability of research findings across different health system contexts. Its relevance is in demonstrating that suboptimal reporting is a widespread issue in the field, even in studies from well-resourced countries where research and publication support is generally more available.

Table 2
Characteristics of included randomised controlled trials

Reference Year	Country of data collection	Clinical indication
28 – 2023	Israel	Fibromyalgia in patients with a history of traumatic brain injury
29 – 2020	Mexico	ST-elevation myocardial infarction
30 – 2022	Israel	COVID19
31 – 2022	Italy	Sudden sensorineural hearing loss
32 – 2022	China	Dog bites
33 – 2019	United States	Persistent post-concussive symptoms
34 – 2022	Greece	Idiopathic sudden sensorineural hearing loss
35 – 2019	Argentina	Type 2 diabetes mellitus
36 – 2022	China	Sudden sensorineural hearing loss
37 – 2022	Israel	Persistent post-concussion syndrome in children following traumatic brain injury
38 – 2023	Sweden	Post-COVID condition
39 – 2021	United States	Central airway stenosis after lung transplantation
40 – 2018	China	Slow coronary flow in patients diagnosed with coronary artery angiography
41 – 2019	China	Traumatic brain injury
42 – 2019	United States	Mild traumatic brain injury
43 – 2021	China	Depression
44 – 2019	Malaysia	Adjunctive treatment for non-healing diabetic foot ulcers
45 – 2019	Sweden, Norway, Denmark, Finland	Late radiation cystitis
46 – 2018	Australia	Chronic venous leg ulcers
47 – 2020	China	Idiopathic sudden sensorineural hearing loss
48 – 2020	Australia	Insulin sensitivity in men with type 2 diabetes mellitus
49 – 2022	China	Cerebral nerve function in comprehensive treatment of poststroke depression
50 – 2018	China	Epithelial-to-mesenchymal transition phenomenon in keloid tissue
51 – 2021	China	Advanced esophageal cancer using a combination of a 125I particle-integrated esophageal covered stent and hyperbaric oxygen
52 – 2022	Israel	Post-COVID condition

Table 3
Characteristics of included observational studies

Reference Year	Country of data collection	Clinical indication
53 – 2021	South Korea	Idiopathic sudden sensorineural hearing loss
54 – 2023	Croatia	Sudden sensorineural hearing loss
55 – 2022	Turkey	Carbon monoxide poisoning
56 – 2021	France	Ventilator-acquired pneumonia
57 – 2018	Taiwan	Endothelial progenitor cells with acute non-cardioembolic stroke
58 – 2018	United States	Sternal wound infections
59 – 2022	Turkey	Retina and choroid tissue
60 – 2018	Turkey	Diabetic foot ulcer
61 – 2019	Israel	Pulmonary oxygen toxicity
62 – 2020	United States	Chronic wounds
63 – 2019	Turkey	Central macular thickness, central choroidal thickness and the stage of retinopathy in patients with type 2 diabetes mellitus
64 – 2020	Turkey	Idiopathic sudden sensorineural hearing loss
65 – 2020	South Korea	Composite grafting for amputated fingertip injury
66 – 2021	South Korea	24-hour post-carbon monoxide poisoning
67 – 2021	Switzerland, France	Frostbite stage 3, 4
68 – 2021	Qatar	Avascular necrosis
69 – 2019	Japan	Carbon monoxide poisoning
70 – 2018	Italy	Autism
71 – 2020	Italy, Bosnia, Herzegovina, Spain, and Bulgaria	Aseptic tibial nonunion
72 – 2018	Turkey	Erectile dysfunction
73 – 2018	Australia and New Zealand	Central nervous system oxygen toxicity
74 – 2018	China	Sudden sensorineural hearing loss
75 – 2020	Japan	Idiopathic sudden sensorineural hearing loss
76 – 2021	Canada	Left ventricular ejection fraction
77 – 2018	Taiwan	Tuberculosis reactivation

Table 4

Completeness of reporting for RCTs investigating HBO based on CONSORT and non-pharmacologic treatments (NPT); items followed by 'NPT' indicate an extension of the CONSORT guidelines specific to non-pharmacologic treatments; AR – item adequately reported; IR – item incompletely reported; NR – item not reported; NA – item not applicable

Section/Topic	Item number and description	AR *n* (%)	IR *n* (%)	NR *n* (%)	NA *n* (%)
Title and abstract	1a. Identification as a randomised trial in the title	11 (44%)	0 (0%)	14 (56%)	0 (0%)
	1b. Structured summary of trial design, methods, results, and conclusions (for specific guidance see CONSORT for abstracts)	24 (96%)	1 (4%)	0 (0%)	0 (0%)
	1b NPT. When applicable, report eligibility criteria for centers where the intervention is performed and for care providers	0 (0%)	0 (0%)	8 (32%)	17 (68%)
	1b NPT. Report any important changes to the intervention delivered from what was planned	0 (0%)	0 (0%)	10 (40%)	15 (60%)
Introduction					
Background and objectives	2a. Scientific background and explanation of rationale	23 (92%)	2 (8%)	0 (0%)	0 (0%)
	2b. Specific objectives or hypotheses	25 (100%)	0 (0%)	0 (0%)	0 (0%)
Methods					
Trial design	3a. Description of trial design (such as parallel, factorial) including allocation ratio	18 (72%)	7 (28%)	0 (0%)	0 (0%)
	3a NPT. When applicable, how care providers were allocated to each trial group	0 (0%)	0 (0%)	8 (32%)	17 (68%)
	3b. Important changes to methods after trial commencement (such as eligibility criteria), with reasons	1 (4%)	0 (0%)	8 (32%)	16 (64%)
Participants	4a. Eligibility criteria for participants	23 (92%)	2 (8%)	0 (0%)	0 (0%)
	4a NPT. When applicable, eligibility criteria for centers and for care providers	0 (0%)	1 (4%)	8 (32%)	16 (64%)
	4b. Settings and locations where the data were collected	22 (88%)	2 (8%)	1 (4%)	0 (0%)
Interventions	5. Interventions for each group with sufficient details to allow replication, including how and when they were actually administered	24 (96%)	1 (4%)	0 (0%)	0 (0%)
	5a NPT. Precise details of both the experimental treatment and comparator	25 (100%)	0 (0%)	0 (0%)	0 (0%)
	5b NPT. Description of the different components of the interventions and, when applicable, description of the procedure for tailoring the interventions to individual participants	22 (88%)	0 (0%)	2 (8%)	1 (4%)
	5c NPT. Details of whether and how the interventions were standardised	24 (96%)	0 (0%)	0 (0%)	1 (4%)
	5d NPT. Details of whether and how adherence of care providers to the protocol was assessed or enhanced	0 (0%)	0 (0%)	6 (24%)	19 (76%)
	5e NPT. Details of whether and how adherence of participants to interventions was assessed or enhanced	0 (0%)	1 (4%)	24 (96%)	0 (0%)

Table 4 continued.

Outcomes	6a. Completely defined pre-specified primary and secondary outcome measures, including how and when they were assessed	13 (52%)	12 (48%)	0 (0%)	0 (0%)
	6b. Any changes to trial outcomes after the trial commenced, with reasons	1 (4%)	0 (0%)	7 (28%)	17 (68%)
Sample size	7a. How sample size was determined	11 (44%)	0 (0%)	14 (56%)	0 (0%)
	7a NPT. When applicable, details of whether and how the clustering by care providers or centers was addressed	0 (0%)	0 (0%)	5 (20%)	20 (80%)
	7b. When applicable, explanation of any interim analyses and stopping guidelines	2 (8%)	0 (0%)	13 (52%)	10 (40%)
Sequence generation	8a. Method used to generate the random allocation sequence	18 (72%)	0 (0%)	7 (28%)	0 (0%)
	8b. Type of randomisation; details of any restriction (such as blocking and block size)	10 (40%)	9 (36%)	6 (24%)	0 (0%)
Allocation concealment	9. Mechanism used to implement the random allocation sequence (such as sequentially numbered containers), describing any steps taken to conceal the sequence until interventions were assigned	13 (52%)	4 (16%)	8 (32%)	0 (0%)
Implementation	10. Who generated the random allocation sequence, who enrolled participants, and who assigned participants to interventions	8 (32%)	2 (8%)	15 (60%)	0 (0%)
Blinding	11a. If done, who was blinded after assignment to interventions (for example, participants, care providers, those assessing outcomes) and how	8 (32%)	6 (24%)	10 (40%)	1 (4%)
	11a NPT. If done, who was blinded after assignment to interventions (e.g., participants, care providers, those administering co-interventions, those assessing outcomes) and how	8 (32%)	6 (24%)	7 (28%)	4 (16%)
	11b. If relevant, description of the similarity of interventions	19 (76%)	0 (0%)	0 (0%)	6 (24%)
	11c NPT. If blinding was not possible, description of any attempts to limit bias	0 (0%)	2 (8%)	12 (48%)	11 (44%)
Statistical methods	12a. Statistical methods used to compare groups for primary and secondary outcomes	25 (100%)	0 (0%)	0 (0%)	0 (0%)
	12a NPT. When applicable, details of whether and how the clustering by care providers or centers was addressed	0 (0%)	0 (0%)	2 (8%)	23 (92%)
	12b. Methods for additional analyses, such as subgroup analyses and adjusted analyses	10 (40%)	0 (0%)	5 (20%)	10 (40%)
Results					
Participant flow (a diagram is strongly recommended)	13a. For each group, the numbers of participants who were randomly assigned, received intended treatment, and were analysed for the primary outcome	25 (100%)	0 (0%)	0 (0%)	0 (0%)
	13a NPT. The number of care providers or centers performing the intervention in each group and the number of patients treated by each care provider or in each center	0 (0%)	1 (4%)	6 (24%)	18 (72%)
	13b. For each group, losses and exclusions after randomisation, together with reasons	18 (72%)	0 (0%)	3 (12%)	4 (16%)
	13c NPT. For each group, the delay between randomisation and the initiation of the intervention	4 (16%)	1 (4%)	20 (80%)	0 (0%)

Table 4 continued.

Recruitment	14a. Dates defining the periods of recruitment and follow-up	20 (80%)	0 (0%)	5 (20%)	0 (0%)
	14b. Why the trial ended or was stopped	1 (4%)	0 (0%)	0 (0%)	24 (96%)
Baseline data	15. A table showing baseline demographic and clinical characteristics for each group	20 (80%)	2 (8%)	3 (12%)	0 (0%)
	15 NPT. When applicable, a description of care providers (case volume, qualification, expertise, etc.) and centers (volume) in each group	0 (0%)	0 (0%)	8 (32%)	17 (68%)
Numbers analysed	16. For each group, number of participants (denominator) included in each analysis and whether the analysis was by original assigned groups	23 (92%)	0 (0%)	2 (8%)	0 (0%)
Outcomes and estimation	17a. For each primary and secondary outcome, results for each group, and the estimated effect size and its precision (such as 95% confidence interval)	18 (72%)	7 (28%)	0 (0%)	0 (0%)
	17b. For binary outcomes, presentation of both absolute and relative effect sizes is recommended	1 (4%)	0 (0%)	1 (4%)	23 (92%)
Ancillary analyses	18. Results of any other analyses performed, including subgroup analyses and adjusted analyses, distinguishing pre-specified from exploratory	9 (36%)	0 (0%)	5 (20%)	11 (44%)
Harms	19. All important harms or unintended effects in each group (for specific guidance see CONSORT for harms)	16 (64%)	0 (0%)	9 (36%)	0 (0%)
Discussion					
Limitations	20. Trial limitations, addressing sources of potential bias, imprecision, and, if relevant, multiplicity of analyses	13 (52%)	10 (40%)	2 (8%)	0 (0%)
	20 NPT. In addition, take into account the choice of the comparator, lack of or partial blinding, and unequal expertise of care providers or centers in each group	1 (4%)	1 (4%)	11 (44%)	12 (48%)
Generalisability	21. Generalisability (external validity, applicability) of the trial findings	14 (56%)	4 (16%)	7 (28%)	0 (0%)
	21 NPT. Generalisability (external validity) of the trial findings according to the intervention, comparators, patients, and care providers and centers involved in the trial	6 (24%)	4 (16%)	11 (44%)	4 (16%)
Interpretation	22. Interpretation consistent with results, balancing benefits and harms, and considering other relevant evidence	24 (96%)	0 (0%)	1 (4%)	0 (0%)
Other information					
Registration	23. Registration number and name of trial registry	9 (36%)	1 (4%)	15 (60%)	0 (0%)
Protocol	24. Where the full trial protocol can be accessed, if available	5 (20%)	0 (0%)	20 (80%)	0 (0%)
Funding	25. Sources of funding and other support (such as supply of drugs), role of funders	18 (72%)	3 (12%)	3 (12%)	1 (4%)

Table 5

Completeness of reporting for observational studies investigating HBO based on STROBE (version combined for cohort, case-control, and cross-sectional studies); AR – item adequately reported; IR – item incompletely reported; NR – item not reported; NA – item not applicable

Section/Topic	Item number and description	AR *n* (%)	IR *n* (%)	NR *n* (%)	NA *n* (%)
Title and abstract	1a. Indicate the study's design with a commonly used term in the title or the abstract	14 (56%)	0 (0%)	11 (44%)	0 (0%)
	1b. Provide in the abstract an informative and balanced summary of what was done and what was found	25 (100%)	0 (0%)	0 (0%)	0 (0%)
Introduction					
Background/ Rationale	2. Explain the scientific background and rationale for the investigation being reported	20 (80%)	5 (20%)	0 (0%)	0 (0%)
Objectives	3. State specific objectives, including any prespecified hypotheses	25 (100%)	0 (0%)	0 (0%)	0 (0%)
Methods					
Study design	4. Present key elements of study design early in the paper	25 (100%)	0 (0%)	0 (0%)	0 (0%)
Setting	5. Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	17 (68%)	8 (32%)	0 (0%)	0 (0%)
Participants	6a. *Cohort study* — Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up *Case-control study* — Give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for the choice of cases and controls *Cross-sectional study* — Give the eligibility criteria, and the sources and methods of selection of participants	24 (96%)	1 (4%)	0 (0%)	0 (0%)
	6b. *Cohort study* — For matched studies, give matching criteria and number of exposed and unexposed *Case-Control study* — For matched studies, give matching criteria and the number of controls per case	5 (20%)	20 (80%)	0 (0%)	0 (0%)
Variables	7. Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	5 (20%)	20 (80%)	0 (0%)	0 (0%)
Data Sources/ Measurement	8. For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	25 (100%)	0 (0%)	0 (0%)	0 (0%)
Bias	9. Describe any efforts to address potential sources of bias	9 (36%)	3 (12%)	13 (52%)	0 (0%)
Study size	10. Explain how the study size was arrived at	19 (76%)	0 (0%)	6 (24%)	0 (0%)
Quantitative variables	11. Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	25 (100%)	0 (0%)	0 (0%)	0 (0%)

Table 5 continued.

Statistical methods	12a. Describe all statistical methods, including those used to control for confounding	12 (48%)	13 (52%)	0 (0%)	0 (0%)
	12b. Describe any methods used to examine subgroups and interactions	11 (44%)	1 (4%)	5 (20%)	8 (32%)
	12c. Explain how missing data were addressed	9 (36%)	1 (4%)	2 (8%)	13 (52%)
	12d. *Cohort study* — If applicable, explain how loss to follow-up was addressed *Case-Control study* — If applicable, explain how matching of cases and controls was addressed *Cross-sectional study* — If applicable, describe analytical methods taking account of sampling strategy	3 (12%)	0 (0%)	4 (16%)	18 (72%)
	12e. Describe any sensitivity analyses	1 (4%)	0 (0%)	1 (4%)	23 (92%)
Results					
Participants	13a. Report numbers of individuals at each stage of study – e.g., numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	18 (72%)	5 (20%)	1 (4%)	1 (4%)
	13b. Give reasons for non-participation at each stage	13 (52%)	0 (0%)	0 (0%)	12 (48%)
	13c. Consider use of a flow diagram	7 (28%)	0 (0%)	18 (72%)	0 (0%)
Descriptive data	14a. Give characteristics of study participants (e.g., demographic, clinical, social) and information on exposures and potential confounders	24 (96%)	1 (4%)	0 (0%)	0 (0%)
	14b. Indicate number of participants with missing data for each variable of interest	8 (32%)	2 (8%)	1 (4%)	14 (56%)
	14c. *Cohort study* — Summarise follow-up time (e.g., average and total amount)	12 (48%)	0 (0%)	1 (4%)	12 (48%)
Outcome data	15. *Cohort study* — Report numbers of outcome events or summary measures over time *Case-Control study* — Report numbers in each exposure category, or summary measures of exposure *Cross-sectional study* — Report numbers of outcome events or summary measures	25 (100%)	0 (0%)	0 (0%)	0 (0%)
Main results	16a. Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (e.g., 95% confidence interval). Make clear which confounders were adjusted for and why they were included	8 (32%)	10 (40%)	7 (28%)	0 (0%)
	16b. Report category boundaries when continuous variables were categorised	7 (28%)	0 (0%)	7 (28%)	11 (44%)
	16c. If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	1 (4%)	0 (0%)	4 (16%)	20 (80%)

Table 5 continued.

Other analyses	17. Report other analyses done—e.g., analyses of subgroups and interactions, and sensitivity analyses	14 (56%)	0 (0%)	1 (4%)	10 (40%)
Discussion					
Key results	18. Summarise key results with reference to study objectives	25 (100%)	0 (0%)	0 (0%)	0 (0%)
Limitations	19. Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	11 (44%)	12 (48%)	2 (8%)	0 (0%)
Interpretation	20. Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	25 (100%)	0 (0%)	0 (0%)	0 (0%)
Generalisability	21. Discuss the generalisability (external validity) of the study results	9 (36%)	6 (24%)	10 (40%)	0 (0%)
Other information					
Funding	22. Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	15 (60%)	3 (12%)	7 (28%)	0 (0%)

Analysis of RCTs identified significant gaps, particularly in NPT reporting. When evaluated solely against CONSORT, 15 studies (60%) were rated as 'good', reflecting moderate adherence to methodological standards. However, this adherence declined when incorporating NPT-specific items, as many items were marked 'Not Applicable' or 'Not Reported'. This may be surprising, as NPT was specifically designed to facilitate reporting of interventions that are more complex than conventional medication treatments. This could suggest these checklists items may not apply to or may not be recognised to apply to HBO-specific characteristics. For instance, items related to clustering by care providers (7a, 12a) and allocation of care providers to trial groups (3a) were frequently deemed inapplicable. This likely reflects the controlled nature of HBO, where treatment administration remains consistent, minimising variability introduced by care providers. Additionally, no studies reported monitoring of care providers' adherence to protocols (5d) or participant adherence (5e).

More importantly, significant gaps in key treatment parameters, particularly regarding chamber type, pressure levels, compression time, and decompression time were noted. Among the 25 RCTs, 19 studies (76%) did not specify the chamber type used, eight studies (32%) did not document compression time, and six studies (24%) failed to report the treatment pressure used. Additionally, 11 studies (44%) did not provide details on decompression protocols, which are essential for understanding the full pressure profile of HBO sessions.

Given that pressure level, session duration, and transition phases (compression and decompression) function similarly to medication dosing, these omissions may critically impact study reproducibility, treatment standardisation, and clinical applicability. While our initial screening rated most studies as adequately reporting intervention details, a more in-depth review suggests that essential HBO parameters were inconsistently documented. These findings underscore the need for HBO-specific reporting guidelines that better capture the distinct characteristics of an intervention.

Observational studies showed slightly better reporting rates, still with no study categorised as 'excellent', but with 19 studies (76%) categorised as 'good'. Critical elements were often omitted, including flow diagrams (18 studies; 72%), control of bias (13 studies; 52%), and missing data handling (13 studies; 52%). This reduces transparency and hinders bias identification, such as loss to follow-up. Underreporting in these domains weakens the credibility of observational findings and limits their utility in clinical guidelines.

Our findings align with prior research in other fields demonstrating persistent reporting deficiencies in both RCTs and observational studies on HBO.[27,78,79] Previous

Figure 2
Representation of completeness of reporting for RCTs investigating HBO, based on CONSORT and NPT

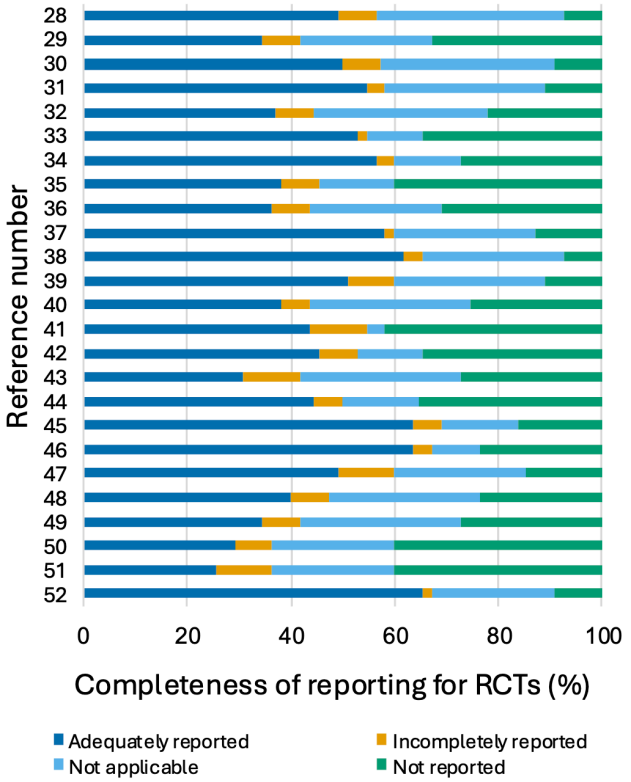
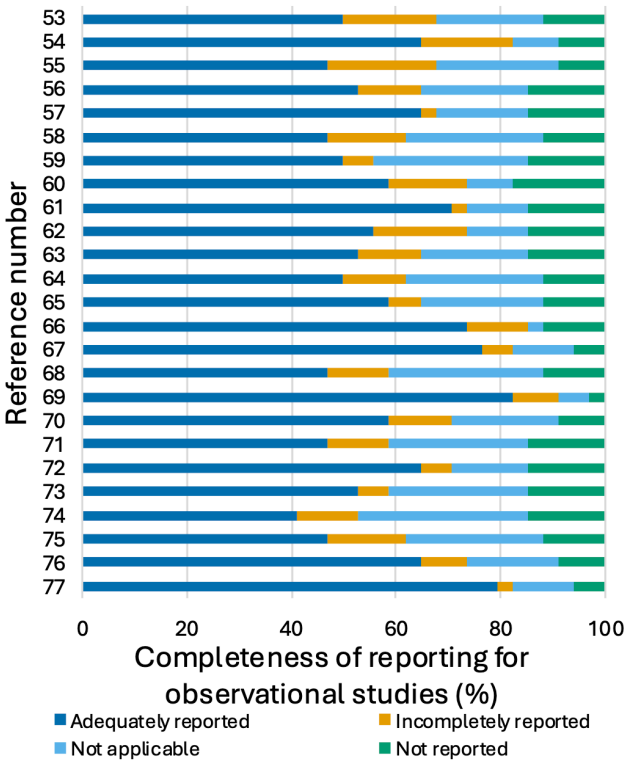


Figure 3
Representation of completeness of reporting for observational studies investigating HBO, based on STROBE



studies suggest that adherence to reporting guidelines improves reporting quality, but widespread endorsement and enforcement remain aspirational.[78,79] Notably, many HBO studies published in journals that endorse CONSORT or STROBE still lacked key methodological details, reinforcing the need for mandatory adherence to these guidelines.[78,79] This suggests that implementation strategies are key to promote adherence to reporting guidelines – a call shared by others previously.[78,79]

One distinctive aspect of HBO is the specialised role of the chamber operator, who 'drives' the chamber, managing pressurisation and a large portion of patient safety, setting it apart from conventional treatment administration in healthcare. Unlike other interventions, HBO requires a trained operator, which is not typically addressed in standard reporting checklists. Incorporating this role into future HBO-specific guidelines may enhance reporting completeness. Additional operational parameters specific to HBO are also important. Details on pressure levels and treatment schedules are important to evaluate protocol adherence. Additionally, the challenges of implementing sham-controlled trials complicate adequate blinding and control.[80,81] Environmental factors within the hyperbaric chamber may influence participant perception and interaction, further affecting blinding and study integrity. Addressing these issues requires the development of HBO-specific reporting guidelines that provide explicit instructions on reporting key aspects, including pressure ('dive') profiles, randomisation procedures, and bias management. Such guidelines would enhance transparency and strengthen the validity of HBO research.

The role of the peer reviewers is critical in improving reporting quality. Studies indicate that structured peer review, particularly when reviewers utilise CONSORT checklists, significantly enhances RCT reporting adherence.[24,82] Given the complexity and specificity of HBO studies, peer reviewers should be engaged in the design and creation of HBO-specific reporting guidelines, and receive specialised training to evaluate reporting quality effectively. Strengthening peer review processes could contribute to promoting more rigorous and transparent HBO research.

This study has limitations. Firstly, we only assessed a sample of recent peer-reviewed reports of clinical trials in HBO. We also did not consider articles accessible only through other literature databases or in languages other than English. This constrained the comprehensiveness of our sample, and thus the generalisability of our findings. Our decision to not assign weighting to missing or incomplete elements weakens the assessment of 'deficiencies'. For example, it is possible that authors did not comment on 'important changes' when there were none. The absence of such a statement may not

necessarily constitute a true reporting deficiency. Finally, our evaluation did not evaluate journals where the articles were published, which can contribute to differences in both quality and completeness of reports.

Conclusions

In a randomly selected sample of 50 clinical trials in HBO, reporting guidelines were not used by the vast majority of HBO trials and, when used, the reporting details were typically incomplete. Future efforts are required to ensure a high degree of relevance for all checklist items and to improve the quality of reporting in HBO trials in order to strengthen the quality of evidence in the field.

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Perceptions of airway protection tools: an international survey on the use of mouthpiece retaining straps in closed-circuit rebreather diving

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Accidents; Drowning; Gas toxicity; Risk management; Safety; Technical diving

Abstract
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Introduction: Rebreather diving carries a high fatality rate (estimated 1.8–3.8 deaths per 100,000 dives), yet its popularity is growing. Among 54 French military divers who lost consciousness underwater, none died when using a mouthpiece retaining strap (MRS) in a team diving setup. Despite this, MRS use remains limited among recreational divers for whom drowning is a major cause of death. This study assessed knowledge, perceptions, and training regarding MRS use within the rebreather diving community.
Methods: An international online survey targeting certified rebreather divers was disseminated via social media. The survey gathered demographic information, diving experience, MRS usage, and details on related training.
Results: A total of 563 responses were collected. Of these, 133 (23.6%) were instructors, and 210 (37.3%) had received MRS training. On a 0 to 100 scale, divers trained on MRS use rated MRS importance higher (median score: 74 [IQR 33–90]) than divers with no MRS training (median: 49 [IQR 16–67]). Barriers to MRS adoption included negative past experiences, poor training, misuse, and concerns about complications during bailout procedures.
Conclusions: While not widely adopted among recreational divers, the MRS is supported by strong safety data. Formal training significantly improves its perceived value and acceptance. Greater involvement from manufacturers, training agencies, and instructors is essential to promote education and encourage MRS adoption as a key safety measure in rebreather diving.

Introduction

The use of closed-circuit rebreathers (CCRs) is rapidly increasing within the technical diving community. By recycling exhaled gas through a carbon dioxide (CO_2) absorbent, CCRs reduce gas consumption and optimise breathing mixtures, enabling longer and deeper dives. However, their complexity increases the risk of technical failures and emergencies.[1] Rebreather diving carries a relatively high fatality rate, estimated at 1.8 to 3.8 deaths per 100,000 dives, with gas toxicity (e.g., hypoxia, hyperoxia, hypercapnia) being a major contributing factor.[2,3]

Although training standards exist, some practices seem to vary, and approaches are often guided more by personal beliefs or anecdotal experience rather than by established scientific evidence leading to ongoing controversy within technical diving community.[4] Even today, the use of a mouthpiece retaining strap (MRS) is not yet fully part of these standards, and not all rebreathers manufacturers are providing it by default.

The MRS is designed to secure the mouthpiece by wrapping around the diver's head and making a seal around the lips. By keeping the mouthpiece in place, it potentially reduces the risk of drowning in the event of mental impairment or unconsciousness. This would allow for a longer window of time in which a successful rescue could be carried out by another diver or a surface observer. While full-face masks (FFMs) also provide airway protection, they are often

considered bulky, technically demanding, and difficult to remove quickly in emergencies. In contrast, the MRS may offer a low-cost, simple, and effective alternative, if correctly used.[5] Data on its effectiveness remain limited, due to the obvious ethical constraints associated with studying unconscious divers, leading to controversies in the recreational community. Nonetheless, a French Navy study reported zero fatalities and only two moderate cases of water inspiration among 54 CCR divers who suffered loss of consciousness (LOC) while using a MRS.[1] The related benefit implied by this military diving study is likely to be translatable to the recreational diving setting.

Despite these findings, many divers remain reluctant to adopt the MRS. Various arguments are frequently put forward against the use of MRS in diver debates, including concerns about bailing-out, perception of risk, and general discomfort. These reservations may, in part, be attributed to a lack of awareness and suitable training.[6]

This survey aimed to assess the overall knowledge and perception about MRSs among the rebreather diving population and how formal training with a MRS influences divers' perception and voluntary use of the device within the rebreather diving community.

Methods

The study was approved by the Institutional Review Board of the Divers Alert Network (DAN) (#037-24-24). Participation was voluntary, and responses were confidential.

A cross-sectional survey was conducted and disseminated online through the DAN website and social media channels and was further distributed through diving-related news outlets such as InDepth magazine and rebreather-specific groups on social media. Data collection lasted from 2 August to 14 September 2024. The anonymous questionnaire was developed on the electronic data capture platform REDCap (REDCap consortium, Vanderbilt University, Nashville, TN, USA). Certified rebreather divers aged 18 years and older were invited to respond. Participants were presented with an information page outlining the study, which was followed by the collection of written informed consent.

The survey collected information regarding gender, age, academic background, rebreather diving experience, and whether they had received specific training in the use of the MRS. Participants with prior formal MRS training were asked about their regular use of the MRS, while those without training were asked about their interest in using it. The influence of formal training on users' perception of the MRS was examined. The perceived importance of wearing the strap for safety during rebreather diving was assessed using a scale from 0 ('not important') to 100 ('very important'). Additionally, the degree of agreement with various statements related to the use of the MRS concerning its interests, constraints, comfort, and diver's personal experience was evaluated. Respondents were able to add free-text comments to more narratively describe diving situations in which they felt the MRS either put them in a dangerous situation or, on the contrary, provided valuable assistance.

STATISTICAL ANALYSIS

Statistical analysis was performed with GraphPad Prism v10.4.1 (GraphPad Software Inc., San Diego, CA, USA). Continuous variables are presented as median and interquartile range (IQR), whereas categorical data are reported as counts and percentages. As data were not normally distributed, comparisons of unpaired continuous variables were performed using the Mann–Whitney test. Categorical comparisons were analysed using the Chi-square test or Fisher's exact test when required by sample size constraints. Statistical significance was defined as a P-value < 0.05.

Results

POPULATION DESCRIPTION

During the available period of the questionnaire, 887 responses were received. Of these, 324 were excluded due to being from non-rebreather divers or the lack of written consent, resulting in the analysis of 563 valid entries from 59 (10.5%) female, 495 (87.9%) male, two (0.4%) non-binary participants. Seven (1.2%) preferred not to disclose their sex. The age classes were 18–24 for 6 (1.1%), 25–34 for 53 (9.4%), 35–44 for 158 (28.1%), 45–54 for 170 (30.2%), 55–64 for 119 (21.1%) and older than 65 years for 53 (9.4%) divers. Four (0.7%) did not specify age. They were CCR certified for six (2–11) years with a median of 30 (50–100) rebreather diving-hours annually. One-hundred-thirty-three (23.6%) were rebreather instructors and 210 (37.3%) had received formal training on the MRS. Among the 353 (62.7%) who had never been trained on an MRS, 108 (30.6%) did not consider trying a MRS. There was no difference between the trained and untrained groups on gender ($P = 0.6$), or age categories ($P = 0.8$). Table 1 depicts the participants' educational background and rebreather diving experience.

PERCEIVED CONFIDENCE AND COMFORT IN THE USE OF MRS REGARDLESS OF TRAINING

After formal training, the MRS was more widely recognised as an important safety device in CCR diving ($P < 0.0001$, Figure 1). A total of 381 (67.7%) divers believed it could help prevent drowning, while 201 (37.7%) reported it might reduce jaw fatigue. Trained divers were significantly more likely to agree with these statements ($P < 0.0001$, Figure 2). Although few participants ($n = 36$) believed that the MRS made bailout impossible, many acknowledged that it introduces procedural complexity, particularly for those untrained ($P < 0.0001$, Figure 3). The MRS was reported as

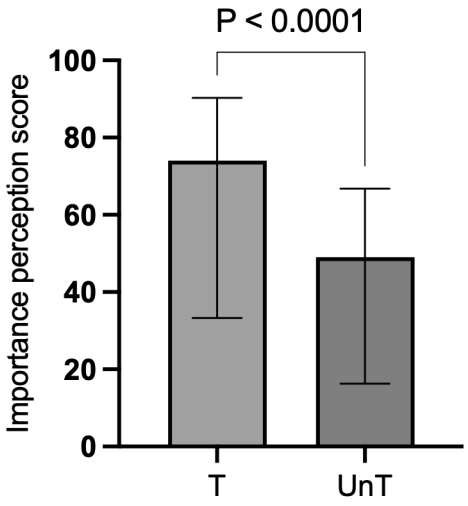
Table 1
Background and experience in rebreather diving among the trained and untrained divers on the MRS; BOV – bailout valve; CCR – closed-circuit rebreather; FFM – full-face mask; IQR – interquartile range; y – years

Parameter	Trained (*n* = 210)	Untrained (*n* = 353)
Education level, *n* (%)		
High school or less	41 (19.5)	46 (13)
University	150 (71.4)	281 (79.6)
Other	18 (8.6)	23 (6.5)
Unknown	1 (0.5)	3 (0.8)
Diving training		
CCR Certification (y), Median (IQR)	6 (3–12)	6 (2–11)
CCR Instructors, *n* (%)	60 (28.6)	73 (20.7)
Diving practice		
Annual CCR dive-hours, Median (IQR)	70 (40–110)	30 (50–100)
Usually have BOV on primary unit, *n* (%)	74 (35.4)	167 (47.3)
Try/use FFM on CCR, *n* (%)	27 (12.9)	32 (9)

incompatible with their diving configuration by 27 (12.9%) trained and 89 (25.3%) untrained divers (P = 0.0004). Trained divers more frequently reported that the MRS may have been both a helpful aid and, at times, a source of danger (Table 2). However, there was no significant difference between groups in the proportion of divers who had experienced a situation in which the MRS could have potentially prevented an accident (P = 0.06). Three divers reported a LOC underwater while using the MRS, with all experiencing favourable outcomes.

Drawing on the more narrative comments from divers about their experiences using the MRS, 43/353 (12.2%) written responses from untrained divers and 56/210 (26.7%) from divers trained to use the MRS were noted. In both cases, divers highlighted the additional workload the MRS is adding to the bail-out procedure and the possible issues it can create with the mask strap when attempting to remove the MRS (e.g., "*Complication during bailout process unless attached to a BOV. In case of a flooded unit or caustic cocktail it would be an even worse scenario*"). Some divers also insist on the importance of training to avoid or limit these issues, or the need for a mouthpiece and a lip cover who fit the user for the system to be fully effective (e.g., "*The strap interacts with the mask and adds more steps to bailout. I did several bailout exercises and got some water in the loop on a few. It might be different if you are trained on it from the start*"). A total of only 6/563 (1.1%) divers in this survey also highlighted that the MRS might have saved them during an emergency or a loss of consciousness (e.g., "*It (MRS) kept the mouthpiece in while fighting a lift bag when I was in odd orientations. It would have had a wet loop without it in that situation*", "*My CCR read PpO_2 incorrectly and caused hyperoxia. Because I was wearing a FFM that helped prevent me from drowning*", "*During a high PpO_2 alert, I had to use my BOV. Without a MRS, the BOV would be not comfortable for long dive because of the weight of mouthpiece*", "*During wreck diving I got falling pipe on my head and lost consciousness. The MRS saved my life.*"), and some suggested combining it with a bailout valve (BOV) to limit the loss of time when switching to the open circuit bailout. Many divers have also pointed the lower jaw fatigue while using the MRS, as well as its efficacy to keep the mouthpiece in place when they received a fin kick from their buddy and one also mentioned that he doesn't use the MRS while cave diving with a helmet.

Figure 1
Self-reported MRS importance for rebreather diving safety; scale 0 to 100 (not important to very important); T – trained; UnT – untrained



CONSIDERATION OF CONTINUED USE OF A MRS AFTER TRAINING

Of the 210 divers trained in the use of the MRS, 138/209 (66%) reported using it more than half the time and these will be considered 'regular' MRS users in the following text, with 127/138 (92%) of them doing so essentially invariably. In contrast, 7/71 (9.9%) used it about half the time, 13/71 (18.3%) less than half, and 51/71 (71.8%) had never used an MRS after their course. These latter groups are considered occasional (or non-) users. Training was received during recreational or technical diving classes for 191 (91%) divers, military courses for nine (4.3%), commercial or scientific diving for seven (3.3%), and other occasions (including possible self-training) for three (1.3%).

Occasional (or non)-users were more experienced and more frequently held CCR instructor certification (Table 3). There were no significant differences in gender (P = 0.2), age categories (P = 0.8), or educational background (P = 0.9). A BOV was used by 46/138 (34.1%) of regular users and

Figure 2

Perceptions on the potential interests and discomfort of wearing MRS following a specific training in rebreather divers; A – agree; D – disagree; IDK – 'I don't know'; NA – not applicable; N – neutral; SA – strongly agree; SD – strongly disagree; T – trained; UnT – untrained

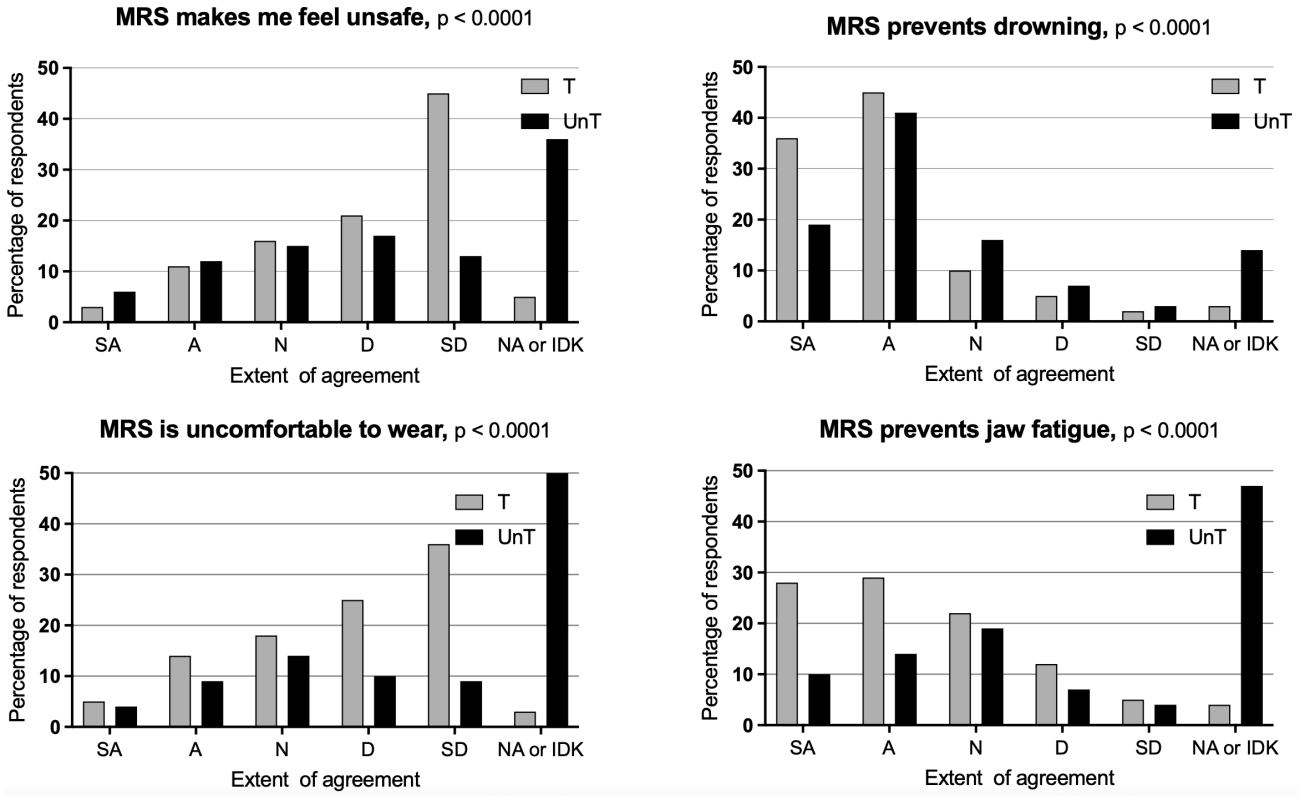
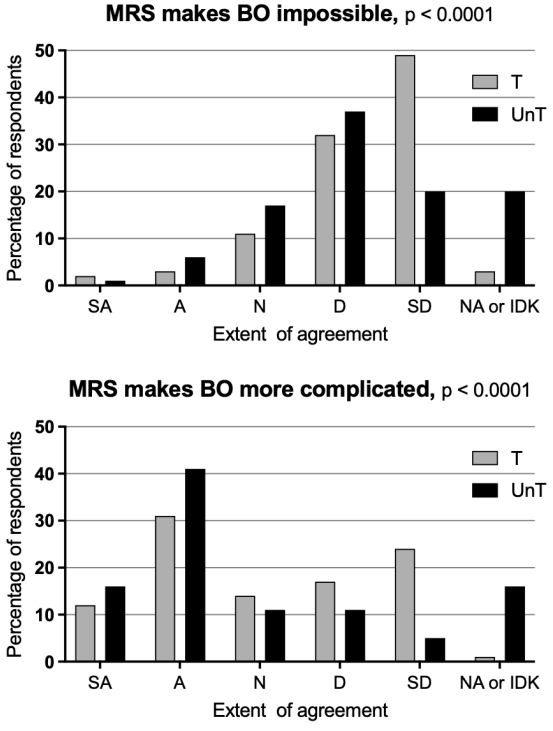


Figure 3

Perceptions of the interaction between MRS and bailout (BO); procedures; A – agree; D – disagree; IDK – 'I don't know'; NA – not applicable; N – neutral; SA – strongly agree; SD – strongly disagree; T – trained; UnT – untrained



27/71 (38%) of other divers ($P = 0.5$). The median MRS safety rating scale from 0 to 100 was 82 (71–98) for regular MRS users vs 22 (7–50) for other divers ($P < 0.0001$). Their instructors specifically emphasised the use of the MRS during CCR courses for 121/138 (87.7%) regular users and for 52/71 (73.2%) of individuals who later discontinued its use ($P = 0.001$). The MRS was judged not compatible with diving configuration for 3/138 (2.2%) of regular users and 24/71 (33.8%) of others ($P < 0.0001$). Among occasional (or non)-users, 19/71 (26.8%) reporting experiencing a dangerous situation involving a MRS, compared to 2/138 (1.5%) of regular users ($P = 0.009$). Conversely, 66/138 (47.8%) users considered a MRS helped them during a dive, versus 6/71 (8.5%) of occasional (or non-) users ($P < 0.0001$). There was no significant difference in the proportion of divers who recalled a past incident they believed could have been prevented by using an MRS ($P = 0.1$).

Discussion

Mouthpiece retaining strap adoption is still not widespread within the recreational CCR diving community. Formal training on the use of the MRS significantly enhances its perceived value, leading to its recognition as a crucial safety component in diving. Moreover, the influence of instructors, along with divers' personal experiences, whether of safety benefits or adverse situations, appears to be linked to their continued use of the MRS (or not) after training.

Table 2

Personal experience with the use of a mouthpiece retaining strap (MRS) during closed circuit rebreather (CCR) diving; 'trained' refers to CCR divers with specific training in use of a MRS whereas 'untrained' refers to CCR divers without such training

Opinion	Trained (*n* = 210) *n* (%)	Untrained (*n* = 353) *n* (%)	*P*
A MRS puts me in dangerous situation	21 (10.0)	13 (3.7)	0.002
A MRS has helped me during a dive	72 (34.3)	30 (8.5)	< 0.0001
I can think of at least one accident that could have been prevented with a MRS	74 (35.2)	97 (27.6)	0.06

Table 3

Comparison of diving experience and qualification between regular and occasional (or non-) users following MRS training; CCR – closed circuit rebreather; IQR – interquartile range

Parameter	Regular users, *n* = 138	Occasional (or non-) users, *n* = 71	*P*
Years CCR certified Median (IQR)	5 (2–10)	10 (5–17)	< 0.0001
Annual CCR dive hours Median (IQR)	58 (40–100)	100 (50–200)	< 0.0001
CCR instructors, *n* (%)	31 (22.5)	29 (48.3)	0.005

Loss of consciousness resulting from a gas toxicity accident was rarely reported by interviewed CCR trimix divers,[4] though a survivor bias might affect such data. Inappropriate breathing gas was frequently observed in CCR fatalities suggesting a potentially higher incidence of LOC with lethal outcomes in this population.[3,7] In contrast, military diving protocols may dramatically improve survival rates following these events.[1] Indeed, protective airway systems (MRS or FFM) have long been employed in military and commercial diving. Despite existing safety evidence[1,5] and the position of the rebreather training council,[8] only 37% of CCR divers in the present study have received formal MRS training mostly during their initial CCR training. Three (0.5%) of them reported a LOC event during a CCR dive and were wearing a MRS. Among them, two declared not having received specific training in its use, though all considered that this device had prevented them from drowning. Similarly in the French Navy study, Gempp reported zero fatalities and only two moderate cases of water aspiration among 54 CCR military divers who lost consciousness while using a MRS.[1] It is important to clarify that none of the divers in the Gempp report died, contrary to misinterpretation elsewhere.[5] The three fatalities reported didn't occur in the subset of the 54 LOC divers, but they involved divers who became trapped in wrecks and subsequently drowned.[1] The military's enforced use of airway-protective systems and rigorous team-diving protocols appear pivotal in converting potential fatalities into survivable incidents. Without these safety measures, the outcomes would very likely have been markedly worse.

There are no strong data quantifying the degree to which a MRS reduces risk for recreational divers, especially if they are diving solo, but this has once again emerged as a concern, as highlighted in the recent Rebreather Forum 4 statements one of which says "*The forum recognises the use of correctly deployed MRS as a strategy for avoiding loss of the mouthpiece and minimization of water aspiration in the event of loss of consciousness underwater*".[9] Recently, some manufacturers have begun integrating these systems into recreational diving equipment and it is now a requirement for CE standards (European standard EN14143; 2013). However, a lack of proper instruction may lead to the perception within the recreational diving community, that a MRS is an unsafe or ineffective tool.

No significant impact of age or education level was associated with MRS use. However, more frequent divers, and especially instructors, were more likely to have received MRS training. Paradoxically, instructors and the most active divers used a MRS less often. This tendency, where the most experienced individuals may be the least diligent about their own safety, is also well known within the mountaineering community.[10] It was expected that most untrained divers and non-users would report fewer events, whether favourable or hazardous, during the use of the MRS, simply because of less frequent use. Unfortunately, the survey cannot confirm whether untrained divers actually use the MRS, although some mentioned doing so in open comments. Two-thirds of divers, regardless of whether they were formally trained or regular MRS users, recognised that it could help prevent drowning, especially when their dive training included instruction on its use. Their perception of comfort benefit was more muted: the main criticism centred on increased procedural complexity, which was cited as a primary reason

for opting not to use the device. The extent and manner in which instructors emphasise the MRS during training appears to influence its uptake. Thus, raising instructor awareness and ensuring proper MRS training are essential prerequisites for broader acceptance among recreational divers. Interestingly, 61% of those trained always use the MRS, while 24% never use it, highlighting that training alone doesn't automatically guarantee adoption.

Beyond improving training on the MRS, it also seems that some progress should be made to develop systems that will fit different morphotypes to improve comfort and lips sealing. The need for quick removal systems also seems relevant to improve engagement with the MRS, as well as the need for the certification agencies to develop proper training programs on how to use it. Rapid removal becomes critical in some scenarios such as caustic cocktail; this can be facilitated by a quick-release clip and proper skill development in the procedure. Correct use of an MRS is of paramount importance because merely having it around the neck is not enough to ensure safety, and the MRS needs to be tight enough to ensure a proper seal between the lips and the lip-covering flange. Indeed, some divers self-reported issues with their MRS, mainly related to incorrect use or insufficient training. The association with a BOV may also reduce the risk as it allows to switch from the breathing loop to an independent open circuit system without removing the mouthpiece.

LIMITATIONS

This study faced several limitations. The dissemination channel may have introduced recruitment bias, and the response rate is unknowable. Since the dissemination took place within the DAN framework and other groups with a similar mindset (e.g., InDepth Magazine readers or technical diving social media groups), it is possible that the responding divers are more safety conscious than the rebreather community at large potentially creating a selection bias. The limited information regarding the frequency and potential use of the MRS by untrained divers may constrain the interpretation of our findings. It is plausible that some respondents categorised as untrained were, in fact, self-taught users who did not report this as an alternative form of training, thereby limiting a comprehensive assessment of MRS use within this population. Additionally, diving behaviours may vary depending on the region.[4] The lack of information about the divers' country of origin and the exclusive use of English language may result in overlooking a significant portion of the rebreather diving community, which may have different perspectives.

Conclusions

This survey suggests an influence of formal training on use of a MRS and its positive perception and use. The MRS is still not widely adopted within the recreational rebreather diving community. However, survey data and operational evidence suggest it may serve as a valuable safety aid, particularly when combined with proper training. Many arguments suggest that combining a MRS and BOV can provide critical protections in periods of great stress and hazard. While not universally applicable to every configuration or diver preference, improved training and equipment compatibility can help divers make informed decisions about its use. Manufacturers, training agencies, and instructors all have a role in enhancing MRS education and encouraging informed, context-appropriate adoption. Furthermore, as pointed by the Rebreather Training Council, "*MRS devices should not be seen as an independent solution, but may offer some protection from drowning, especially when used properly, and in concert with other safe diving practices (e.g., completing pre-dive checklists, careful monitoring of gas supplies, buddy/team-based diving, progressive experience building, and skill refinement)*".[8]

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Use of in-water recompression for decompression illness after deep freediving: a case series

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Abstract
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Introduction: There are increasing anecdotal reports of in-water recompression in freedivers who surface with neurological symptoms, likely suffering from decompression illness (DCI). Given the remote locations where many cases occurred, divers often struggled to access medical care, including the gold-standard hyperbaric oxygen treatment (HBOT), thus resorting to in-water recompression (IWR). Currently, IWR guidelines have only been discussed for scuba and surface supplied divers in specific scenarios, with protocols prescribing oxygen breathing at depths ≤ 9 metres maximum for around 1–3 hours.

Methods: We conducted detailed interviews with six competitive freedivers on signs, symptoms, management, and resolution of 13 cases of DCI. We additionally requested records of medical evaluation and treatment, with their consent.

Results: Three cases were suggestive of decompression sickness, six were consistent with arterial gas embolism, and four were ambiguous. Six cases were treated with IWR for 20–90 min at 5–25 metres with partial to complete resolution of symptoms. Four of these cases received HBOT afterwards. One diver reported significant permanent disability. Divers made several regimen changes after these incidents, including staying well-hydrated, reducing lung-packing, slowing their ascent rate, and/or employing prophylactic IWR when diving beyond a specified depth.

Conclusions: Given the remote locations of many incidents, freedivers often faced challenges in accessing HBOT. Self-treatment with IWR was widely used, either as a bridge to HBOT or as a standalone remedy. IWR poses potential risks, especially at the deeper depths reported in this study. This treatment modality is being utilised sometimes without medical oversight and recommended guidelines for IWR for freedivers should be developed.

Introduction

Severe debilitating neurologic symptoms after breath-hold diving (BHD), traditionally coined ‘Taravana syndrome’, have been well documented, and while thought to be linked to decompression illness (DCI), their pathophysiology has not been clearly delineated. DCI, a collective term encompassing decompression sickness (DCS) and arterial gas embolism (AGE), arises from evolution of bubbles from dissolved nitrogen gas (DCS) and/or air entering the arterial circulation after pulmonary barotrauma (AGE).[1] Theories of gas sources include de novo formation of bubbles in the arterial circulation with passage of bubbles through a patent foramen ovale, lung shunt, or frank lung barotrauma releasing alveolar gas.[2–6] DCI symptoms have also been attributed to endogenous cerebral ischaemia and blood-brain barrier disruption due to endothelial dysfunction.[7] Nonetheless, the pathophysiology underlying DCI particularly in BHD is still currently debated.[8]

The range of reported neurologic symptoms after BHD extends a wide spectrum from mild symptoms such as weakness, dizziness, and headaches to severe symptoms such as complete paralysis and loss of consciousness. Mild symptoms may be barely perceptible to divers and easily dismissed as post-dive fatigue or the lingering effects of hypoxaemia. Meanwhile, severe symptoms are rare with a recent systematic review of literature documenting 14 cases.[1,9] In addition, there are cases to suggest that this entity may not always be associated with depth given neurologic symptoms have been seen after shallow breath-hold diving and even lung-packing (glossopharyngeal insufflation).[10,11] Given the low incidence of severe neurologic symptoms, gathering data on such cases and

developing guidelines for the management of DCI in BHD has been difficult, with the added challenge of breath-hold depth diving often occurring in austere, remote locations often with no clinical medical support or oversight.

Given the rise of both recreational and competitive breath-hold diving, the medical community should have increased awareness of the possibility of DCI with severe symptoms, as well as a mechanism for gathering data that can inform medical management. Many hyperbaric practitioners may not be aware of injuries related to BHD. This case series challenges the historical dogma that DCI after BHD is not possible given the short time at depth.[12,13] In addition, many divers today are notably self-diagnosing and self-treating with in-water recompression (IWR). In-water recompression involves rapid re-immersion following the onset of symptoms, most often to shallow depths around 9 m, preferably while breathing pure oxygen.[14] Although this technique remains controversial and its actual benefits are difficult to assess due to the wide variety of protocols, it is increasingly being considered in various management guidelines under specific conditions.

Methods

This case series presents six divers with 13 cases of neurologic symptoms following BHD, documented through personal interviews conducted by two physician researchers after an initial survey of DCI in BHD. It is important to note that the symptoms and outcomes reported are anecdotal. We conducted detailed interviews after informed consent with six competitive freedivers on signs, symptoms, management, and resolution of 13 incidents that could represent DCI. We additionally requested medical records as available, with each subject's consent. This study was conducted with approval from the Institutional Review Board of University of California, San Diego, under protocol #810598 – Survey of Decompression Illness Incidents in Breath-Hold Divers. Each subject agreed to the publication of their case or cases in medical literature.

Results

Six divers reported 13 cases of suspected DCI summarised below (Table 1). Median depth of the deepest dive for each case was 100 m (interquartile range [IQR] 89−110 meters) with a median dive time of 3:08 minutes:seconds (IQR 2:15–3:16). Three cases were suggestive of DCS, six were consistent with AGE, and four were ambiguous. All reported incidents involved male divers, with suspected provoking factors being dehydration, lung packing, exhaustion, repetitive dives with short surface intervals, and lung barotrauma. Six cases were treated with IWR. The median maximum depth for IWR was 8.5 m (IQR 6.5−15.0) with a median time of 24.5 minutes (IQR 10.5–34.3). Four of the six cases received hyperbaric oxygen treatment (HBOT) afterwards. One diver reported significant residual deficits even after HBOT. A map of incident locations can be found in Figure 1. They have been unassigned from the diver and incident to protect their identity. The cases are described below. Following each case is our hypothesis of the cause for the reported signs and symptoms.

CASE 1 (DIVER A)

A male diver in his 30s had just completed a transatlantic flight and reportedly felt dehydrated. He performed four BHDs to 40, 60, 80, and 100 m and served as a safety diver for other divers as well. His final dive was a 100 m constant weight dive, with a dive time of three minutes 15 seconds. Upon reaching the shore around 10–15 minutes after the dive, he felt dizzy, had facial numbness, and was unable to walk due to muscle weakness. Around 45 minutes after symptom onset, he went to a local doctor who thought his symptoms were related to hypoglycaemia. He breathed around 10 minutes of surface oxygen at the clinic, and his symptoms were completely resolved. He attributes this incident to the number of dives, short surface intervals, exhaustion from travel, and dehydration with possible hypoglycaemia.

Analysis

We speculate his symptoms could represent DCS from repetitive dives successfully treated with surface oxygen. We believe the diagnosis of hypoglycaemia could be a red herring as the treatment for hypoglycaemia is glucose, not supplemental oxygen.

CASE 2 (DIVER A)

Diver A performed a competition constant weight dive with an announced depth of 125 m. At 90 m depth, he could no longer equalise and turned back to the surface. After this dive, he breathed prophylactic oxygen for five minutes on the surface. On his way back to shore around 3–5 minutes after the dive, he reported extreme vertigo and right-sided paralysis. After 30 minutes of breathing surface oxygen, his symptoms persisted. He proceeded to breathe 30 minutes more of surface oxygen, with symptoms resolving in the last 10 minutes of this oxygen administration. He attributes this incident to lung overpacking and competition nervousness. Of note, he reports that two days before this competition dive, he dove to 120 m twice, after which he experienced word-finding difficulties. He also reports that they were in phone contact with a local hyperbaric chamber who advised against transportation and to continue oxygen administration.

Analysis

We speculate that the patient's hemiplegia within minutes of surfacing could represent a cerebral AGE. The diver reported overpacking so he could have suffered asymptomatic

Table 1
Cases of severe neurologic symptoms after and reported treatment; CPR – cardiopulmonary resuscitation; HBOT – hyperbaric oxygen treatment; IWR – in-water recompression; n/a – not available; Surf O_2 – surface oxygen

Diver	Case	Dive depth (m)	Dive time (min:sec)	Symptoms	Treatment	IWR depth (m)	IWR time (mins)
A	1	Multiple dives to 40, 60, 80 and 100	1:40	Dizziness, facial numbness, inability to walk	Surf O_2		
	2	125	n/a	Extreme vertigo, right-sided paralysis	Surf O_2		
B	3	Multiple dives to 40	2:00	Numbness and tingling in the right leg	IWR, Surf O_2	10	3
	4	127	6:38	Unconsciousness, paralysis to all extremities	CPR, IWR, Surf O_2	5–20	29
C	5	Multiple dives to 55	n/a	Severe headache, coughing fit, balance issues	Surf O_2		
	6	92	3:04	Left-side paralysis, blurry vision, slurred speech, impaired coordination, fatigue, dizziness, shortness of breath	Surf O_2		
	7	100	3:20	Aphasia	IWR	15–25	60
	8	114	3:14	Left arm paralysis	IWR, Surf O_2	6–7	10
	9	105	3:10	Left arm paralysis, slurred speech, dizziness, fatigue, shortness of breathing	IWR, HBOT	5–7	12
D	10	89	3:15	Right arm tingling, difficulty breathing	Surf O_2		
	11	88	3:12	Abnormal behavior, facial drooping, right-sided paralysis	IWR, Surf O_2, HBOT	10	35
E	12	110	n/a	Aphasia, right arm, and leg paralysis	IWR, Surf O_2, HBOT	10–20	32
F	13	86	2:20	Right-hand numbness, tingling, right leg paralysis	IWR, HBOT	5	20

Figure 1
Map of incident locations, some of which are in remote, austere island locations

overexpansion barotrauma. Prophylactic surface oxygen did not prevent the incident, but it was successfully treated with surface oxygen.

CASE 3 (DIVER B)

A male diver in his 40s performed a 40 m constant weight dive with a 45 second hang (staying at depth) for a total dive time of two minutes. Of note, he had previously completed six dives to 40 m with a 45 second hang; therefore, this was not new or different from previous. He experienced numbness and tingling in the right leg. He did not have oxygen readily available and performed IWR on apnoea. The symptoms were not resolved. He was able to procure oxygen about one hour later, after which he re-entered the water for three minutes at 10 m. His symptoms partially resolved and dissipated over one month. He later received

a magnetic resonance imaging (MRI) scan which reportedly showed damage to the hypothalamus.

Analysis

We speculate this is a case of DCS from prolonged time at depth partially treated with IWR. The MRI findings of hypothalamus damage are likely unrelated to the case.

CASE 4 (DIVER B)

Around three years later, Diver B performed a 127 m personal best attempt free immersion dive with 50 lung packs. His previous personal best in this discipline was 126 m. The total dive time was six minutes and 42 seconds. On his ascent, he blacked out at 42 m. Per his dive computer, he sank back down to 76.8 m at 5:20. He was brought back to the surface at 6:42 unconscious. Cardio-pulmonary resuscitation (CPR) was performed for 30 seconds with a successful return of spontaneous circulation and consciousness. He had weakness over his extremities but could speak. He then self-administered IWR, descending to 20 m followed by a slow ascent breathing pure oxygen for eight minutes. He then conducted a second IWR, spending three minutes at 15 m, eight minutes at 10 m, and 10 minutes at 5 m. After this treatment, his symptoms were completely resolved.

Analysis

We speculate this is a case of drowning from a deep underwater blackout. The reason for the blackout is unclear but could include DCI.

CASE 5 (DIVER C)

A male diver in his late 30s performed breath-hold dives five times to around 55 m with a surface interval of around 20–30 minutes between each dive. At that time, his personal best was 80 m. He noted that he exerted himself after pulling up the bottom weight at the end of the dive session. As he was raising the bottom weight to the surface, he reports choking on some water that induced a coughing fit. When the cough subsided, he reports experiencing a sharp headache. After swimming to shore, he experienced balance issues, falling to the ground multiple times as he attempted to exit the water. Two hours later, he breathed surface oxygen for about 30 minutes, which partially resolved his symptoms. He noted that the episodes of dizziness persisted for about two weeks. He noted that perhaps decreasing the frequency of deep dives would have prevented this incident.

Analysis

We speculate this is a case of DCS from repetitive dives partially treated with delayed surface oxygen. His coughing is attributed to aspirating water and not pulmonary injury.

CASE 6 (DIVER C)

Around seven months later, Diver C performed a free immersion dive to a depth of 92 m, with a dive time of three minutes and four seconds. He packed approximately 10 times. At that time, his personal best in the discipline was 100 m. Of note, earlier in the month, he contracted COVID and was in quarantine before this dive. He reported nine days of rest before this dive. He noted that he also exerted himself after this dive by pulling up the bottom weight. Approximately 15–20 minutes after the dive, he experienced complete left-sided paralysis, blurry vision, slurred speech, impaired coordination, fatigue, dizziness, and shortness of breath. He breathed pure oxygen at the surface for around 40 minutes. His symptoms began to gradually improve at around 25 minutes of using oxygen with full resolution by the end of the 40 minutes on oxygen. He reports that over the course of the next four months he would wake up each morning with hand tremors.

Analysis

We speculate this could be neurological DCS from the release of gas in tissues triggered by exertion after diving. However, cerebral AGE could also cause his unilateral neurological deficits. Given his recent pulmonary infection, glossopharyngeal insufflation, and concurrent shortness of breath, we suspect pulmonary barotrauma could have led to gas emboli. This case was successfully treated with surface oxygen. The hand tremors are likely unrelated to the case.

CASE 7 (DIVER C)

Around eight months later, Diver C performed a free-immersion training dive to 100 m, with a dive time of three minutes and 20 seconds. He noted that on this day he was dehydrated. Around 20 minutes after the dive, he experienced aphasia. He also struggled to get oxygen as it was on shore, which caused a delay. He self-administered IWR with pure oxygen at 15 m for 20 minutes, which completely resolved his symptoms. He completed a second IWR, where he breathed pure oxygen at 25 m while being slowly brought up to surface, which took a total time of 40 minutes. He had no residual symptoms after this incident.

Analysis

We speculate that this was a case of DCI successfully treated with IWR alone.

CASE 8 (DIVER C)

Around one year later, Diver C performed a constant weight mono-fin dive to 114 m over three minutes and 40 seconds. During prophylactic IWR at around 6–7 m, which he routinely performed immediately after the dive, he noticed weakness to his left arm. He stayed underwater for around 10 minutes, converting his prophylactic IWR to a treatment

session. Afterwards, he breathed oxygen at the surface for 10 minutes, which completely resolved his symptoms.

Analysis

Prophylactic IWR did not prevent this DCI incident, but the combination of IWR and surface oxygen successfully treated the case.

CASE 9 (DIVER C)

Around one year later, Diver C performed a constant weight bi-fin training dive to 105 m, with a dive time of three minutes 10 seconds with approximately 20 lung packs. Around two minutes after this dive, he experienced left arm paralysis, slurred speech, dizziness, fatigue, and shortness of breath. He immediately self-treated with IWR with pure oxygen at 5 m for around 6–7 minutes, which completely resolved his symptoms at the time. After surfacing, he finished the oxygen in the tank (the symptoms returned within 15 min after surfacing). Fifteen minutes after finishing the oxygen, his symptoms returned, notably his left arm deficits. About 1 hour and 30 minutes later, he was able to self-administer IWR again with pure oxygen at 7 m for five minutes, which resolved all his symptoms. However, once again, he ran out of oxygen, and 5–10 minutes after surfacing, he reported that all his symptoms were returning. He was then taken by ambulance to the nearest hospital and later transferred to a hyperbaric chamber, which took up to 72 hours. There, he underwent 11 sessions of hyperbaric treatment. Workup including complete blood count and basic metabolic panel, chest X-ray, and transthoracic echocardiogram was grossly normal. He has residual deficits in balance and weakness in his left arm.

Analysis

We speculate this is a case of cerebral AGE given the rapid onset of symptoms from surfacing. Given his concurrent shortness of breath along with focal neurological symptoms, we suspect he had a component of pulmonary barotrauma that could have led to arterial gas emboli. He was partially treated with IWR and HBOT.

CASE 10 (DIVER D)

A male diver in his 60s performed a free immersion dive to 89 m with a dive time of three minutes and 15 seconds. Within seconds after the dive, he experienced right arm tingling. He breathed surface oxygen for five minutes, which completely resolved his symptoms. He had increased breathing hours after the dive.

Analysis

This case was successfully treated with just five minutes of surface oxygen, suggesting that it was extremely mild DCI or transient hypoxemia after a dive.

CASE 11 (DIVER D)

Around two years later, Diver D performed a competition-free immersion dive to 88 m with a dive time of three minutes and 12 seconds. Of note, he noted that five days leading up to this competition dive, he dove to 93 m where he felt some chest pressure post-dive but otherwise recovered appropriately. He dove to 81 m the day before this dive without incident. Five to ten minutes after the competition dive, fellow competitors noticed abnormal behaviour and facial drooping. Competition staff noted that he could not follow directions or count fingers and experienced paralysis in his right leg and arm. Medical staff administered IWR with pure oxygen to 10 m with a slow ascent, with a total in-water time of 35 minutes. He had complete recovery after this treatment. He continued to breathe oxygen at the surface for 30 more minutes. Based on the recommendation of the medical staff, the diver underwent a second round of IWR breathing pure oxygen at 5 m with a slow ascent, for a total time of approximately 30 minutes. He continued to have no residual deficits. Three hours later, he was evacuated by helicopter to the nearest hyperbaric chamber. At this hospital, he was haemodynamically normal and neurologically intact, with a normal non-contrast cerebral computed tomography (CT) scan. His lung CT showed "*discrete areas of scattered ground-glass opacities in the pulmonary hemifields. Findings are nonspecific but suggestive of barotrauma-related lesions in the context of the incident*". He underwent intravenous hydration as well as two HBOT treatments (US Navy Table 5a). Months later, he underwent a transthoracic echocardiogram with bubble contrast which showed a small patent foramen ovale (PFO).

Analysis

We suspect this is a case of cerebral AGE given the rapid onset of unilateral neurological symptoms after surfacing. Even though the diver did not report any respiratory symptoms, he had cross-sectional lung imaging that demonstrated evidence of lung injury, which could have led to arterial gas emboli. The patient was also found to have a PFO, which is another potential pathway for gas entry into the arterial circulation. This case was successfully treated with IWR, surface oxygen, and HBOT.

CASE 12 (DIVER E)

A male diver in his late 40s performed a free immersion dive to a depth of 110 m. At that time his previous personal best was 120 m. Five minutes after the dive, he became aphasic with right arm and leg paralysis. Oxygen was procured from shore and brought out to the platform. He self-administered IWR with pure oxygen at around 13 m for around 15 minutes. At this time his paralysis was completely resolved but he still found it hard to find words and felt that his speech was not fully there. Several hours later, he self-administered IWR again following this protocol: 20 m for two minutes, 15 m for five minutes, 10 m for 10 minutes,

slow ascent to 5 m, and then to the surface. At this time, though his physical symptoms improved, he still could not do simple math and felt brain fog and sluggish. Two days later, he flew to a hyperbaric chamber where he completed five sessions of HBOT. Today, while he is not entirely sure, he feels that he has completely recovered, at least grossly his mental and physical function. He endorses plentiful rest and use of prophylactic oxygen (pure oxygen at 10 m for 5–6 minutes) for dives deeper than 105 m.

Analysis

We suspect this is a case of cerebral AGE successfully treated with IWR and HBOT.

CASE 13 (DIVER F)

A male diver in his 30s performed a training dive to 86 m with a dive time of two minutes and 20 seconds. He packed 40 times prior to this dive, exhaling prior to surfacing at a depth of less than 10 m on ascent. This was a personal best dive, where his previous personal best was 84 m. He notes that he experienced an increased urge to cough and 1–2 episodes of haemoptysis. Starting 30 seconds after this dive, he experienced right hand numbness and tingling and progressed to complete paralysis of his right leg. At that time, there was no oxygen readily available. He returned to shore and was unable to ambulate or feel his right leg. Around 30 minutes later, he self-administered IWR, where he breathed pure oxygen at 5 m for five minutes for four times with a surface interval of around five minutes. After the third round of IWR, his symptoms improved; however, he still could not move or feel his toes. He was taken to the nearest hyperbaric chamber and started HBOT around three hours after his symptoms started. He reported undergoing 10 treatments using US Navy Treatment Table 6 over the next 10 days. In this admission, he had a transthoracic echocardiogram with bubble contrast which showed no PFO. Laboratory tests were within normal limits. His symptoms significantly improved, although he has residual mild paresthesia in his right shin. He endorses packing less, hydrating well, slowing down on ascent in the last 30 m, exhaling before surfacing, and prophylactically breathing oxygen at depth after dives deeper than 85 m.

Analysis

We suspect this is a case of cerebral AGE from pulmonary barotrauma of ascent given the haemoptysis reported by the diver. This case was partially treated with IWR and HBOT.

Discussion

Of these cases, three cases were suggestive of DCS, six were consistent with AGE, and two were categorised as DCI given not enough data to distinguish between DCS and AGE, as outlined in Table 2. Reported risk factors included repetitive dives with short surface intervals, exhaustion, dehydration, and overpacking. There were no clear patterns or consensus of risk factors. There were also no patterns on timing of repetitive deep dives and surface intervals, with divers reporting up to one week of rest (cases 6 and 9) before the target dive that resulted in neurologic deficits. IWR was able to provide partial to complete resolution of symptoms in many of these cases, although there was no consistency between protocols used. Only one diver reported significant permanent neurological deficits, which likely resulted from the multi-day delay to HBOT.

Current prehospital management guidelines for DCI, including DCS and/or AGE, emphasise the importance of first aid and activating emergency services. While the DAN diving hotline provides global support, it cannot replace local emergency services. Initial management focuses on airway protection, administering oxygen, and maintaining normothermia. Oral hydration is recommended for conscious patients. Patients presenting with symptoms beyond mild DCS should receive definitive recompression therapy as soon as possible, as delays beyond six hours are associated with less complete recovery.[15,16] Air transport should be as close to sea-level atmospheric pressure as possible or conducted at the lowest feasible altitude (ideally below 150–300 m).

Given the remote island locations where many cases occurred, as shown in the map (Figure 1), divers struggled to access medical care, including hyperbaric chamber treatment. Thus, many self-administered IWR, either to temporize their symptoms as a bridge to hyperbaric treatment or in some cases, fully treat their physical impairment. Most divers in this series survived these incidents without permanent deficits. However, one case resulted in a moderate permanent neurologic deficit, highlighting the importance of immediate treatment. Divers must weigh the benefits and risks of seeking hyperbaric chamber treatment, which may not be readily accessible, versus conducting IWR in remote locations with no medical support or supervision. Only one case of IWR was administered by medical staff and all other incidents appear to be self-designed and self-administered.

Existing protocols for IWR have been designed for compressed air divers who have been supersaturated by breathing compressed gas, but it is unclear if the divers in this case series were aware of those protocols.[17] Recommended safety practices include using a full-face mask or mouthpiece retaining strap, tethering the diver to prevent sinking, limiting treatment depth to 9 m and having a buddy assist in case of a seizure related to oxygen toxicity.[14] Contraindications to IWR include hearing loss, vertigo, vomiting, altered mental status, shock, respiratory distress, or severe physical incapacitation.[14] The fact that many of these cases had severe symptoms that could be contraindications to IWR brings up safety concerns over the oversight and availability of oxygen. There is a need for these safety practices with the consideration for air or sea evacuation to be specific to the BHD environment to minimise improvisation during times of emergent need.

Table 2
Case symptomatology, presumed diagnosis, reported potential risk factors; AGE – arterial gas embolism; DCI – decompression illness; DCS – decompression sickness; PFO – patent foramen ovale

Diver	Case	Symptoms	Presumed diagnosis	Residual deficits	Potential risk factors
A	1	Dizziness, facial numbness, inability to walk	DCS	None	Repetitive dives, exhaustion, dehydration, overexpansion barotrauma
	2	Extreme vertigo, right-sided paralysis	AGE	None	
B	3	Numbness and tingling in the right leg	DCS	None	Prolonged time at depth
	4	Unconsciousness, paralysis to all extremities	Syncope, drowning	None	
C	5	Severe headache, coughing fit, balance issues	DCS	None	Repetitive dives (Case 5) Overexpansion barotrauma (cases 6 and 9)
	6	Left-side paralysis, blurry vision, slurred speech, impaired coordination, fatigue, dizziness, shortness of breath	AGE	None	
	7	Aphasia	DCI	None	
	8	Left arm paralysis	DCI	None	
	9	Left arm paralysis, slurred speech, dizziness, fatigue, shortness of breathing	AGE	Gait, balance, left upper extremity weakness	
D	10	Right arm tingling, difficulty breathing	DCI vs hypoxia	None	Overexpansion barotrauma, PFO
	11	Abnormal behavior, facial drooping, right-sided paralysis	AGE	None	
E	12	Aphasia, right arm, and leg paralysis	AGE	None	None obvious
F	13	Right-hand numbness, tingling, right leg paralysis	AGE	Paresthesia to right lower extremity	Overexpansion barotrauma

Conclusions

In conclusion, severe neurologic symptoms consistent with DCI can occur after breath-hold diving. The reported incidents occurred as shallow as 40 m, with several cases noted after repetitive dives with extended time at depth. All incidents reported in this case series occurred in male divers. Suspected contributors included dehydration, overpacking, exhaustion, and frequent deep dives, with two cases linked to lung barotrauma based on reported symptoms or imaging findings. Oxygen treatment, both normobaric and hyperbaric, was the most used treatment modality, with seven cases using self-administered IWR to achieve partial or full symptom resolution.

The incidents reported in this series underscore the importance of understanding DCI and the need for proper prehospital management and treatment protocols. Breath-hold divers are increasingly self-treating with IWR without seeking definitive hyperbaric oxygen therapy in a chamber under the supervision of a physician. IWR poses significant potential risks such as drowning and hyperoxia, especially at the depths reported in this study, and is currently not a recommended practice. In addition, after an incident has occurred and seemingly resolved, it is imperative that a diver seek definitive treatment and evaluation from a medical professional knowledgeable in hyperbaric medicine. As BHD practices continue to evolve, it remains critical to document and research these incidents to increase awareness of DCI incidents and develop best-practice guidelines for the management and prevention of these incidents.

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Decompression illness in breath-hold divers: insights from an online survey

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Arterial gas embolism; Decompression sickness; Freediving; Hyperbaric oxygen treatment; In-water recompression

Abstract
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Introduction: Breath-hold divers can surface with neurological symptoms consistent with nitrogen buildup in tissues or gas entry into the arterial circulation, collectively termed decompression illness (DCI). While DCI has historically been attributed to diving with compressed air, breath-hold divers have reported similar syndromes. The causes, diagnosis, and management of DCI in breath-hold divers is poorly understood.
Methods: We developed an online survey that queried breath-hold divers on the symptoms they experienced during decompression illness events and the medical management of each event.
Results: A total of 36 (31 M, 5 F) breath-hold divers filled out the survey. A majority identified as recreational freedivers, competitive freedivers, and/or spearfishers with an average age of 45 years and 18 years of breath-hold diving experience. Of those surveyed, 33 (92%) held a certification from an accredited training agency. A total of 18 (50%) reported experiencing DCI, with 21 DCI incidents reported by 13 individuals from 1999–2024. Sixteen (76%) of DCI incidents occurred during training, with an average depth of 83.4 m and average speed of 1.0 $m \cdot s^{-1}$. Thirteen (62%) percent of DCI incidents occurred while diving to depths shallower than a previous personal best. The most common symptoms were weakness, numbness, slurred speech, and fatigue. The most common treatment modalities were surface oxygen, in-water recompression, and hyperbaric oxygen therapy. Sixteen divers (76%) had partial or complete resolution of their symptoms. The top cited contributors to the DCI incidents were depth, short surface interval between dives, and pulmonary barotrauma.
Conclusions: Breath-hold divers can experience DCI even when diving within their limits. The most cited contributors to DCI were depth, short surface interval between dives, and pulmonary barotrauma. Most divers' symptoms resolved after treatment with surface oxygen, in-water recompression, and/or hyperbaric oxygen therapy.

Introduction

Breath-hold divers, commonly called freedivers, have in the past described neurological symptoms after certain dives that have been attributed to both nitrogen loading in cerebral tissue as well as gas entry into the arterial circulation.[1] This syndrome was initially titled Taravana syndrome which means "*to fall crazily*" by physicians who noticed Polynesian pearl fisherman with ataxia after repetitive dives,[2] then later attributed to cerebral decompression sickness as the knowledge of decompression-related injuries became more well-described in compressed-air divers, including scuba, surface-supplied, and saturation divers. Neurological symptoms consistent with decompression illness (DCI), which encompasses both decompression sickness as well as arterial gas embolism, have been described in the literature by commercial,[3] competitive,[4] as well as recreational breath-hold divers.[5] Risk factors attributed to the development of decompression illness from breath-hold diving include repetitive dives with short surface intervals,[3] long deep dives > 100 m,[6] and a fast rate of ascent.[7]

DCI is diagnosed clinically with frequent and thorough neurological examination and re-examination after a dive, as many symptoms can overlap with those of extreme physical effort, hypercapnia, and hypoxaemia. Common symptoms include headache, dizziness, vertigo, confusion, vision changes, focal paresthesias, generalised weakness, and speech difficulty.[8] The distribution may not follow specific vascular regions typical for ischaemic strokes, thereby DCI should still be considered when the pattern of neurological symptoms do not follow classical patterns of

cerebrovascular accidents. Non-neurological manifestations of DCI can include dermatological, musculoskeletal, pulmonary, and constitutional symptoms.[9] While DCI in freedivers is becoming more recognised in the diving medical community, treatment has largely mirrored that of DCI in scuba and commercial divers, focusing on oxygen therapy and recompression. A large challenge to treatment is that freediving often occurs in remote locations with no immediate access to medical facilities and/or hyperbaric chambers. We therefore developed a survey which sought to explore the medical management of DCI in freedivers and the outcome of various treatments.

Methods

The study was approved by the University of California San Diego Institutional Review Board. Data collection was open for seven months from June–December, 2024.

This study was designed as a retrospective study with convenience sampling by survey. The survey was developed using Qualtrics and distributed via the Divers Alert Network and University of California San Diego to social media and freediving message groups. There was no paper version. The study population included divers 18 years or older who could read English and had access to the internet. Participants were recruited with a tagline of "*Breath-Hold Divers: Have you ever experienced decompression illness? We want to hear from you! Fill out our short survey below*." Participants had to consent to both conditions on an initial consent page before accessing the survey. This survey did not have an intervention as it was retrospective. There was no comparison group, as this was an attempt to gather data on a group where DCI is not well documented. The desired outcome of the survey was to gather anonymous data on the manifestations, circumstances, and treatment surrounding suspected DCI incidents in breath-hold divers.

The survey was divided into two parts: the diver's demographic data, and the opportunity to report on DCI incidents. In the first part, each diver's age, sex, gender, past medical history, experience and training in breath-hold diving, and familiarity with decompression illness medical terminology was collected. In the second part, divers were invited (but not required) to provide information on any decompression incident they experienced, including their symptoms, treatment, and likely causes. The survey had 11 demographic questions per diver and 14 questions per incident and was estimated to take 10 minutes to complete (*Appendix 1). Descriptive statistics were used for all data points.

The survey was closed after two months of no further responses despite multiple repeated postings on social media and in messaging groups.

Results

RESPONDENTS

A total of 54 responses were recorded, of which 18 respondents did not complete the first half of the survey, which collected individual demographics. Of the complete 36 respondents, the biological sex of 31 were male. The gender identities of the respondents were 30 men, four women, one nonbinary, and one chose not to respond. Their ages ranged from 21–73 years of age, with a mean of 45.6 (standard deviation [SD] 14.6) years. Four of the respondents reported a known patent foramen ovale, two had previous strokes, and one had a previous spinal cord injury. Three of the respondents had previously had surgery on their back and/or neck and two had previously had brain surgery.

Respondents had a range of 2–45 years of experience diving, with a median of 11 (interquartile range [IQR] 23). The types of breath-hold diving they participated in included recreational freediving (33), competitive freediving (24), spearfishing (14), underwater rugby and/or hockey (5), underwater target shooting (1), underwater aquathlon (1), and fin swimming (1).

Thirty-three of the respondents (92%) held a certification from an accredited training agency. Certifying agencies included Association Internationale pour le Développement de l'Apnée (AIDA) (12), Confédération Mondiale des Activités Subaquatiques (CMAS) (12), Molchanovs (9), Scuba Schools International (SSI) (8), Freediving Instructors International (4), Professional Association of Diving Instructors (4), Apnea Academy (2), Apnea Total (2), Performance Freediving International (2), National Association of Underwater Instructors (1). Levels of certification the respondents had achieved included 10m depth (1), 20m depth (1), 30m depth (3), 40m depth (1), instructor (13), instructor trainer (9), competitor (10), safety diver (8) and judge (1). Thirty of the respondents (83%) were also certified in scuba.

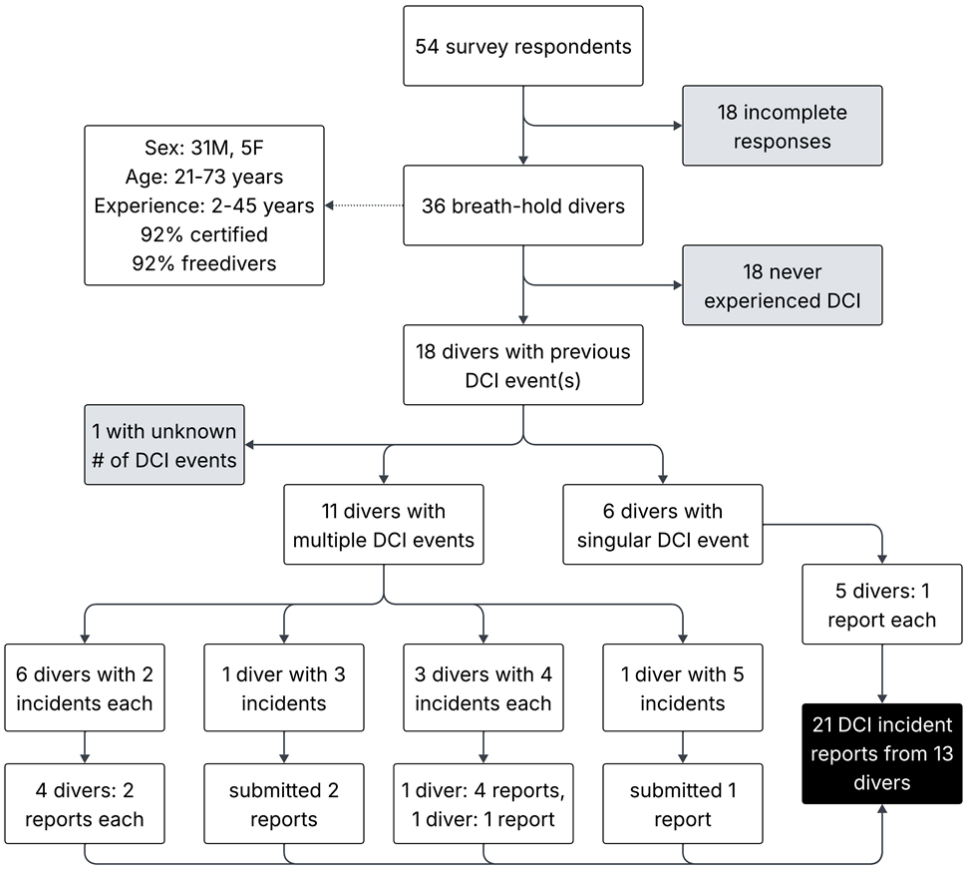
Regarding decompression terminology, respondents were familiar with the terms decompression sickness (32), arterial gas embolism (29), decompression illness (30), and Taravana Syndrome (17). Eighteen (50%) reported experiencing a decompression related event. Eleven (65%) respondents reported to have experienced more than one event (Figure 1).

DECOMPRESSION ILLNESS INCIDENTS

A total of 24 DCI incidents were reported in the survey, of which three incidents had incomplete data. The reported incidents occurred between the years of 1999–2024. The 21 complete incidents were from 13 individual divers (Table 1). The types of dives resulting in the incidents were training

***Footnote:** Supplementary Appendix 1 is available to view at https://www.dhmjournal.com/index.php/journals?id=375

Figure 1
Breakdown of survey respondents; DCI – decompression illness; F – female; M – male



(16), competition (2), recreational (2), and instructing (1). The disciplines resulting in the incidents were free immersion (FIM, 7), constant weight monofin (CWT, 4) constant weight bi-fins (CWTB, 2), scooter (2), constant weight no fins (CNF, 1), and unknown (5). The target depth of the incident dives ranged from 20–127 m (mean 83.5 m, SD 28.9 m) and the achieved depths ranged from 20–127 m (mean 83.4 m, SD 26.8 m). Thirteen (62%) of the incident dives were shallower than a previous personal best (PB); three (14%) were deeper, two (9.5%) were the same depth as a previous PB, and three (14%) were unknown. The speed of the incident dives ranged from 0.5–2.0 $m \cdot s^{-1}$ (mean 1.0 $m \cdot s^{-1}$, SD 0.4).

Fifteen (71%) of the incident dives involved pre-dive lung 'packing' (glossopharyngeal insufflation), with a range of 1–50 packs (mean 21.3, SD 11.9). On sixteen (76%) of the incident dives, divers did not perform pre-surfacing exhalation. Three (14%) of the incident dives had concurrent blackout in addition to DCI. Two of the three blackouts (66%) were underwater.

The symptoms described during the DCI incidents included constitutional, neurological, cardiopulmonary, and dermatological. Treatment modalities included cardiopulmonary resuscitation (CPR, 1), surface oxygen (14), in-water recompression (IWR, 9), and chamber-based hyperbaric oxygen treatment (HBOT, 7). Surface oxygen administration ranged from 10–90 min (mean 50, SD 26.3). IWR regimens ranged from 6–29 min at depths of 5–18.7 m; HBOT regimens ranged from 1–11 sessions (mean 4.9, SD 4.3) with various tables used (Table 2). After treatment, eight had complete resolution of symptoms, eight had partial resolution of symptoms, two had no change in symptoms, and three were unknown (Table 3).

Eleven of the incident dives were reported to the Divers Alert Network (DAN). Divers sought medical evaluation after 15 of the incident dives, and nine were admitted to the hospital for their symptoms. Medical workup included laboratory studies (7), computed tomography (6), ultrasonography (6), and magnetic resonance imaging (4). The respondents reported what they perceived as the plausible cause(s) of DCI incidents as repetitive dives with short surface interval (5), depth of the dive (5), pulmonary barotrauma (4), duration of dive (3), fast ascent (3), diving beyond their limits (2), overpacking (2), exertion at depth (2), no pre-surface exhale (2), blackout (1), dehydration (1), exhaustion (1), and competition nerves (1).

Table 1

DCI incidents and reported symptoms; AMS – altered mental status; CP – chest pain; L – left; R – right; SOB – short of breath

Diver	# Reports	Symptoms											
		Weakness	Numbness	Δ Speech	AMS	Δ Vision	Hearing	Ataxia	Fatigue	Dizzy	SOB	CP	Other
1	2	x	x						x	x	x	x	
		x	x		x				x	x	x	x	
2	1												itching to arms/legs
3	1		x										
4	2		R leg										
		legs	legs	x	x	narrowed	x	x	x	x	x	x	
5	1	x		x	x	x		x	x	x			
6	1	x											
7	1	R leg	R leg						x		x		
8	1	x											
9	4							x					headache
		L side	arms	x		blurry		x	x	x	x		
		L arm		aphasia		blurry			x		x		
		L arm		x					x	x	x		
10	2	x	face		x			x	x	x			
		R side	x	x						x			nausea/vomiting
11	2	x	x	x	x			x					
		x	x	x	x	x		x		x			
12	2		R arm								x		
		x		x	x	blurry		x					
13	1	R side		x	x				x				

Table 2
Recompression treatments reported by respondents; HBOT – hyperbaric oxygen treatment; US – United States

Recompression modality			
In-water recompression		**HBOT in a hyperbaric chamber**	
Time	**Depth**	**Protocol**	**Sessions**
5 minutes	5 m (repeat x 4)	US Navy Table 6a	1
6 minutes	5 m	US Navy Table 9	1
10 minutes	6 m	unknown	5
20 minutes	7–10 m	US Navy Table 6	5
20 minutes	15 m	US Navy Table 6a THEN US Navy Table 9	10
8 minutes 3 minutes 8 minutes 10 minutes	18.7 m THEN 15 m THEN 10 m THEN 5 m	US Navy Table 6a THEN Modified US Navy Table 5	11

Table 3
Outcomes after different treatment modalities reported by respondents; HBOT – hyperbaric oxygen treatment; IWR – in water recompression

Treatment modality			Symptom resolution
Surface oxygen	**In-water recompression**	**HBOT**	
No Treatment (1)			Unknown (1)
Surface O_2 Only (9)			Complete (3) Partial (3) Unchanged (2) Unknown (1)
	IWR Only (3)		Complete (2) Unknown (1)
Surface O_2 + IWR (1)			Complete (1)
Surface O_2		+ HBOT (2)	Partial (2)
	IWR + HBOT (3)		Complete (2) Partial (1)
Surface O_2 + IWR + HBOT (2)			Partial (2)

Discussion

This survey represents responses from a group of majority male middle-aged divers with a decade of breath-hold diving experience. Most of them were certified by a freediving organisation and two thirds of them participated in competitive freediving. Several respondents reported more than one DCI incident. Most incidents occurred during training, which may have resulted in less access to medical resources than a competition setting.

Most DCI incidents occurred during deep dives, with mean depths deeper than 80 m. Divers often attributed depth as a cause of DCI, likely due to deeper dives taking longer, thereby allowing for gas diffusion into fast tissues. It has been postulated that venous gas emboli that develop at depth may enter the arterial side through intrapulmonary arteriovenous anastomoses[10] in the absence of a patent foramen ovale (PFO), that nanobubbles can form on active hydrophobic spots within blood vessels,[11] and that profound cerebral hypotension at depth can result in low perfusion and cerebral infarct.[12]

Of the four divers who had a known PFO, three reported experiencing a DCI incident. While the survey did not specifically ask if the PFO was discovered before or after the DCI incident, it is possible that these divers only know about their PFO at the time they filled out the survey because they had an agitated saline contrast echocardiogram 'bubble study' in the aftermath of their incident. If they did discover this, it is interesting that none of the divers considered the PFO to be the cause of their DCI incident.

Multiple divers reported short surface intervals between repetitive dives as a primary cause for a DCI incident. If divers completed multiple dives with short surface intervals, it is plausible that nitrogen loading in fast tissues could result in DCI. Predictive modelling has led to a recommendation of a surface interval twice the duration of the dive,[13] although many freedivers adhere to even longer intervals beyond certain depths.[14]

A majority of divers lung-packed before the dive and did not perform a pre-surface exhale, which may increase the risk of pulmonary barotrauma from overexpansion, which can create communication between the pulmonary and arterial systems and lead to gas embolisation. While packing is not encouraged in recreational courses, it is still common practice amongst competitive breath-hold divers. In addition to the neurological symptoms expected in DCI, some divers additionally reported cardiopulmonary symptoms that could indicate concurrent pulmonary barotrauma.

Four divers reported a concurrent blackout on the incident dive. Blackouts are often attributed to hypoxia of ascent, which can also present as a loss of motor control. Many constitutional or generalised neurological symptoms reported in these incidents could be due to transient hypoxia or inert gas narcosis from the breath-hold dive. This may explain why some incidents resolved after surface (normobaric) oxygen protocols as short as 10 minutes. In DCI, normobaric oxygen should be applied as a bridge to HBOT, with a minimum of 30 minutes and average treatment time of over 2 hours.[15]

One diver reported receiving cardiopulmonary resuscitation (CPR) before other treatments. This is likely due to the underwater blackout that occurred on this incident dive, resulting in an aspiration/drowning event. It is therefore possible that the symptoms reported in this case are sequelae of the cardiopulmonary arrest and not decompression illness.

Many divers self-treated with IWR using a wide range of recompression regimens. The lack of medical oversight is likely why there was no agreement between the utilised regimens, as none follow previously published protocols meant to be administered by divers trained in decompression procedures.[16,17] We suspect that IWR was self-administered when there was delayed or no access to a hyperbaric chamber, as many training and competition sites are far from chambers. Cost may also be a factor, as oxygen may be readily available from a dive shop whereas medical treatment can be a significant expense in many locales. Many dive insurance companies do not cover competition dives, and therefore competitors may have attempted to substitute on-site IWR for otherwise costly HBOT.

Only one third of the divers received HBOT, which is the gold standard for DCI. This could be due to location and cost issues, or due to symptoms resolving with normobaric oxygen and/or IWR. It could also be due to lack of medical knowledge about DCI in breath-hold divers leading to medical facilities denying or withholding HBOT for neurological symptoms after breath-hold diving.

LIMITATIONS

The authors acknowledge that this was a retrospective survey with self-reported responses. The survey title included the phrase ‘decompression illness’, therefore only breath-hold divers with familiarity with DCI may have responded. Additionally, divers who perceived that they knew what DCI was may have responded but may have discovered in the course of filling out the survey that they had not in fact experienced DCI, thereby resulting in less than half of the original respondents actually submitting an incident report.

The respondents were athletes and may have omitted details that are relevant to medical providers in the diagnosis of decompression-related injuries. Some reported incidents that occurred years before taking the survey, and it is possible that memories of the event were modified over time or are inaccurate.

This survey did not collect information on surface intervals between previous dives and therefore cannot draw conclusions on whether a diver’s surface interval could have led to DCI. This survey did not ask about any potential adverse events as a result of medical treatments. Not all divers volunteered contact information for follow-up or clarification of their incident(s).

Conclusions

This retrospective survey sought to collect data about decompression illness in breath-hold divers from self-reported incidents. We found that breath-hold divers can experience symptoms consistent with decompression illness even after a single dive. Deep dives, short surface intervals between dives, and concurrent pulmonary barotrauma were the most cited suspected causes of DCI. Many incidents resolved after normobaric oxygen or self-administered in-water recompression, with only a third of divers receiving gold-standard hyperbaric oxygen therapy in a chamber. Less than 75% of divers sought formal medical evaluation. The IWR regimens utilized varied and did not follow any previously published IWR treatment regimens. Further research is needed to determine the best management of breath-hold diving related DCI and consensus on treatment regimens.

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Effects of fluid loss on the physiology of closed-circuit rebreather divers after 100- and 45-metre dives

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Decompression sickness; Dehydration; Diving medicine; Risk management; Technical diving; Vascular gas emboli

Abstract
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Introduction: Diving induced immersion diuresis predisposes divers to dehydration. Dehydration is considered a risk factor for decompression sickness (DCS) but there is very little evidence to prove it. Dehydration also potentially modifies venous gas emboli (VGE) formation and impairs endothelial function. The purpose of this study was to report the effects of fluid loss during a dive on the diver's physiology.
Methods: Nine divers performed a 45 metre fresh water (mfw) and a 100 mfw dive with predetermined dive profiles. Body weight was measured before and after the dive. Post-dive detection of VGE was performed according to the extended Eftedal-Brubakk scale. We also measured haematocrit and flow mediated dilation before and after the 100 mfw dives.
Results: After a 68-minute dive to 45 mfw, median weight loss was -1.1 kg, (IQR -1.2, -1.0; range -2.0, -0.6), $P = 0.009$ and VGE were detected in all divers. After a 170-minute dive to 100 mfw, median weight loss was -1.5 kg (IQR -1.8, -1.1; range -2.2, -0.8), $P = 0.009$ and VGE were detected in seven divers. Weight loss after the dive was statistically significant and there was a negative correlation between weight loss and bubbling after the 45 mfw dives. None of the divers suffered any symptoms of DCS.
Conclusions: We found significant weight loss after both decompression dives but there were no clinical DCS symptoms in any of the divers. This study does not offer new evidence supporting the notion that dehydration increases decompression stress in divers.

Introduction

Scuba dives, and especially deeper and longer technical dives, pose stress on the diver's body. Due to increased ambient pressure underwater, inert gasses dissolve in tissues and during ascent, inert gases are eliminated from the tissues. During the off-gassing, venous gas bubbles tend to form and these bubbles initiate a complex cascade of events that can lead to decompression sickness (DCS).[1] The deeper the dive, the more inert gas dissolves and divers need to decelerate ascent speed by adding decompression stops to allow sufficient time for off-gassing. Hence, great depths result in long dive times and increased decompression stress and risk of DCS.

Increased hydrostatic pressure also causes redistribution of venous blood from the caudal portions of the body to the intrathoracic circulation. This is augmented by cold-induced vasoconstriction that reduces heat loss by constricting peripheral thermoregulatory shunts yet increasing centralisation of blood flow. As a result, central blood volume and cardiac preload increase which triggers humoral responses leading to immersion diuresis and

decreased plasma volume. The effect of immersion on haemodynamic and fluid rearrangements is well established in the literature.[2–4] In addition to immersion-induced diuresis, divers lose fluids via respiration and evaporation, although diving with closed-circuit rebreathers (CCRs) reduces fluid loss due to humid breathing gas.

Increasing diuresis predisposes divers to dehydration. Dehydration is considered to be a risk factor for DCS but there is very little evidence to prove it.[1] There are also conflicting results regarding the role of fluid balance in the DCS risk in animals.[5–7] It is speculated that the risk of DCS increases because of dehydration and subsequent haemoconcentration due to higher blood viscosity and reduced perfusion of peripheral tissue such as skin and skeletal muscle which would reduce inert gas washout during decompression.[6]

Dehydration might also impair endothelial function.[8] An increase in blood flow-associated shear stress in blood vessels induces the release of vasodilators e.g., nitric oxide (NO) from the endothelium. This phenomenon is called flow-mediated vasodilatation (FMD). Flow-mediated vasodilatation is an important response regulating homeostasis of the peripheral circulation.[9,10]

Previous studies suggest dehydration modifies venous gas emboli (VGE) production. Gempp et al. found pre-dive fluid intake reduces VGE formation in divers.[11] On the other hand, Skogland et al. did not find dehydration causing more VGE in rats.[6] Though VGE potentially contribute to development of DCS, VGE grades are considered an imperfect outcome measure in DCS studies. Yet, the number of bubbles does have a weak positive predictive value, in correlation to the symptoms of decompression sickness.[12]

Dehydration is only one of the factors considered to predispose divers to DCS. However, the knowledge regarding dehydration and the risk of DCS is scarce, there are no human studies, using DCS as an endpoint, addressing this topic. The purpose of this study was to report the physiological effects of fluid loss in a group of divers that performed two different decompression dives.

Methods

The study adhered to the Declaration of Helsinki. Ethical approval was granted by the Ethical Committee of Helsinki University Hospital (HUS/976/2019). Research permission was received from Helsinki University Hospital (HUS/151/2022 and 124/2023).

STUDY DESIGN

Nine experienced, healthy and non-smoking subjects, male ($n = 8$) and female ($n = 1$), took part in the tests. The subjects were recruited from the Finnish recreational technical diving community. Two of the divers had experienced DCS in the past. None of the divers had any other medical conditions. Each diver performed one dive to 45 metres of fresh water (mfw) and one dive to 100 mfw. Each subject filled out a health survey, and a physician performed a fit-to-dive examination on the morning of the dive.

PREPARATIONS AND DIVING PROTOCOL

No alcohol was allowed for 24 hours before the dive. During the 45 mfw diving day, subjects were instructed to hydrate according to their regular routines until two hours before the dive. Thereafter, only 5 dL of sports drinks (Gatorade, PepsiCo, Nordic Finland Ltd, Helsinki, Finland) were consumed. For the 100 mfw dive, the divers were allowed to hydrate as they normally do. Preparations for the dives were made in a room with constant air temperature (19°C).

The 45 mfw test dives were conducted at an old water-filled mine in Ojamo (Lohja, Finland) during winter in January. Diving conditions were normal for this time of year: the water was covered with a thin sheet of ice; water temperature was 0–2°C near the surface and 4°C at a depth of 45 mfw. The divers made an identical dive to the depth of 45 mfw by following a preset line. The divers were instructed to start the ascent at 30 min runtime resulting 15 min time at the bottom depth. During the ascent, divers followed an earlier defined decompression profile: Suunto Fused™ RGBM 2 (Suunto Ltd, Vantaa, Finland) with personal adjustment +2 (Suunto EON Core and Suunto D5 dive computers). The median total dive time was 68 min (interquartile range [IQR] 63–71 min).

The 100 mfw test dives were conducted at an old water-filled mine in Montola (Pieksämäki, Finland) during one weekend in October. The water temperature was 4°C at depths below 25 mfw and 8°C above 25 mfw. The divers also followed an identical route to the depth of 100 mfw and spent five min at the bottom depth before starting the ascent. Decompression was performed according to Shearwater computers (Shearwater Research Inc, Richmond, BC, Canada) using gradient factor (GF) 20/70. The median total dive time was 170 minutes (IQR 155–178 min).

Divers used their own diving equipment during the test dives. These included their usual undergarments, heating vest and dry suits. The divers were allowed to use pee-valves during the dive, the amount of urine output was not measured. All subjects used their own CCR unit (JJ-CCR [$n = 8$], rEVO [$n = 1$]). All CCR devices used standardised diluent; trimix 20/40 for the 45 mfw dive and 10/70 for the 100 mfw dive. The oxygen controllers maintained constant oxygen partial pressure in the breathing loop (PO_2 = 70 kPa at the beginning of the dive and PO_2 = 120 kPa after reaching 21 mfw throughout the bottom time and ascent).

MEASUREMENTS

Preparations and measurements were made in a room with constant air temperature (19°C). Subjects' weight was measured with an InBody 720 composition analyzer (Biospace Ltd, Seoul, South-Korea) approximately two hours before the dive. After the dive, subjects were instructed to empty the bladder and be weighed as soon as possible before any fluid intake. Weighing was done wearing only underwear (no diving undergarments were allowed) before and after the dive.

The presence of VGE in the cardiac chambers was determined with a 2D echocardiographic probe using an apical four-chamber view with a transthoracic approach (GE Vivid i, GEMS ultrasound, Tirat Carmel, Israel, transducer 2D 3S-RS). Subjects were placed in the supine left lateral decubitus position. Monitoring was performed at 30, 60, 90 and 120 minutes after surfacing, at rest and after performing Valsalva and leg and arm flexion. The observation was recorded and verified with at least one additional observer. Obtained images were graded from 0 to 5 according to the method described by Eftedal and Brubakk.[13]

The venous blood samples for measuring haematocrit (Hct) were taken from the antecubital vein 1–2 hours before and maximum one hour after the 100 mfw dives. Haematocrit was measured using the capillary (microhaematocrit) method. Blood samples were collected in heparinised microcapillary tubes, sealed at one end, and centrifuged at 10,000–12,000 rpm for five minutes to separate red cells, buffy coat, and plasma. The haematocrit value was then determined as the ratio of packed red cell column length to the total blood column length, expressed as a percentage.

Using a digital diagnostic ultrasound system (V-Scan Air, General Electric-Netherlands), FMD, an established measure of the endothelium-dependent vasodilation mediated by NO,[10] was used to assess the effect of diving on main conduit arteries after the 100 mfw dives. Brachial artery diameter was measured immediately before and one minute after a five-minute ischaemia induced by inflating a cuff placed on the forearm.[10] All ultrasound FMD assessments were obtained 60 min after surfacing while participants stayed at rest in the supine position for at least 15 min. During image analysis, the brachial artery boundaries were identified manually with an electronic caliper (provided by the ultrasonography software) in a threefold repetition pattern. The artery diameter was averaged over these three measurements. FMD was calculated as the percent increase in arterial diameter from the resting state to maximal dilation.

STATISTICS

We present the numerical data using medians, interquartile ranges (IQRs) and ranges, and the categorical data as counts and percentages. Comparisons were done using Mann-Whitney U tests and Spearman correlation (ρ) with 95% confidence intervals (CI). We visually assessed the normality of the variables and decided to use non-parametric tests. We considered *P*-values below 0.05 significant. All analyses were done using R software version 4.5.0 and the ggplot2 package was used for creating the figures.[14]

Taking the baseline measures as 100%, FMD changes were calculated for each diving protocol, allowing an appreciation of the magnitude of change rather than the absolute values.

Results

Median age of the divers was 45 years (IQR 42–50 years) and they had a long experience in diving, median experience was 16 years (IQR 14–19 years). All nine divers (one female, eight male) completed both of the decompression dives as planned. After diving and on the following day controlled, none of the divers presented any symptoms suggesting a diving incident. Subjects' demographics are presented in Table 1.

After a 68 min dive to 45 mfw, the median weight loss was -1.1 kg (IQR -1.2, -1.0; range -2.0, -0.6; $P = 0.009$) and after a 170 min dive to 100 mfw, the median weight loss was -1.5 kg (IQR -1.8, -1.1; range -2.2, -0.8; $P = 0.009$). Weight loss after the dives is presented in Figure 1. Weight loss relative to diver's body mass was 1.39% (IQR 1.04, 2.04; range 0.8, 3.1) after the 45 mfw dive and 2.05% (IQR 1.26, 2.4; range 0.9, 2.6) after the 100 mfw dive.

After the 45 mfw dives venous inert gas bubbles were detected in all nine divers during the 120-minute follow-up. After the 100 mfw dives, venous inert gas bubbles were detected in seven divers. Two of the divers did not produce any visible bubbles, not even when provoked with Valsalva manoeuvres and/or limb flexions. One diver with no history of DCS expressed a few occasional bubbles in the left heart also after the 45 mfw dive. There was a statistically significant negative correlation between weight loss and

Table 1
Subject demographics before the 100 mfw dive; data are median (interquartile range) unless otherwise indicated; BMI – body mass index; DCS – decompression sickness

Parameter	*n* = 9
Age (years)	45 (42–50)
Weight (kg)	82.9 (80.5–87.1)
BMI (kg·m^{-2})	26.5 (25.3–28.4)
Diving experience (years)	16 (14–19)
Diving experience (dives)	850 (700–1500)
Previous DCS (*n*)	2

Figure 1
Weight change (kg) after 45 mfw/68 min and 100 mfw/170 min dives; the median weight loss was -1.1 kg ($P = 0.009$) and -1.5 kg ($P = 0.009$) respectively

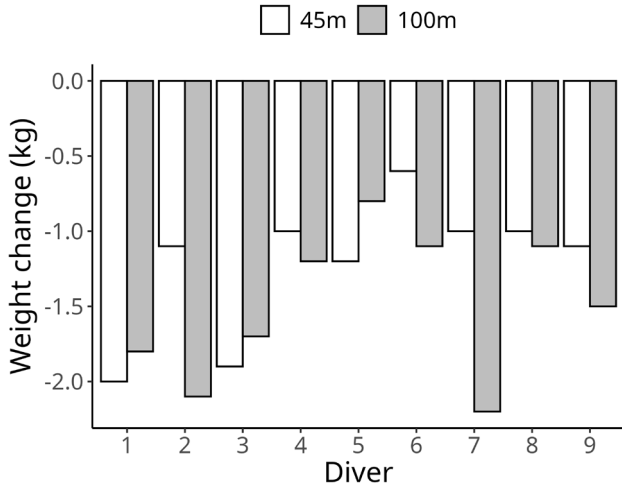
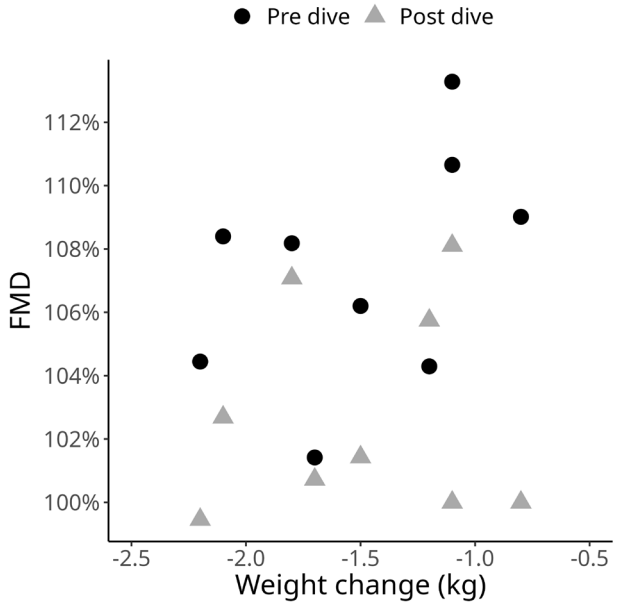


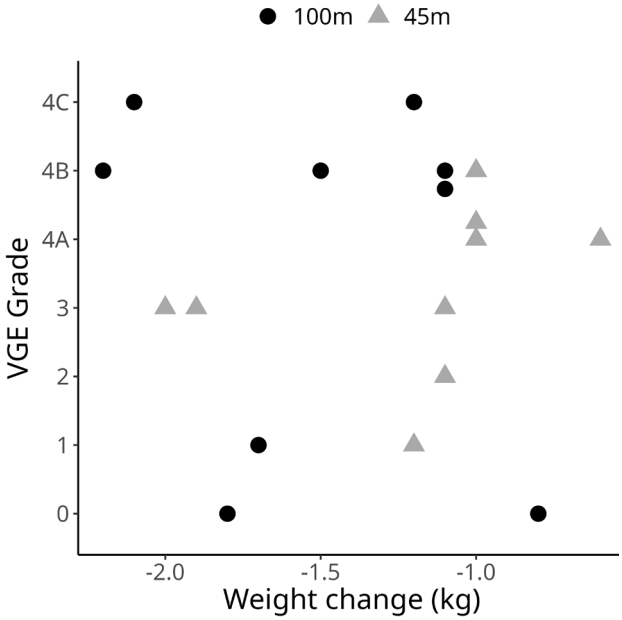
Figure 3
The flow-mediated dilation (FMD) measured before and after the 100 mfw dives and its relation to the divers' weight change. There was no correlation between FMD and weight change



bubbling after the 45 mfw dives (ρ -0.72; 95% CI -0.95, -0.01; $P = 0.028$) but no correlation after the 100 mfw dives (ρ 0.20; 95% CI -0.54, 0.77; $P = 0.601$). The relation between VGE and weight change is presented in Figure 2.

After the 100 mfw dives, the haematocrit decreased by 0.8% (IQR -2.8, 2.0; range -3.7,11.0; $P = 1.0$). FMD decreased by 5.0% (IQR -5.7, 1.1; range -10.7, 1.5; $P = 0.020$). There was no correlation between haematocrit and weight change (ρ 0.16; 95% CI -0.57, 0.75; $P = 0.30$) or FMD and weight

Figure 2
The relation between maximum venous gas embolism grade and weight change; there was a statistically significant negative correlation after the 45 mfw dives but no correlation after the 100 mfw dives



change (ρ -0.36; 95% CI -0.83, 0.42; $P = 0.34$). The relation between FMD measured before and after the 100 mfw dives and weight change is presented in Figure 3.

Discussion

In this study, we focused on the effects of fluid loss and decompression stress, and furthermore on DCS risk factors in a group of divers after two decompression dives to different depths. The same nine divers did a short 45 mfw dive and a longer and deeper 100 mfw dive in cold water. Though the 100 mfw/170 min dive in cold water poses great decompression stress on divers' bodies, we did not observe any symptoms of DCS. Weight loss was statistically significant after both diving depths.

The divers lost weight up to 2.2 kg, corresponding to a volume depletion of 2.2 liters. Overall, weight loss was greater after the 100 mfw dive, though three of the divers lost more weight during the 45 mfw dive. The difference in weight loss between the 45 mfw/68 min and 100 mfw/170 min dives was not clinically very significant, being 1.1 kg and 1.5 kg respectively. These results are in line with previous studies that report weight loss after diving indicating fluid loss due to immersion.[11,15,16] There are also data showing that during hyperbaric conditions, divers also become dehydrated without water immersion.[17]

We found a statistically significant, negative correlation between weight change and VGE after the 45 mfw dives.

This result suggests dehydration might decrease VGE formation. However, due to the small size of our study population, which may result in an imprecise correlation estimate, causal interpretations should be made with caution. This finding is in concordance with Skogland et al. study, that found no difference in VGE formation between dehydrated and normally hydrated rats.[6] Yet the only human study Gempp et al. found pre-dive fluid intake reducing VGE formation in eight divers.[11] Given the limited number of divers in both studies, the findings may be influenced by random variability. In our study, the divers were allowed eat and drink in the morning according to their usual routines (approximately 4–5 hours before the dive). The controlled fluid intake was only two hours before the dive and the amount was less than in the study by Gempp et al,[11] being 500 ml vs 1,300 ml.

Weight loss was significant after the 100 mfw dive, but we did not find a significant difference in haematocrit, showing that the compensatory mechanism shifting fluid from the extravascular compartment to the vascular one was not overwhelmed. Therefore, despite the fluid loss, divers seemed to retain intravascular volume and we found no correlation between weight change and VGE formation after the 100 mfw dives. For safety reasons, the fluid intake was not controlled before the 100 mfw dives, most probably resulting greater fluid intake than before the 45 mfw dives (observed at the dive site but not quantified). The deeper dives were also done in warmer decompression water temperature and with a different decompression algorithm that produces longer decompression stops at the shallow depth. Hence, inert gas washout should be better during 100 mfw dives. Overall, after the 100 mfw dives, the divers had more VGE as predicted after deeper dive and greater helium content in the breathing gas. Yet, there were two divers with no visible bubbles even after limb movement after the 100 mfw dives. The intra- and interpersonal variability in bubbling in our study is also is in line with previous studies.[12,18]

The divers in this study experienced fluid deficit (percentage reduction in body mass due to fluid loss) up to 3.1% and 2.6% after the 45 mfw and the 100 mfw dives respectively. The state, when fluid deficit exceeds 2% of body mass, is called hypohydration. Hypohydration results in water redistribution largely from the intra- and extracellular fluid spaces of muscle, gut and skin.[19] Divers dehydrate via immersion induced diuresis, respiration and evaporation. In order to preserve homoeostasis, the body immediately counterbalances by shifting extravascular water into the intravascular space. Fluid shift effects on blood volume and hemoconcentration. For example, haematocrit level depends on the difference between intracellular and extracellular dehydration, and if the loss of extracellular water is lower than the loss of intracellular water, we observed a decrease in the haematocrit level and vice versa. This might explain why we did not see a statistically significant change in haematocrit level in our study, though divers lost significant amount of weight indicating fluid loss.

Dehydration of up to 2% should not pose a threat to health or even be noticeable in normal life.[20] But hypohydration has been shown to possibly affect cognition; the loss of body mass of greater than 2% can lead to reductions in the subjective perception of alertness and ability to concentrate.[21,22] It has been suggested that for every 1% of total water loss, the body's physical capacity decreases by approximately 10%.[17] Hypohydration also leads to vasoconstriction and therefore causes a reduction in blood flow to the skin.[23] Blood flow also declines in the exercising muscles due to a lowering in perfusion pressure and systemic blood flow,[24] thus potentially compromising inert gas washout from tissues when diving.[1] Hence, for safety reasons, divers were not instructed to hydrate according to a certain protocol before the 100 mfw dive: The divers were allowed to consume drinks according to their usual procedures. All of the divers started focusing on hydration at least the day before the dive and all of them drank more than 500 ml in the morning of the dive.

Hydrating well with water or isotonic drinks before the dive is common among divers, especially technical divers. Increasing total body water above the normal, referred to as pre-exercise hyperhydration, provides a strategy to delay or reduce the adverse effects of hypohydration caused by exercise. But hydrating with large amounts of fluid alone is not reasonable as it inhibits the release of anti-diuretic hormone (ADH), leading to increased diuresis.[25] Also, hyperhydration may increase the risk of immersion pulmonary oedema among other risk factors, however the research to support this risk in scuba divers is limited.[26] Recent review of nutritional recommendations for scuba divers suggests all divers should take special care to hydrate themselves with an absolute minimum of 500 ml of fluid per hour when diving for more than 3 hours.[27]

FMD decrease reached 5% compared to the pre-dive value; this statistically significant change is very common in diving and does not seem to change in relation to the depth; it seems to be more related to the increase in inspire PO_2.[28,29] Previous studies suggest even mild levels of hypohydration impair endothelial function as assessed by FMD,[8] but we did not find any correlation between weight loss and FMD; thus, our results show a rather 'constant' acute vascular function reduction after diving.

LIMITATIONS

Our study was performed in a real diving environment with challenging conditions. The number of participants was limited by the depth of the dives and type of study, and therefore the results comparisons between the groups should be interpreted with caution. With this limited number of

divers in our study, the observed results may fluctuate due to underlying stochastic variability. A larger study group could have given a more precise understanding, and the correlations in various parameter could have been stronger.

For safety reasons, the fluid intake before the 100 mfw dive was not controlled as well as the decompression model used was not the same making comparison between the dive depths difficult. Another limitation of the study was the unavailability of FMD and Hct measurements in the 45 mfw dive. This did not allow comparison of these parameters in two different dive profiles and environmental conditions.

Conclusions

In conclusion, we found significant weight loss after both decompression dives and there was a negative correlation between weight loss and VGE after the 45 mfw dives. Because our study population is relatively small, leading to less precise correlation estimates, caution is needed when interpreting causal relationships. There were no clinical DCS symptoms in any of the divers. There was also no correlation between weight loss and haematocrit or FMD after the 100 mfw dives. This study does not offer new evidence supporting the notion that dehydration increases decompression stress in divers. Further studies with a greater number of participants are needed to potentially support the widely accepted concept that dehydration predisposes divers to DCS.

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Review articles

Hyperbaric oxygen therapy for acute idiopathic sudden sensorineural hearing loss; a systematic review with meta-analysis

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Keywords
Deafness; Inner ear; Treatment; Review article

Abstract
(Newth A, Perleth M, Sherlock S, Romero L, Bennett MH. Hyperbaric oxygen therapy for acute idiopathic sudden sensorineural hearing loss; a systematic review with meta-analysis. Diving and Hyperbaric Medicine. 2025 December 20;55(4):398–406. doi: 10.28920/dhm55.4.398-406. PMID: 41364864.)
Introduction: Idiopathic sudden sensorineural hearing loss (ISSHL) is hearing loss of unknown cause with greater than 30 dB loss over 72 hours or less across three consecutive frequencies. Hyperbaric oxygen therapy (HBOT) is a widely accepted treatment for this condition. HBOT protocols and outcomes measured vary between studies.
Methods: To update a systematic review with meta-analysis of relevant randomised trials to both quantify and estimate the quality of evidence to support or refute the use of HBOT for ISSHL. We followed the Cochrane Handbook for Systematic Reviews of Interventions methodology. We conducted a focussed search of the following databases – AMED, BIOSIS Previews, CENTRAL, CINAHL, Embase, Emcare, Global Health, Medline, Scopus and Web of Science. There were no language or publication status restrictions. The updated search covered 1 April 2012 to 22 February 2023. A total of 148 papers were found with 24 randomised and pseudo-randomised studies identified of which seven contributed to the final analysis. Studies using usual treatment (steroids) plus HBOT or no treatment plus HBOT were included. The ROBB 2 tool for risk of bias and the GRADE tool for certainty of evidence were utilised.
Results: Data pooling was hampered by variation in reporting of changes in pure tone average across these studies. Pooled analysis from five studies suggested the chance of improvement following HBOT and steroids was greater than after steroids alone (RR 1.6, 95% CI 1.3 to 2.0). Pooled data from four trials suggested a greater mean improvement following HBOT (mean difference 15.6 dB, 95% CI 1.5–29.8).
Conclusions: There is moderate evidence that HBOT improves hearing when applied up to 30 days after the onset of ISSHL. HBOT in combination with steroids (oral or intra-tympanic) can be justified as a routine treatment. Future trials should address optimal dose and timing of HBOT and ensure outcomes enable pooling of data in future reviews, as well as addressing some measure of the functional significance of any improvement.

Introduction

Idiopathic sudden sensorineural hearing loss (ISSHL) is an acute hearing impairment that is usually one-sided and may be accompanied by tinnitus and vertigo. The US National Institute on Deafness and Other Communication Disorders defines ISSHL as a sudden sensorineural hearing loss of no identifiable cause, with greater than 30dB loss in at least three consecutive frequencies developing over 72 hours or less.[1] Sensorineural is further defined as "*abnormal functioning of the cochlea, auditory nerve, or higher aspects of central auditory perception or processing*".[1] While the most common proposed causes are vascular and viral, the aetiology remains elusive.

The incidence is estimated as 27 per 100,000 per year in the USA and is somewhat higher in patients 65 years and older (77 per 100,000) based on a pharmaceutical claims database.[2] A national epidemiological survey in Japan included 3,419 patients from 30 centres between 2014 and 2016 and found 31% were 65 years or older, 78% had tinnitus and 35% vertigo or dizziness. More than 90% of patients received corticosteroids, most commonly systemic.[3]

Historical treatments for ISSHL have mostly been designed to improve the blood circulation and oxygenation of the inner ear and include vasodilators, plasma expanders, steroids, anticoagulants, diuretics, contrast dye and antivirals. None have been proven of benefit in large, randomised trials or meta-analyses and for the most part the use of these agents has been abandoned.[4,5]

A UK clinical guideline recommends corticosteroids either orally, as intra-tympanic injections or a combination of both as first line-treatment for ISSHL despite no evidence supporting their benefit over placebo when given orally.[6] The guideline of the American Academy of Otolaryngology–Head and Neck Surgery Foundation additionally recommends hyperbaric oxygen therapy (HBOT) as either initial or salvage therapy in combination with steroids based on a systematic review of RCT evidence with a medium grade of confidence.[1]

The assessment of treatment response is complicated by a variable spontaneous recovery rate, with estimates ranging from 32% to 65% within 14 days quoted in the historical literature.[7] More recently, such high rates of resolution have been questioned.[1]

The variance in clinical practice in Australia and costs associated with increasing referrals for HBOT has prompted a review of the literature. The aim of this review was to perform meta-analysis, where possible, to assist in defining the benefit, if any, of the use of HBOT as adjunctive therapy to standard treatment in acute ISSHL. We specifically address the clinical question "*Does the additional administration of hyperbaric oxygen to people with idiopathic sudden sensorineural hearing loss result in an increase in the proportion attaining a useful improvement in hearing?*" We have not included any evidence concerning the treatment of long-standing hearing loss, nor any impact on the severity of the tinnitus that often accompanies acute hearing loss.

Methods

SEARCH STRATEGY

The Methodological Expectations of Cochrane Intervention Reviews (MECIR) reporting guideline was used. The same search algorithm which was used in previous updates was applied. This update was performed as it provides more up to date information to address health decisions. Electronic searches were conducted in 10 databases (Ovid Medline, Ovid Embase, Ovid Global Health, Ovid Emcare, AMED, CINAHL, Biosis Previews, Cochrane CENTRAL, Scopus and Web of Science). After an initial search for articles in MEDLINE and Embase, an analysis of the text words contained in the title and abstract, and of the index terms used to describe these articles was conducted. A second search using identified key words and subject index terms was then undertaken from database inception to 22 February 2023 across all ten databases. The search strategies used a combination of subject headings and free text terms that aimed to cover the areas of (1) sensorineural hearing loss or tinnitus, AND (2) hyperbaric oxygenation AND (3) randomised controlled trials/pseudo-randomised controlled trials.

Searches were adapted as appropriate to the specifications of each of the 10 databases. Hand-searching and reference checking of citations and reference lists was also undertaken to identify any studies that were not retrieved in the search.

There were no language or publication status restrictions, and we restricted publication date from 1 April 2012 to 22 February 2023 acknowledging the previous search had been performed on 2 May 2012. This protocol follows the standard protocol published in the Cochrane Handbook for Systematic Reviews of Interventions.[8]

Two review authors (AN and MB) independently screened the titles and abstracts of all retrieved citations against the inclusion criteria. Three authors (AN, MB, and MP) examined the electronic search results and identified studies that may have been relevant. These studies were retrieved in full and considered for inclusion in this review (see Figure 1). The same three authors reviewed these studies independently and reached a consensus decision on inclusion or rejection for this review. If included, the data were extracted using a form developed for the original review which follows the same process described in the Cochrane Handbook for Systematic Reviews used in previous systematic reviews.

STUDIES FOR INCLUSION

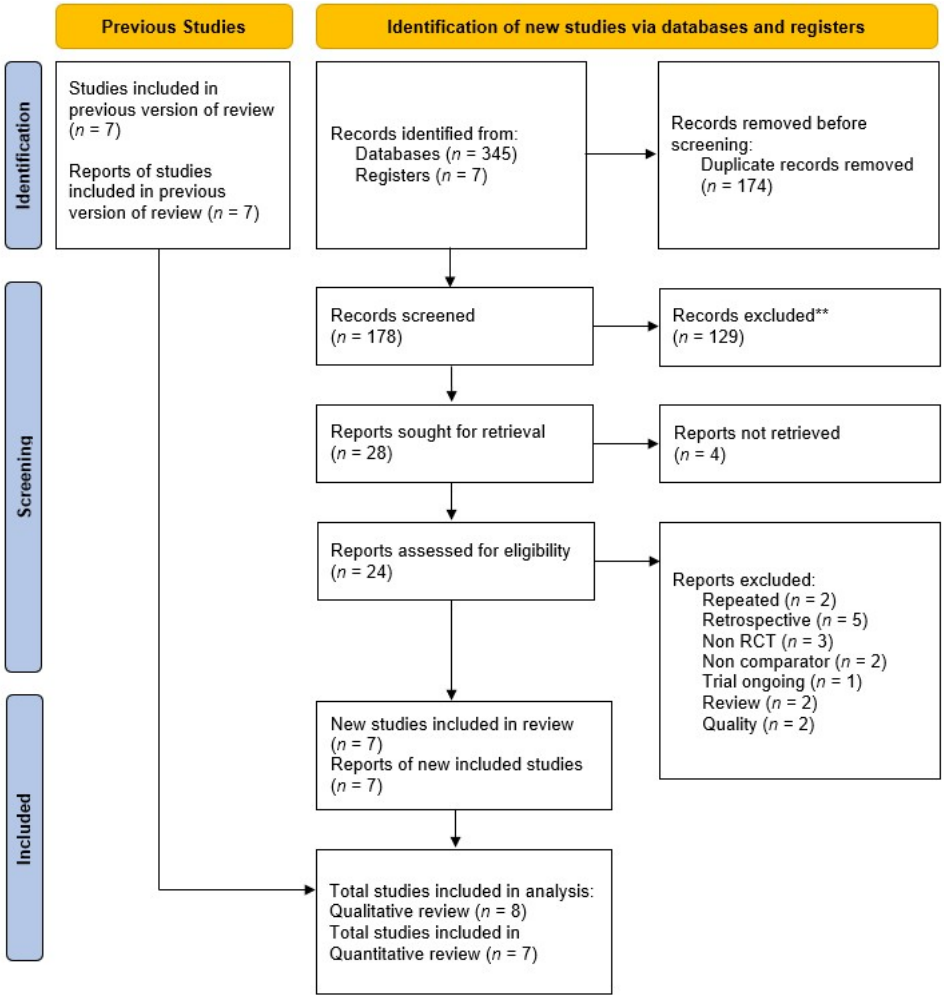
We identified and included all randomised and pseudo-randomised controlled trials that compared the effect of treatment for acute ISSHL (under two weeks versus over two weeks) in patients with ISSH, regardless of age where HBOT was included and compared to any treatment (or no specific treatment) in the absence of HBOT. We included studies irrespective of allocation concealment or blinding status.

In addition, the trials included in the systematic review must have reported an outcome relevant to our pre-defined primary or secondary outcomes. Primary outcome: a documented assessment of pure-tone audiometric thresholds at a number of frequencies (pure tone average values – PTA) after treatment. Secondary outcomes: activities of daily living (ADLs); subjective or objective improvements in depression or mood disturbance; hearing handicap inventory change; word discrimination score; or any adverse events associated with HBOT and comparators.

ASSESSMENT OF RISK OF BIAS IN INCLUDED TRIALS

Two authors (AN and MB) undertook assessment of the risk of bias of the included trials independently, with the

Figure 1
PRISMA Study Flow Diagram for updated systematic review



following taken into consideration, as guided by the *Cochrane Handbook for Systematic Reviews of Interventions.*[8] This methodology (ROB2) assesses the potential of bias from six domains – sequence generation method, allocation concealment, blinding, incomplete outcome data, selective outcome reporting and 'any other' potential bias detected by the reviewers. The GRADE methodology was also used as a tool for grading the certainty of evidence.

DATA SYNTHESIS

For proportions (dichotomous outcomes), we used risk ratio (RR). We used a fixed-effect model where there was no evidence of significant heterogeneity between studies (I^2 < 30%) and employed a random-effects model when such heterogeneity was likely. Where the 95% confidence interval (CI) did not include parity between treatments (RR = 1), we also calculated the number needed to treat (NNT) and 95% CI. For continuous data we compared the mean differences (MD) between hyperbaric oxygen and control groups and defined a statistically significant difference as existing if the 95% CI did not include a zero MD. We undertook all analyses using the RevMan Web© online systematic review tool.[9] We also intended to perform sensitivity analyses for missing data and study quality where possible. For missing data, we planned a 'best-case' and 'worst-case' approach to the imputation of missing data (best-case assumes none of the originally enrolled patients missing from the primary analysis in the treatment group had the negative outcome of interest whilst all those missing from the control group did. The worst-case scenario was the reverse). We also intended to conduct a sensitivity analysis by study quality based on our estimate of the risk of bias and an assessment of adequate sample size to detect the clinically important difference in outcome for which the study was designed.

Results

We present the main findings of this meta-analysis in Table 1.[10]

We repeated our original search strategies in February 2023 and in total, from 294 citations, we identified 24 randomised and pseudo-randomised studies of which seven contributed to this quantitative analysis (Figure 1).[11–25]

Table 1
Summary table for outcomes

Summary of all outcomes designed to show a differential improvement in hearing between HBOT and control; bold suggests benefit from HBOT; *according to Siegel's criteria; **no steroids used in trial; +calculated where proportional outcomes are statistically significant; #sensitivity analysis on removal of non-contributing studies and the mild hearing loss group reported in Topuz 2004[23]; CI – confidence interval; HBOT – hyperbaric oxygen treatment; MD – mean difference; NNT – number needed to treat; PTA – pure tone average; RR – relative risk

Outcome No.	Outcome name	Included trials	*n*	Event outcomes	Result Point est (95% CI)* (NNT and 95%CI)+
1.1	Proportion of participants with > 50% return of hearing	Cavallazi 1996[12] Fattori 2011[17]	114	HBOT: 35/64 Control 18/50	RR 1.5 (0.9–2.8)
1.2	Proportion of participants with > 25% return of hearing	Cavallazi 1996[12] Fattori 2011[17]**	114	HBOT: 50/64 Control 28/50	**RR 1.4 (1.1–1.8)** **NNT 5 (3–21)**
1.3	Proportion of participants with mean improvement of > 20 dB	Hoffmann 1995[18] Chi 2008	80	HBOT: 5/40 Control: 2/40	RR 2.2 (0.5–9.2)
1.4	Proportion of patients with a significant improvement*	Cavaliere 2022[11] Chi 2018[13] Cho 2018[14] Dova 2022[16] Hu 2020[19] Piniara 2022[25] Zhang 2022[24]	562	HBOT: 238/293 Control: 148/26	**RR 1.5 (1.2–1.7)** **NNT 4 (3–5)**
1.5	Mean improvement of PTA (% of baseline)	Fattori 2011[17] Hu 2020[19]	157	N/A	**MD 17.5% (2.5–32)**
1.6.1	Mean improvement (dB from baseline)	Pilgramm 1985[21] Piniara 2022[25] Hoffmann 1995[18] Schwab 1998[22] Topuz 2004[23] Zhang 2022[24]	341	N/A	**MD 13.0 dB (6.2–19.7)**
1.6.2#		Pilgramm 1985[21] Piniara 2022[25] Topuz 2004[23] Zhang 2022[24]	245	N/A	**MD 15.1 dB (8.2–22.0)**
1.7	Mean final PTA after treatment (dB)	Cho 2018[14] Hu 2020[19]	165	N/A	**MD 10.0 dB (2.7–17.3)**

The newly identified trials added to the analysis were published between 2012 and 2022, and the authors are aware of one possible ongoing randomised study at this time whose authors were uncontactable.[26] Four identified trials were unable to be retrieved as published in non-English publications. In total, these newly identified trials were added to historical Cochrane data resulting in 989 participants, 552 receiving HBOT and 437 control. All trials included participants with acute hearing loss of unknown aetiology, but other inclusion criteria, the dose of oxygen, comparator treatments and time to follow-up all varied across these studies. Trial details are included in Table 2. All included studies reported at least one clinical outcome of interest. Of the outcomes identified above, these trials reported data on the primary outcome (pure-tone audiometric documented change in hearing) but none of the secondary outcomes of interest.

RISK OF BIAS

The methodology of these trials was inconsistently reported. The assessments of risk of bias are summarised in Figure 2. Allocation concealment was not adequate in any of the studies. Randomisation procedures were only described in four studies where a computer-generated sequence was employed.[11,21,24,25] Allocation may not have

Table 2

Details of included studies; *three groups, HBOT/both/control; **three groups, HBOT daily/HBOT twice daily/control; #twice daily HBOT; HBOT – hyperbaric oxygen treatment; IT – intratympanic; IV – intravenous

Study	*n* Total (HBOT/control)	Intervention	Oxygen dose: kPa x mins and (number of sessions)	Maximum time from onset to treatment (days)	Control regimen	Final follow-up (days)
Cavaliere 2022[11]	171 (56/55/60)*	HBOT only	253 x 90 (15)	30	Oral steroids	20
Cavallazzi 1996[12]	62 (32/30)	HBOT + multiple drug treatment	243 x 60 (15)	Unclear	Multiple drug treatment	Treatment end
Chi 2018[13]	60 (30/30)	HBOT + oral steroids	253 x 90 (10)	14	Steroids + other drugs	180
Cho 2018[14]	60 (30/30)	HBOT + steroids (oral and IT)	253 x 60 (10)	8	Oral and IT steroids	90
Cvorovic 2013[15]	50 (25/25)	HBOT only	203 x 60 (20)	28	IT steroids	Treatment end
Dova 2022[16]	50 (25/25)	HBOT + IV steroids	223 x 80 (15)	11	IV steroids	90
Fattori 2001[17]	50 (30/20)	HBOT only	223 x 90 (10)	2	Oral vasodilator	Treatment end
Hoffmann 1995[18]	20 (10/10)	HBOT only	152 x 45 (10–20)	14	Nil	90
Hu 2020[19]	107 (38/27/42)**	HBOT + oral steroids	Not stated (10–20)	Unclear	Oral steroids	20
Krajcovicova 2018[20]	68 (47/21)	HBOT + steroids (oral and IV)	203 x 90 (10)	7	Oral and IV steroids	Treatment end
Pilgramm 1985[21]	37 (18/19)	HBOT + multiple drug treatment	253 x 60 (10)	14	Multiple drug treatment	28
Piniara 2022[25]	102 (50/52)	HBOT + steroids (IV and IT)	253 x 90 (10)	10	IV, oral and IT steroids	90
Schwab 1998[22]	75 (37/38)	HBOT only	152 x 45 (10–20)	14	Nil	28
Topuz 2004[23]	55 (30/21)	HBOT + multiple drug treatment	243 x 90 (25)	14	Multiple drug treatment including steroids	28
Zhang 2022[24]	70 (35/35)	HBOT + steroids	203 x 60 (20)	7	Steroids + other drugs	Treatment end

Figure 2
Risk of bias for all included studies across six domains

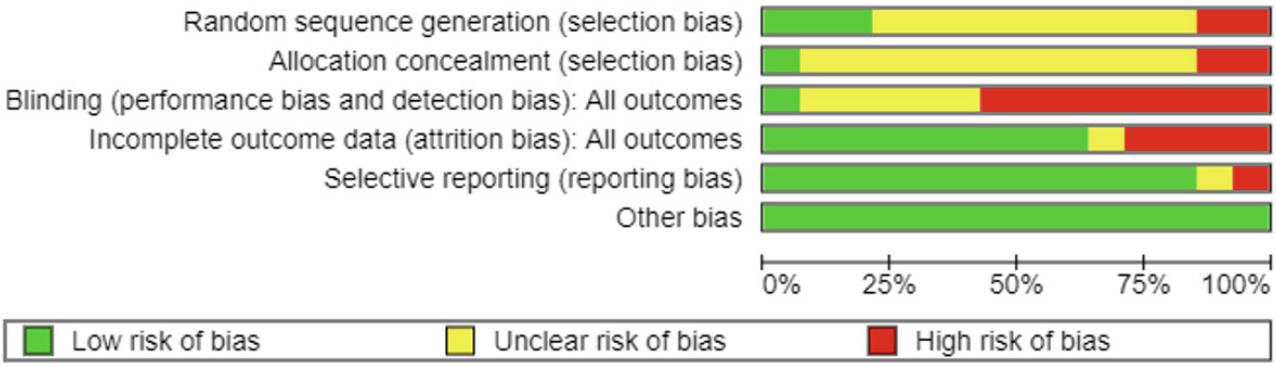
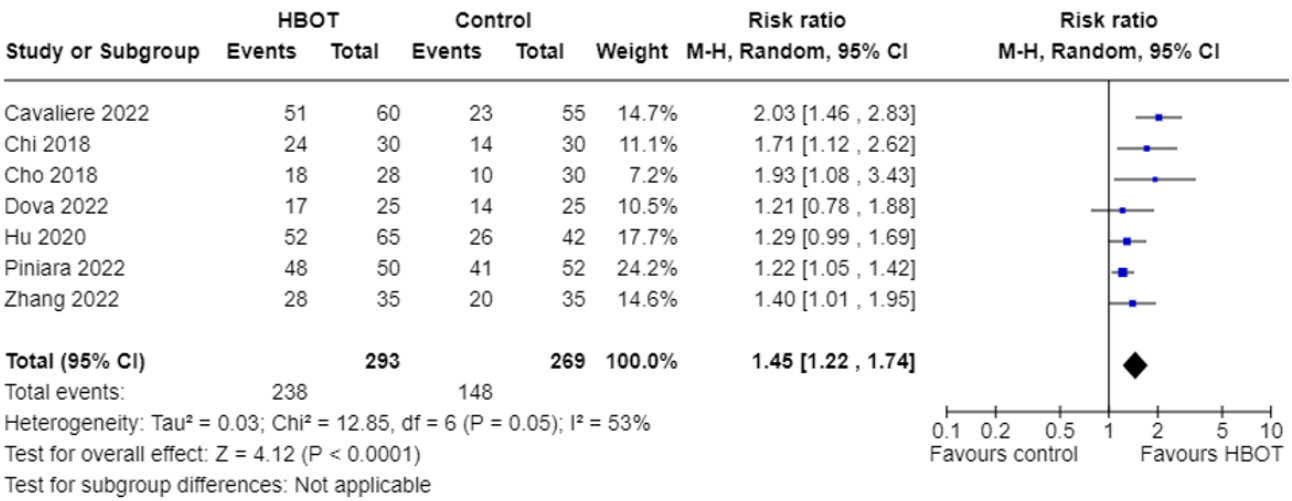


Figure 3
Forest plot for the proportion of participants with significantly improved pure tone average after treatment



been truly random for Cavallazi et al.,[12] while Hu et al. 2020 was pseudo-randomised by alternate allocation.[19] Only Hoffmann et al.,[18] described sham therapy with blinding of participants to the allocated therapy and only Piniara et al.,[25] clearly described blinding of the outcome assessors. One study enrolled 31 participants with both ISSHL and tinnitus, and 43 with one diagnosis or the other, making an intention-to-treat analysis for hearing loss problematic.[22] As no trials with potentially important losses to follow-up (less than 20 percent) reported any dichotomous outcomes, we have not performed sensitivity analysis making best and worst-case analyses. As there was relatively little variation in the risk of bias, we did not use study quality as a basis for sensitivity analysis.

EFFECTS OF THE INTERVENTIONS ON OUTCOMES

Together, these studies reported on seven different methods for assessing any improvement in PTA after treatment, hampering our ability to pool these results. Various groups have defined the different methods used including absolute change, percentage of change and percentage improvement. In general, there was reasonable evidence in favour of using HBOT for these patients with six of the eight synthesised analyses showing statistically important improvements. The results are summarised in Table 1 and the Forest plot in Figure 3 showing the proportion of participants with significantly improved pure tone average after treatment.

We were able to perform eight meta-analyses on these seven different means of measuring improved hearing across ten studies. The greatest number of studies contributing to any outcome was seven for the proportion of patients assessed as having a clinically important (defined as Siegel's criteria 'complete or partial recovery' or a similar assessment) improvement with HBOT versus the control intervention.[27] The chance of improvement following HBOT was greater than the control, RR 1.5 (1.2 to 1.7). This analysis suggests we would need to treat four patients with HBOT to improve one extra person's hearing by a clinically important amount. Six studies also reported the mean improvement from baseline PTA in decibels, but two did not give any estimate of the variance (e.g., standard deviation) and so could not contribute to the analysis. The remaining four trials together

suggested a greater mean improvement following HBOT the MD of improvement from baseline was 15.1 dB with a 95% CI of 8.2 dB–22.0 dB.

In summary of our analyses, with HBOT we found an improvement in the proportion of participants with > 25% return of hearing (RR 1.4, 95% CI 1.1–1.8). This analysis suggests we would need to treat five patients with HBOT to improve one extra person's hearing by 25% (NNT 5, 95% CI 3 to 21), an improvement in mean PTA expressed as a percentage of the baseline (MD 17.5%, 95% CI 2.5–32), and a better mean final PTA in decibels after treatment (MD 10.0 dB, 95% CI 2.7–17.3).

For the remaining two analyses, although the point estimate of effect was in favour of HBOT, any benefit was unclear as the 95% confidence intervals included no difference between the groups (the proportion of participants with either > 50% return of hearing (RR 1.5, 95% CI 0.9–2.8) or a mean improvement of > 20 dB (RR 2.2, 95% CI 0.5–9.2).

Only a single trial reported on any direct functional outcome.[14] These authors reported the word discrimination score (WDS) expressed as a percentage of words correctly identified on a standard test at three months after treatment and reported a better WDS following HBOT combined with both systemic and intratympanic (IT) steroids compared to the same steroid regimen alone (mean WDS at three months 66% [standard deviation14%] with HBOT versus 57% [19%] in the control group, $P < 0.05$).

ADVERSE EVENTS

None of these trials systematically reported adverse effects with HBOT or control therapies, although several did report a number of individuals who experienced some middle ear pain and effusion on compression with HBOT.[14–16,21] One study reported six participants who were withdrawn from HBOT with either aural barotrauma or confinement anxiety[21] and another reported two patients withdrawn for aural barotrauma and one with confinement anxiety.[25] For the other studies, the proportion of patients affected was 6% to 10% and all completed their planned treatment. Only a single study reported any adverse effects in the control group.[15] This study reported five of 25 patients (20%) complained of ear pain related to injection of IT steroids.

Discussion

This review has included data from 15 trials, and we believe these represent all randomised human trials in this area, both published and unpublished, at the time of searching the databases. We found reasonable evidence that HBOT improves hearing when applied as an early treatment in ISSHL (inclusion criteria for time from onset to treatment ranged from 48 hours to 30 days). These trials together reported on seven different approaches to assessing an improvement in PTA, of which five were statistically significant and the remaining two yielded a point estimate of effect in favour of those patients receiving HBOT. There was some indication from the analysis of pooled data from two trials that HBOT increases the proportion of patients gaining more than 25% improvement in hearing (RR 1.4).[12,17] The clinical significance of a 25% improvement in hearing from baseline is not clear and will depend greatly on the starting level of impairment.

An analysis of seven trials assessing a 'significant improvement' in hearing suggested those receiving HBOT had a 1.5 x better chance of improving compared to control. Six trials also suggested improvements in mean hearing measured in decibels following HBOT, with some evidence that more severely affected patients will improve most with the application of HBOT.[18,21–25] We found no evidence to support or refute the use of HBOT in those individuals with long-standing hearing loss.

Only a single trial reported any outcome designed to evaluate the functional impact of hearing loss on the individuals enrolled, and found a statistically significant benefit in word disrcrimination.[14] Other problems for this review were the poor methodological quality of many of these trials (see Figure 2), variability and poor reporting of entry criteria, the variable nature and timing of outcomes, and poor reporting of outcomes. Given the high rate of spontaneous recovery from ISSHL, there is a possibility of bias due to delay to entry in these small trials, as well as from non-blinded management decisions in all trials. The conclusions of this review are therefore to be interpreted with caution.

Previous trials were published over a 37-year period and are from a wide geographical area. We had planned to perform subgroup analyses with respect to the time between onset and therapy, the dose of oxygen received (pressure, time and number of treatments) and the nature of the comparative treatment modalities. None of these strategies were appropriate in the small number of pooled analyses. Response rates stratified by severity of hearing loss on presentation were reported by two studies.[12,23] Whilst one suggested a trend to greater treatment effect in those more severely affected,[23] this was not the case for the patients in the other,[12] and we cannot draw any firm conclusion.[23,24] The interpretation of outcomes by severity are complicated because for some methods of assessment, those with more severe hearing loss can improve to a much greater extent than those mildly affected (e.g., mean improvement in dB) while for other outcomes this is not the case (e.g., percentage return of hearing).

While we have made every effort to locate further unpublished data, it remains possible this review is subject to a positive publication bias, with generally favourable trials more likely to achieve reporting. Regarding long-term outcomes following HBOT and any effect on the quality of life for these patients, we have located no relevant data.

Conclusions

There is moderate quality evidence that HBOT improves hearing in patients with ISSHL who present up to 30 days after onset (however most patients included were of less than two weeks duration). Further good quality randomised trials are likely to improve our confidence in the effect estimate. There is no evidence available to establish or refute the functional importance of the improvements reported. The small number of studies available for pooled analyses, and the methodological and reporting inadequacies of the primary studies included in this review demand some caution and further studies are highly recommended, particularly addressing the impact of HBOT on functional and activities of daily living outcomes.

The evidence in favour of HBOT is stronger than for the established approaches with oral or IT steroids, and the routine use of HBOT in these patients can be justified. There is no compelling basis from this review for recommending HBOT solely as a rescue treatment following failure to respond to steroids.

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'*Power under pressure*' – defibrillation during hyperbaric oxygen therapy: a scoping review

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Keywords
Cardiac arrest; Defibrillation; Hyperbaric oxygen treatment; Medical conditions and problems; Resuscitation; Review article; Risk assessment

Abstract
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Introduction: Although defibrillation is the standard treatment for cardiac arrest with shockable rhythms, its safety during hyperbaric oxygen therapy (HBOT) remains uncertain, as the oxygen-enriched atmosphere and increased ambient pressure could, in case of sparking, increase fire and explosion risk. As established guidelines are lacking, this scoping review synthesizes the current knowledge, addressing the unique challenges that arise in this special environment.
Methods: A systematic literature search was conducted in CINAHL, Cochrane Library, EMBASE and PubMed. Two authors independently screened titles and abstracts, with a third author resolving discrepancies. Duplicate records were removed after initial screening. Full-text screening was also performed independently by two authors. Manual data extraction focused on actual defibrillation during HBOT, including outcomes, safety concerns, recommendations and further helpful information.
Results: The search initially identified 10,348 publications, ten of which were included. Screening of reference lists yielded another 23 publications, resulting in 33 finally included publications. Of these, four publications presented five patient cases of actual defibrillation during HBOT, while the remaining publications provided additional information on the topic.
Conclusions: Findings highlight a lack of standardised guidelines and limited empirical data, necessitating cautious consideration of defibrillation during HBOT. Safety protocols, including oxygen level and equipment specifications, vary between monoplace and multiplace hyperbaric chambers, influencing the feasibility of in-chamber defibrillation. There is strong consensus that defibrillation is strictly contraindicated inside monoplace chambers, while in multiplace chambers, risks and benefits must be assessed individually. While defibrillation during HBOT is rare, ensuring its safety remains of paramount importance. Future research should focus on refining safety protocols and establishing guidelines to optimise patient outcomes during HBOT-associated emergencies.

Introduction

Originally developed for diving medicine, hyperbaric oxygen therapy (HBOT) is now used for a wide range of conditions, though the strength of evidence supporting these varies considerably.[1] Monoplace and multiplace hyperbaric chambers present distinct treatment environments with certain technical and structural requirements, each having specific and stringent safety measures, ensuring safety and efficacy.[2,3]

Given the critical conditions treated, managing life-threatening emergencies is crucial. Defibrillation is the primary treatment for cardiac arrest with shockable rhythms. Although chest compressions maintain blood flow, they cannot restore a normal heart rhythm.[4] Early

defibrillation significantly improves survival rates, while any delay decreases likelihood of hospital discharge in out-of-hospital cardiac arrest by eight to ten percent per minute.[5] Ensuring timely access to defibrillation is one of the most critical interventions improving patient outcomes. So far, only two defibrillators are manufacturer approved for use during HBOT (Corpuls3 HBO and Haux Hyperbaric Defibrillator),[2,6,7] while a further two have been tested.[7,8] Defibrillators certified or tested for use under hyperbaric conditions are shown in Table 1.

Nevertheless, hyperbaric defibrillation carries risks, most notably, potential for chamber fires triggered by sparks, which are particularly hazardous in increased ambient pressure and potentially oxygen-enriched chamber air.[9] Evidence on the appropriate course of action in such situations is limited. The current European Resuscitation Council guideline "*Cardiac arrest in special circumstances*" provides no recommendations for resuscitation during HBOT, unlike initial guidelines for space or underwater resuscitation.[10,11]

To address this issue, Schmitz et al., conducted a comprehensive literature review in 2023 examining cardiopulmonary resuscitation within the context of HBOT.[12] Notably, two present authors (SN, TS) were part of the working group. Building on this foundation, the current scoping review was specifically directed at defibrillation – a vital component of cardiopulmonary resuscitation, that was not explicitly captured by the original search string. Consequently, it was refined to uncover unrecognised publications, enabling a more detailed analysis of defibrillation during HBOT.

This scoping review aims to compile the available evidence, providing a deeper investigation into the considerations and evaluating whether defibrillation under increased pressure could be performed safely, following a risk-benefit analysis. Thus, contributing to the development of future guidelines on safety and feasibility of hyperbaric defibrillation.

Given the added complexity of defibrillation in other hyperbaric environments e.g., during saturation diving,[13–16] these contexts are not included in this review.

Methods

This review is part of the doctoral thesis of author SN, for which ethical approval was granted by the Ethics Committee of Witten-Herdecke University (No. S-261/2022). This scoping review predominantly followed the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) extension for scoping reviews,[17] other than the deviations described in the text. It was conducted without a previously registered protocol and received no financial support or external funding.

Table 1
Defibrillators manufacturer approved or tested for use under hyperbaric conditions; HBOT – hyperbaric oxygen therapy

Company	Website	Device	Specific features
Corpuls	https://corpuls.world	Corpuls3 HBO	HBOT-certified by manufacturer[2,6,7]
Haux	https://hauxlifesupport.de	Hyperbaric defibrillator	HBOT-certified by manufacturer[2]
Stryker (formerly Physio Control)	https://www.stryker.com	Lifepak 1000	Not HBOT-certified by manufacturer, but has been tested successfully under hyperbaric conditions[7]
Zoll	https://www.zoll.com	AED Plus	Not HBOT-certified by manufacturer. Testing under hyperbaric heliox conditions revealed results that cast doubt on the unit's performance reliability after pressurisation[8]

SEARCH STRATEGY

Search string used in databases

A comprehensive literature review was conducted adapting the search string used by Schmitz et al.,[12] with regard to defibrillation. The literature search was performed in CINAHL, Cochrane Library, EMBASE and PubMed up until 1 October 2024. Details on the search string, used search modes and filters can be found in the *Appendix.

Additional sources

Following screening of publications identified through the database search, reference lists of those ultimately included in the review were screened.

INCLUSION CRITERIA

All publications explicitly describing defibrillation during HBOT, encompassing both patient cases or general information on defibrillation during HBOT, were included. Publications were included regardless of the type of article or year of publication.

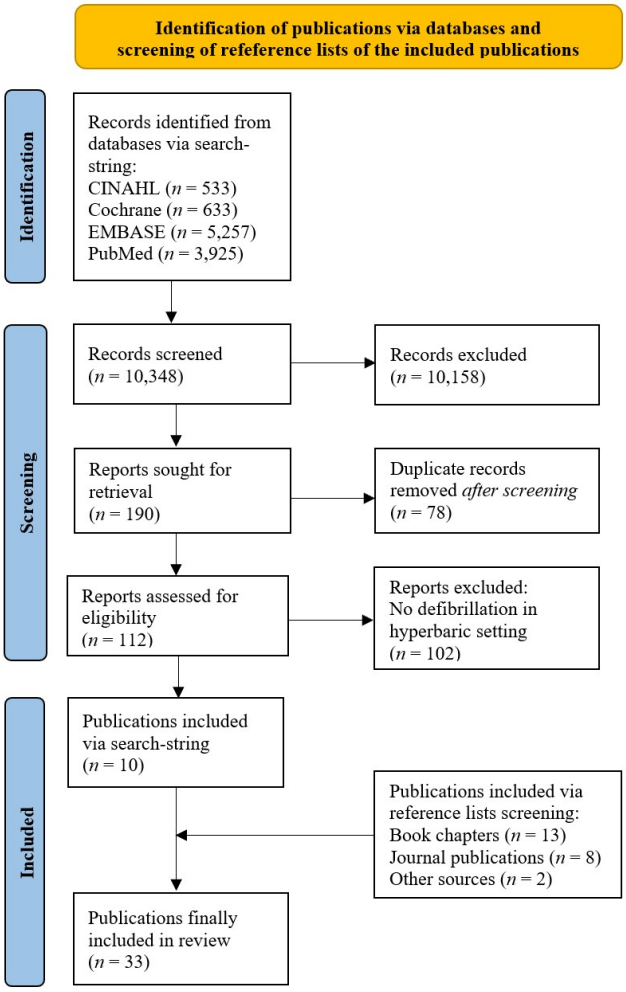
EXCLUSION CRITERIA

Publications only providing indirect hints that could relate to hyperbaric defibrillation without concretely addressing the topic were excluded. Furthermore, we excluded articles dealing with hyperbaric environments other than HBOT (such as saturation diving as mentioned above). Additionally, articles written in languages other than English or German and animal studies were excluded.

EVALUATION AND DATA INTERPRETATION

The screening process was conducted by two authors independently (SN, TS), commencing with title, followed by extended title and abstract screening. In instances of discordance, a third author (DG) was consulted as a referee. Duplicate records were removed after initial screening. All records deemed possibly relevant were sought for retrieval. Full-text screening was conducted independently by two authors (SN, TS), who also made the final decision on which publications to include in the review. All relevant data from the included publications were extracted manually, focusing on patient cases of actual defibrillation during HBOT and their reported outcomes, safety concerns and recommendations as well as other helpful information on the topic (SN, TS, DG, CB). This information was then interpreted descriptively in form of a narrative evidence synthesis (SN, TS). The most important contents were then summarized in a table to provide a clear and structured overview of the key information (SN, TS). Screening of the reference lists of the publications included in this review was carried out subsequently (SN, TS). In case of newly found articles deemed relevant, the screening process started from the beginning as described above.

Figure 1
Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) flowchart for the present review



Results

A total of 10,348 publications were identified through our primary search strategy via databases using the adapted search string. After the multi-stage screening process, 112 publications were fully reviewed, and 10 publications[2,6,7,9,12,18–22] were initially included in this review. Screening of reference lists of the included publications led to the addition of 23,[3,23–44] bringing the total number of publications finally included in this scoping review to 33.

This process is presented in a PRISMA flowchart[45] in Figure 1.

DEFIBRILLATION DURING HBOT

Of the 33 identified publications, four include case reports

***Footnote:** The Appendix is available to download from https://www.dhmjournal.com/index.php/journals?id=376

describing a total of five cases of actual defibrillation or cardioversion during HBOT,[19,20,24,40] as reported in the end of this section. The remaining 29 provide general information on the topic. These results are summarised in Table 2.

Of the additional 29 publications identified as thematically appropriate, only two dealt exclusively with hyperbaric defibrillation.[9,34] A further three were devoted to defibrillation as part of recommendations for cardiopulmonary resuscitation in the hyperbaric environment.[6,12,21] Further information can be found in 14 other publications, which mainly deal with HBOT of critical care, intensive care or emergency patients.[2,3,18,22,25–28,36,37,39,41,42,44] Six publications dealt with equipment and procedures that can be carried out in hyperbaric chambers.[7,23,31–33,35] The remaining four focused on safety guidelines,[43] general principles of HBOT and possible complications,[29,30] or its use in treating specific conditions.[38]

Within the aforementioned additional 29 publications, the review by Schmitz et al., identified 22 cases of cardiopulmonary resuscitation performed during HBOT,[12] including a defibrillation case that is consistent with one identified in our search.[12,24] Over a 20-year period involving more than 120,000 HBOT cases, Kot reported encountering a small number of fatal cases where defibrillation was indicated. Unfortunately, the source does not provide information on whether these were merely indicated or actually performed.[7]

TYPE OF DEFIBRILLATOR AND LOCATION DURING HBOT

Six publications provide information on the defibrillator itself.[2,3,9,22,27,34] Only a few are manufacturer approved or have been reported reliable for use under hyperbaric conditions.[2,6,7,22]

Various authors mention that a stand-alone battery-powered defibrillator can be used inside a multiplace chamber,[2,6,7,26] which should be stored outside and brought inside via a lock in an emergency.[6] Others recommend storing and operating the defibrillator from outside, and connecting to the patient via cables through the chamber wall.[2,3,7,9,12,20,22,28,30–35,44]

POTENTIAL RISKS ASSOCIATED WITH HYPERBARIC DEFIBRILLATION

The increased risk of fire, attributed to the oxygen-enriched atmosphere and high partial pressure, was identified as the greatest concern when performing defibrillation during HBOT.[3,6,7,9,12,18,20,21,23,25–29,31–34,36–42,44] In addition to a possible risk of explosion,[3,6,9,36,37] there are also general safety concerns.[2,22] Potential power transmission to bystanders during shock application is also described.[9,12,21] Defibrillator malfunction under increased pressure and possible operating errors due to nitrogen narcosis in chamber staff might also pose a risk.[9,12,34,37]

SAFETY PRECAUTIONS AND RECOMMENDATIONS FOR PERFORMING HYPERBARIC DEFIBRILLATION

Defibrillation inside monoplace chambers is contraindicated due to the high fire risk associated with the 100% oxygen environment.[7,27,28,30–32,34–37,39–44] Some authors state that defibrillation is possible in multiplace chambers if strict safety measures are fulfilled,[2,6,7,9,12,20,22,23,26–28,30–35,42,44] while others recommend that the patient should first be decompressed on an emergent basis and defibrillated outside in a normobaric setting.[2,3,7,9,18,21,25,29,37,38] The differences between considerations for defibrillation in monoplace and multiplace chambers are shown in Table 3.

PATIENT OUTCOMES

Some authors mention that hyperoxygenation during HBOT may have a protective effect, potentially extending the time a patient can tolerate cardiac arrest and thereby giving healthcare providers a certain time buffer to intervene.[2,7,9,25,28–32,39–41]

Of the three patient cases of actual defibrillation during HBOT in a multiplace chamber, identified in our review, two initially survived.[19,20,24] In both, the defibrillation and cardioversion followed emergency decompression of the monoplace chamber, and the procedure appeared to be successful.[40] No further information was given on the long-term outcome.[19,20,24,40]

CASE REPORTS

Wolf et al., described a 69-year-old woman treated in a monoplace chamber due to her non-healing foot ulcer. She suffered pulmonary barotrauma with air embolism to the brain and was transferred to a multiplace chamber, where she was recompressed to 284 kPa (60 feet, 18.3 metres sea water equivalent) and received 100% oxygen. During decompression she suffered from multiple episodes of ventricular tachycardia and hypotension and was therefore defibrillated and received antiarrhythmic drugs and pressor agents. She later died in the intensive care unit.[24]

Murphy et al., described a 17-year-old female with severe carbon monoxide intoxication. When HBOT began, the patient was comatose. Five minutes after starting HBOT, she suffered pulseless ventricular tachycardia. She was immediately cardioverted by the intensive care nurse present in the chamber, successfully converting into sinus tachycardia.[19]

The third case report described a successful defibrillation performed at 265 kPa in a multiplace chamber without any safety issues. Unfortunately, very little information on the medical history is provided.[20]

Table 2

Results of the literature search - each row represents one or more studies by an author or authorship group. ASAP – as soon as possible; atm abs – atmospheres absolute; CA – cardiac arrest; CO – carbon monoxide; CPR – cardiopulmonary resuscitation; DCS – decompression sickness; ETI – endotracheal intubation; ft – feet; HBOT – hyperbaric oxygen therapy; ICU – intensive care unit; i.v. – intravenous; msw – metres of sea water; MoPC – monoplace hyperbaric chamber; MuPC – multiplace hyperbaric chamber; O_2 – oxygen; PVT – pulseless ventricular tachycardia; VF – ventricular fibrillation; VT – ventricular tachycardia

Reference	Commentary / recommendations
6	Fire/explosion hazard; if applicable, precordial punch Battery-operated defibrillator outside chamber; CPR, then emergency lock-in; reduce energy for first shock to 150 J
28	MuPC: special precautions; possibly operate from outside with cables through chamber wall MoPC: never inside; rapid but controlled decompression (2–4 minutes); don't defibrillate directly after opening, oxygen spilling from inside for 10-30 seconds; remove patient, defibrillate as far as away as practical
23	Fire hazard higher in MoPC than in MuPC Not standard, but still possible if safety precautions are observed
29	Risk of fire; defibrillator placed outside; overall questionable safest approach: decompress while CPR, defibrillate outside; super-oxygenated, short time delay should be no risk
30	MuPC: possible with certain precautions; otherwise emergency decompression; note decompression of inside tender MoPC: remove patient (< 1 minute); '*grace period*' of several minutes; oxygen will diffuse to floor, tests showed no rise at level of patients chest outside chamber; dissipated within 30 to 40 seconds
7, 31, 32	Rarely needed;[7] dangerous, risk of fire caused by electrical discharges and voltaic arc between paddles[7,31,32] MoPC: absolutely contraindicated;[7,31,32] emergency decompression, but could cause complications if unresponsive[32] MuPC: can be performed safely with several precautions; compressed with air, oxygen below 21.5%; large surface adhesive plates, use gel, cables of wide diameter and low resistance, defibrillator outside; three persons needed: attendant inside, external defibrillator's operator, chamber operator[7,31,32] if impossible, defibrillate outside after emergency surfacing; preoxygenation might buffer for a few more minutes[7,31,32] two modern, stand-alone defibrillator approved for hyperbaric use, makes it easier than ever, but still risk of fire[7]
26	MuPC possible with battery-operated defibrillator MoPC: Use of any technical devices due to O_2 enrichment and fire hazard impossible
33	High risk of fire Defibrillator outside chamber with connection inside, use large adhesive plates, operated from outside doctor
34	MoPC: contraindicated; MuPC: hazardous, fire, implosion, operator error, minimised by leaving monitor outside Test of Life Pak 6S defibrillator with R-2 defibrillator adapter; self-adhering pads anteriorly and posteriorly, reducing risk of arching; average power loss of 9%, but within acceptable limits by manufacturer
3	Risk of explosion '*Triangle of fire*' Safe defibrillation: attach self-adhesive pads prior to HBOT, defibrillator outside Advantage of defibrillation during HBOT not proven
2	Battery-operated; alternatively, outside chamber with cable connection through wall Shockable rhythm unlikely; O_2-reserve, therefore decompression first
17	Possible in MuPC, device outside chamber; risk of fire; removal of flammable material; if no pads, consider gel Case report: successful defibrillation in MuPC (2.7 atm abs/ 265 kPa) without complications

Table 2 continued.

35	MuPC: Fire risk if sparking or combustible materials near paddles; use low-resistivity conductive gel or preapplied conductive disposable pads; defibrillator outside with connection inside MoPC: cannot be safely performed in chamber compressed with oxygen
19	· Patient with severe CO intoxication requiring resuscitation; comatose at start of HBOT · Suffered PVT 5 minutes after onset of HBOT, immediately cardioverted in chamber
18, 36, 37	· MoPC: impossible due to prevailing 100% oxygen atmosphere;[36,37] emergency decompression without DCS risk; totally decompress, remove patient completely, oxygen outflow increases fire and explosion risk[37] · MuPC: CPR increases nitrogen uptake for inside staff, DCS risk; decompress slowly with attendants breathing O_2 at 1.9 atm abs until normobaric;[18,36,37] defibrillation possible, if chamber oxygen not elevated; yet critical due to fire and explosion risk[36,37] as well as equipment malfunction;[37] oxygen contamination seems to be inevitable during CPR[37] · MuPC less fire hazard than MoPC; use specially equipped resuscitation bag to prevent oxygen contamination[37] · If necessary: electrodes with largest contact surface, use gel and adhesive pads; defibrillator outside chamber; ensure fire extinguishing equipment available inside;[36,37] strongly recommended to avoid defibrillation inside MuPC; safest method: decompress while CPR, defibrillate in unpressurised chamber with door wide open[18,37]
22	· Defibrillation in chamber dangerous; only few devices approved for HBOT · Safest shock delivery: controlled outside the chamber, cable through the chamber wall, adhesive pads; reduce risk of flammability: chamber air like ambient air
9	· Risk of arrhythmias during HBOT low; HBOT appears to prolong tolerance for CA · Risk of fire, implosion, operating errors, malfunction; therefore, defibrillator outside · Reduce risk of shock transmission: grounding, insulating shoes, adhesive electrodes, biphasic defibrillation · Consider medical therapy for VF if monitoring, central i.v. access, ETI; depending on indication, emergency decompression could cause CA to worsen; overall, emergency decompression and normobaric defibrillation
44	· Fire hazard due to electrical sparking in chamber · MuPC: Area around paddle has to be free from flammables; use gel; defibrillator stored and operated from outside · MoPC: do not defibrillate in chamber filled with pure oxygen
12	· Risk of fire biggest concern + poses further risks (e.g., risk of current transmission) · Store defibrillator outside; use biphasic device with chest panels connected to patient with cables through chamber · If defibrillation necessary oxygen concentration shouldn't exceed 21.5% · Avoid current transmission: earthing of chamber, grounding footwear, sufficient distance from patient
38	· Risk of fire due to elevated oxygen level of chamber air; staff performing CPR increased nitrogen uptake · Avoid defibrillation inside; slowly decompress with staff breathing oxygen from 9 msw until normobaric · Only use specialised resuscitation bags to avoid oxygen contamination of the chamber to reduce fire risk
43	· MoPC: in chambers compressed with 100% oxygen, defibrillation at least 6–8 ft away from open door of recently decompressed chamber

Table 2 continued.

27, 39–42	. MoPC: patient supersaturated; O_2 pours from chamber into room, move stretcher away to keep distance; remove clothing due to increased fire risk; brain and heart presumably high O_2, taking a few seconds acceptable[39-41] . Measured oxygen levels after emergency decompression; oxygen falls to floor, dissipates within 30 seconds, does not remain elevated at patient level, doesn't rise in room;[39,40] . MoPC: under no circumstances in pure oxygen atmosphere of chamber; defibrillate outside[39] . Case reports: successfully defibrillated one patient and cardioverted another who were rapidly removed from MoPC for dysrhythmias; a few seconds elapsed following extraction before defibrillation; both treated while on the gurney attached to the MoPC; no evidence of spark or fire;[40] only two cases in more than 10 years[41] . MuPC: Defibrillation and cardioversion can be performed, if no excess oxygen build-up[27,42] . MoPC: decompress first, get patient out of chamber; during decompression, switch to air; if not possible, wait at least 40 seconds; remove clothing as it will be oxygen enriched and thus increase the risk of fire[27,42]
25	. Decompression ASAP: defibrillation when normobaric as there is a risk of fire . Hyperoxia may prevent tissue hypoxia during CA and may improve survival
24	. Case report: 69-year-old woman, non-healing foot ulcer, about 20 HBOTs in MoPC; suffered pulmonary barotrauma with air embolism; transferred in MuPC; recompression to 60 ft with 100% O_2; during decompression multiple episodes VT + hypotension; defibrillation, antiarrhythmics, pressor agents in chamber; died in ICU
21	. Defibrillation may cause dangerous sparks, risk of fire due to arcing . Decompression and defibrillation in normobaric atmosphere

The fourth report described the successful defibrillation of one patient and cardioversion of another, both of whom were rapidly removed from the monoplace chamber due to dysrhythmias. Defibrillation was conducted after a few seconds elapsed following extraction, while the patients were still on the gurney connected to the chamber. Notably, no evidence of sparks or fire were observed during either intervention.[40]

Discussion

To the best of our knowledge, this is the first review since 1999[9] to specifically address defibrillation in the hyperbaric setting – a potentially life-saving but high-risk procedure. With the increasing number of critically ill patients undergoing HBOT, a re-examination of defibrillation practices is both timely and necessary.

DEFIBRILLATION DURING HBOT

Defibrillation during HBOT itself is an exceptionally rare event with very few documented cases.[7,12,19,20,24,40] This is also reflected in our results, where we were only able to identify five cases of defibrillation or cardioversion.[19,20,24,40] Unfortunately, the case reports provide only limited insight, making it difficult to draw reliable conclusions from them. It must be noted that the case reports by Wolf et al.,[24] and Murphy et al.,[19] contain inaccuracies in terminology used for electrical therapy. Although the terms used in both cases are incorrect,[19,24] it is important to note that both defibrillation and cardioversion rely on electrical energy, albeit with differences in shock delivery and intensity.[46]

The life-threatening conditions of these patients underscore the urgent need for comprehensive guidelines on defibrillation during HBOT to optimise patient outcomes.

TYPE OF DEFIBRILLATOR AND LOCATION DURING HBOT

There are only a few manufacturer approved or tested defibrillators for hyperbaric use,[3,7–9,22] and reliability under pressure may vary.[9,22] Due to the niche market and financial constraints, many manufacturers do not pursue hyperbaric approval, shifting responsibility for use of uncertified devices to the physician.[3,44] Others may also be suitable, but remain unverified due to absence of testing. It is important to distinguish between the defibrillator's ability to safely withstand pressurisation and depressurisation, and its use when connected via a penetrator outside the hyperbaric chamber. Depending on the defibrillator itself, there is risk of possible malfunction due to the increased ambient pressure.[9,12,37] However, there are data showing that the defibrillator as a device *per se* is safe up to 304 kPa chamber pressure.[3] Some portable, battery-operated and CE-certified defibrillators have been available for several years.[2,6,26] Alternatively, a defibrillator may also be stored and operated

Table 3
Differences between monoplace and multiplace hyperbaric chambers regarding defibrillation; CA – cardiac arrest; HBOT – hyperbaric oxygen therapy; O_2 – oxygen

Parameter	Monoplace chamber	Multiplace chamber
Capacity (patients)	One	4–12
Patient accessible (e.g., through lock)	No	Yes
O_2 fraction inside chamber	100%	Should not exceed 21.5%
Defibrillator instantly accessible	No	Yes
Defibrillation possible inside chamber and procedure for defibrillatable CA	No, emergency decompression and normobaric defibrillation	Yes, feasible but only with strict safety precautions

from outside, connected to the patient via cables through the chamber wall, under the assumption that this arrangement reduces fire risk by minimising device-induced sparking and thereby lowering overall risk.[2,3,7,9,12,20,22,28,30–35,44] Wright et al.,[21] state that defibrillators are equipped with large capacitors and small brushed motors, known to discharge and spark. While older or specialised models may have used motor-driven components, the main concern remains capacitor discharge and potential arcing between paddles. However, technological advances, including the shift from paddles to adhesive pads, have significantly reduced risk of spark-induced fires. Jacobs et al. noted that no spark-related fires have been reported since these improvements.[47]

Direct patient access is impossible inside a monoplace chamber, making urgent decompression mandatory in emergencies.[27] In the prevailing 100% oxygen environment, defibrillation is contraindicated due to fire hazards. The victim must first be brought outside to a normobaric atmosphere.[7,27,28,30–32,34–37,39–44] In contrast, multiplace chambers might allow defibrillation under rigorous safety measures.[2,6,7,9,12,20,22,23,26–28,30–35,42,44] Direct hands-on treatment by specialised in-chamber staff is possible.[26,27] Recent data conclude that defibrillation could be safe if stringent safety precautions are observed.[12]

Millar explicitly states that most centers do not locate a defibrillator in the multiplace chamber itself, since cardiac arrest caused by a shockable rhythm is unlikely during HBOT and the patient usually has sufficient oxygen reserves to first perform complete decompression.[2] This contrasting approach of avoiding a possibly hazardous defibrillation by decompressing and subsequent normobaric defibrillation is also advised by other authors.[2,3,7,9,18,21,25,29,37,38] So far, our data does not support any standardisation on whether defibrillation is performed inside or outside the multiplace chamber. However, when the decision is made to perform in-chamber defibrillation, it is essential to prioritise devices that are either manufacturer approved or specifically tested for hyperbaric use, as this ensures adherence to fundamental safety standards.

POTENTIAL RISKS ASSOCIATED WITH HYPERBARIC DEFIBRILLATION

In addition to general safety concerns[2,22] and the potential explosion risk,[3,6,9,36,37] the risk of fire is regarded as the primary safety concern associated with defibrillation during HBOT.[3,6,7,9,12,18,20,21,23,25–29,31–34,36–42,44] Risks increase with each use of electrical, high voltage equipment, which may contribute to the '*triangle of fire*'. This includes increased oxygen concentrations, the presence of flammable substances and possible ignition sources.[3] Risks derive from sparking and voltaic arcing possibly occurring between the paddles, and oxygen that may originate from the respiratory system.[12,21,34,35,44] In monoplace chambers the overall fire risk is higher than in multiplace.[23,37]

Many authors derive their conclusions about the fire risk of hyperbaric defibrillation from fire incidents reported in earlier literature such as the publication by Simini[48] and the review from Sheffield and Desaultels.[49] Simini described the devastating consequences of the multiplace chamber fire in Milan in 1997, which resulted in eleven deaths.[48] Sheffield and Desautels analysed 25 chamber fires from 1923 to 1996 resulting in 60 deaths. The analysis showed, that oxygen concentration inside the chamber appears to have a major influence on probability of survival.[49] Although none of these incidents was caused by defibrillation, they demonstrate the catastrophic consequences a fire or explosion in a pressure chamber may have.[48,49]

Operating errors resulting from nitrogen narcosis in chamber personnel may represent a potential safety risk.[9,12,34] Defibrillator malfunction under increased ambient pressure, together with limited availability of approved devices, seem to present a safety risk to the patient and all individuals present.[9,12,22,37] Bystanders might be at risk of current transmission due to shock delivery.[9,12,21] If the decision is made to perform defibrillation inside the pressure chamber, it is of utmost importance to minimise the associated risks in order to perform defibrillation as safely as possible.

It is crucial to highlight that the majority of concerns regarding fire risk associated with defibrillation during HBOT are not based on direct empirical evidence, but rather inferred from real-life fire incidents that occurred under hyperbaric conditions without being triggered by defibrillation itself. To the best of our knowledge, there have been no published reports of any adverse events, regardless of severity, directly resulting from defibrillation under these conditions. Furthermore, much of the literature is based on older publications, the associated risks are often reiterated anecdotally, which may lead to an overstatement of its practical significance. Since then, advancements in technology, such as introduction of biphasic defibrillators, enhanced monitoring of oxygen concentrations, and use of adhesive pads may have contributed to risk reduction. These improvements call into question the accuracy of current risk assessments and suggest that comprehensive, updated studies are warranted to better evaluate risk management.

SAFETY PRECAUTIONS AND RECOMMENDATIONS FOR PERFORMING HYPERBARIC DEFIBRILLATION

Monitoring heart rhythm and detecting the necessity of defibrillation during HBOT is feasible in both chamber types.[3,23,40] As described above, they differ fundamentally in the way HBOT is applied.[23] Thus, recommendations must be considered separately.

Defibrillation inside monoplace chambers is strictly contraindicated. The patient must be evacuated after decompression first.[7,27,28,30–32,34–37,39–44] Emergency decompression can be performed within approximately one minute,[27,30,32,41,43] allowing the patient to be removed within 90 seconds.[43] Holcomb et al., recommend a rapid yet controlled decompression within two to four minutes, as the patient is likely to be sufficiently oxygenated, thus preventing potential complications.[28] It is worth considering whether the risk of barotrauma truly justifies delaying emergency decompression. Given the time-critical nature of cardiac arrest, the fastest possible decompression appears to be the more appropriate course of action. Safety precautions also include reducing 100% oxygen to normal air during decompression, if impossible, then at least 40 seconds should elapse before defibrillating so that the oxygen can dissipate.[27,42] Tests show that oxygen pouring out of the recently decompressed chamber dissipates within 30 to 40 seconds.[30,39,40] Removing the patient completely and maintain a distance[28,39–41] of six to eight feet from the recently decompressed chamber is a potential safety strategy.[37] The patient's oxygen saturated clothing should be removed beforehand.[27,39–42] All these measures aim to reduce the risk of fire. After evacuation, shocks may be delivered with recommended energy, as advised by the European Resuscitation Council.[46]

In multiplace chambers, some state that decompression must be performed beforehand.[2,3,7,9,18,21,25,29,37,38] Emergency decompression may take several minutes, as it must also align with decompression obligations of the personnel.[6,7,18,21,22,27,30,36,37] This is crucial to avoid putting them at risk, for example, from possible decompression sickness.[3,18,25,26,36–38] Muth recommends slow decompression while rescuers breathe 100% oxygen from 1.9 atmospheres absolute (atm abs) until normobaric.[18,36,37] This is supported by others,[7] as, depending on the indication (decompression sickness, arterial gas embolism), emergency decompression could potentially aggravate the cause of cardiac arrest.[9] In some clinical scenarios, such as extended recompression protocols, rapid emergency decompression is contraindicated due to the tender having a decompression obligation,[7,21] for example while using the extended US Navy Treatment Table 6. In these cases, in-chamber defibrillation might be indicated, with the potential delay in decompression carefully balanced against the hazards of performing hyperbaric defibrillation. This reinforces the necessity of defibrillation during HBOT in certain situations. Another option is evacuation of the patient through the patient lock, providing care outside, thus ensuring that staff and other patients continue their regular decompression scheme.[21]

Twenty-five years ago, Pitkin attempted to clarify this issue within his review on defibrillation in hyperbaric chambers.[9] He concluded that defibrillation can be performed during HBOT, but some safety issues remain. The potential benefits must be carefully weighed against the risks. Drug therapy for arrhythmias could be considered in special circumstances. Pitkin recommended emergency decompression and normobaric defibrillation if cardiac arrest occurred.[9]

Dieterich et al.,[6] developed an algorithm for shockable cardiac arrest during HBOT by implementing resuscitation training at their center considering that defibrillation is associated with a certain risk of fire and explosion. A battery-operated, portable defibrillator approved for hyperbaric use is stored outside. Using an emergency lock, the first shock may be delivered after just one to two minutes. The authors recommended delivery of the first shock with a reduced energy of 150 joules and also consider delivering a precordial thump if cardiac arrest has been observed and it does not delay defibrillation.[6]

In order to defibrillate despite the risks in multiplace chambers, strict safety precautions have to be obeyed.[2,6,7,9,12,20,22,23,26–28,30–35,42,44] These include maintaining chamber oxygen concentration < 21.5%, reducing risk of flammability and defibrillator storage and operation outside the chamber. Due to lack of data, no conclusive statement can yet be made about the use of battery-operated portable defibrillators. Use large adhesive pads and remove all flammable materials in the vicinity.[7,9,12,20,22,28,31–35,44] If no pads are used, gel should be applied to reduce impedance.[20,31,32,35,44] Maintain sufficient distance from the victim in order to reduce risk of shock transmission.[12] It should be noted that hyperbaric chambers are not ideal for defibrillation

because of their confined design and the possibility of shocking others. Appropriate measures include adequate grounding, use of insulating footwear, adhesive pads and biphasic current requiring less energy.[9,12] The majority of aforementioned measures were also recommended in Schmitz et al.'s review on cardiopulmonary resuscitation during HBOT.[12]

PATIENT OUTCOMES

It is hypothesised that hyperoxia during HBOT could prevent tissue hypoxia and thus have a positive effect on survival due to prolonging tolerance of cardiac arrest.[2,7,9,25,28–32,39–41] So far, there is a lack of sufficient data for this physiologically plausible construct.[21,25] These considerations may also not apply during oxygen breaks.[9,50] Moreover, if cardiac arrest were to occur during HBOT, it could nevertheless indicate presence of myocardial hypoxia despite general hyperoxia. In this situation, standard resuscitation protocols should be promptly applied, with immediate defibrillation considered if indicated and deemed safe.

In our scoping review, we identified a reported survival rate of 80% in cases where defibrillation or cardioversion was performed during HBOT, with no adverse effects mentioned – an outcome that appears promising. However, it is important to note that this is based on five patient cases, making any meaningful statistical analysis impossible. Additionally, there was no information provided on long-term outcomes, which limits the ability to draw definitive conclusions about the overall effectiveness and safety of defibrillation in this setting.[19,20,24,40] The risk of delayed defibrillation due to prior decompression must always be weighed against the risk of performing defibrillation during HBOT, as both can significantly impact patient outcomes. Up to now, the superiority of immediate defibrillation during HBOT compared to chest compression with rapid decompression and subsequent normobaric defibrillation has not yet been proven.[3]

LIMITATIONS

The limited evidence available may pose challenges to the robustness of this scoping review. This could be further compounded by restricting the selection of articles to those in English and German. It is important to acknowledge potential for publication bias regarding reported cases. This may result in an underrepresentation of actual patient cases in the literature. Addressing this issue in future research is essential to identify and account for the number of unreported cases, thereby providing a more accurate understanding of the clinical landscape. In a world where resuscitation algorithms have been developed to manage emergencies in extreme environments (such as space), the establishment of dedicated HBOT guidelines is equally justified and essential. Such guidelines would ensure optimised patient care, even in these highly specialised and challenging settings.

Conclusions

Defibrillation during HBOT remains an exceptionally rare, but potentially life-saving procedure, albeit one that might carry risk for chamber personnel and fellow patients. Expert opinions diverge, underscoring that any decision must be made on a case-by-case basis, with appreciation of local chamber characteristics and available safety measures. Regarding monoplace chambers there is strong consensus that defibrillation is strictly contraindicated. In multiplace chambers the benefit must always be weighed against the risk and the decision should ideally be made with consent of all those involved. Under stringent safety protocols and careful risk assessment, defibrillation may be performed safely. At present, given the uncertainty regarding whether the risk of severe complications is overestimated, it may be advisable to adopt a careful approach and consider in-chamber defibrillation only as a last-resort, e.g., when emergency decompression is unfeasible. Until the evidence base matures to support a more definitive recommendation, caution is advised. Future studies should incorporate additional endpoints, such as complications, patient mortality, one-month survival and neurological outcome. Further research and standardised guidelines are essential to show whether immediate defibrillation provides a benefit in survival and overall outcome compared to emergency decompression and normobaric defibrillation and to enhance safety and efficacy in these critical situations.

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Short communication

Critical flicker fusion frequency measurement through a chamber porthole

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Diving research; Narcosis; Hyperbaric research

Abstract

(Schipke JD, Muth T, Brebeck AK, Dreyer S. Critical flicker fusion frequency measurement through a chamber porthole. Diving and Hyperbaric Medicine. 2025 December 20;55(4):419–422. doi: 10.28920/dhm55.4.419-422. PMID: 41364866.) The critical flicker fusion frequency (cFFF) is a non-invasive measure of central nervous system function and cortical arousal, increasingly used in diving and hyperbaric medicine to assess the effects of breathing gases under pressure. This feasibility study aimed to evaluate whether cFFF can be reliably measured through the porthole of a hyperbaric chamber. Forty-five experienced male divers underwent cFFF testing at various pressures (101.3 kPa outside chamber, then 101.3, 608, 132, 101.3 kPa inside [1.0 bar outside then 1.0, 6.0, 1.3, 1.0 bar inside]) using a manually operated LED flicker-device while standing at a fixed distance from the chamber window. Results showed that cFFF values were higher inside the chamber at 101.3 kPa (1.0 bar) compared to outside (45.6 Hz vs. 40.2 Hz), decreased under hyperbaric conditions (608 kPa [6 bar], 43.5 Hz), and declined further during decompression (132 kPa [1.3 bar], 42.1 Hz; 101.3 kPa [1.0 bar], 43.5 Hz). These findings support previous observations of gas-induced central nervous system effects and highlight the sensitivity of cFFF to pressure-related neural changes. The successful external measurement protocol addresses challenges associated with observer narcosis and movement artifacts in underwater settings. While limited by the homogenous participant group and lack of confirmatory measures, this approach may still be a valuable tool for future research into the temporal dynamics of gas narcosis and cortical excitation.

Introduction

At or above the critical flicker fusion frequency (cFFF), a flickering light stimulus is perceived as steady by the observer. As such, cFFF serves as a measure of the brain's temporal resolution and has proven valuable for assessing cortical arousal and central nervous system (CNS) function – a property recognised over 30 years ago.[1] More recently, particularly in diving contexts, this non-invasive and easy-to-use technique has been employed to evaluate alertness and cognitive performance in divers.[2] CFFF has also been used more broadly to assess CNS function and arousal.[3,4]

In diving and hyperbaric medicine, cFFF has helped investigate the neurological effects of breathing gases under pressure, though findings remain inconsistent. For example, cFFF decreased under 142 kPa (1.4 bar) oxygen but recovered at 284 kPa (2.8 bar), indicating dose-dependent neural effects,[5] whereas 100% O_2 at 101.3 kPa and 284 kPa (1 and 2.8 bar) also reduced cFFF – but not at 608 kPa (6 bar).[6] Nitrox breathing produced mixed results: transient cFFF increases followed by decline,[7] enhanced post-dive alertness,[8] or a protective effect versus air.[9] Scuba dives to 33 m showed initial cFFF elevation, followed by lasting decreases post-dive.[10] Rebreather dives with air, trimix, or heliox revealed mild increases without long-term effects,[2,11] whereas trimix bounce dives to 100 m showed no change.[12]

One possible explanation for the contradictory results is likely described by Freiberger and colleagues.[13] These authors indicate that CO_2 plays a critical role under hyperbaric conditions, interacting with N_2 and exercise to cause narcosis, which may be worsened by O_2, while at the same time, sea level O_2 partially rescued motor and memory reaction time impaired by CO_2.

The heterogeneous outcomes highlight that the effects of nitrogen, oxygen, CO_2 and inert gases – individually or in combination – on CNS function under pressure remain only partially understood.

Given the need for further investigation, this study aimed to evaluate whether cFFF measurements taken externally through the porthole of a hyperbaric chamber are feasible and yield results consistent with previous findings.

Methods

The study protocol was reviewed and approved by the Ethics Committee of the Medical Faculty, Heinrich Heine University Düsseldorf, in accordance with the Declaration of Helsinki,[14] Approval No. 2023-2493.

Forty-five experienced male divers from five professional fire departments and a diving club participated in the study (mean age 34.6 [standard deviation (SD) 6.6] years; height 182 [SD 7] cm; weight 84.6 SD [6.6] kg; body mass index 26.4 [SD 2.1] $kg \cdot m^{-2}$). All subjects completed a training dive at 608 kPa (6 bar) in a hyperbaric chamber at the hyperbaric oxygen therapy facility of the University Hospital Düsseldorf (Germany).

A manually operated flicker light (Scaleo, Esslingen, Germany; single LED, 8000 K) was used for cFFF testing. A consistent distance of 1.5 m between the device and the participant was maintained by the same experienced examiner throughout all measurements.

STATISTICAL ANALYSIS

A repeated measures ANOVA was conducted (ChatGPT), followed by a Bonferroni post-hoc test. The measurements differed significantly from each other at $P < 0.05$.

Approximately a decade ago, the American Statistical Association recommended caution in the use and interpretation of *P*-values and statistical significance.[15] Accordingly, for our simple five-step protocol, we evaluated differences in cFFF values using Cohen's d.[16]

PROTOCOL

Critical flicker fusion frequency was first measured outside a 12-person hyperbaric chamber (Haux, Life Support, Karlsbad, Germany). Subsequently, and following the same participant order, cFFF was measured inside the chamber through the door's porthole at ambient pressures of 101.3, 608, 132, and again at 101.3 kPa (1.0, 6.0, 1.3, 1.0 bar). For each measurement, the flicker frequency was continuously increased from a low to a high value until the participant gave a predefined 'OK' hand signal, indicating their individual cFFF threshold had been reached.

Since the examiner remained the same throughout and the participants were thoroughly informed in advance about the procedure for determining the fusion frequency, we limited the assessment to a single measurement, although performing three measurements would likely have yielded even more precise cFFF values.

Participants were instructed to stand still near the porthole and maintain steady gaze fixation without moving their eyes during the measurement. A fixed distance of 1.5 m between the observer and the participant was maintained at all five time points.

Results

Normobaric (101.3 kPa) cFFF measured inside the chamber was significantly higher than the values obtained outside the chamber (mean 45.6 [SD 4.7] Hz vs. 40.2 [SD 4.3] Hz), with a large optically related effect size indicated by Cohen's $|d| = 1.2$.

Following compression to 608 kPa (6 bar), cFFF decreased compared to the initial 101.3 kPa (1 bar) measurement inside the chamber (mean 43.5 [SD 4.6] Hz vs. 45.6 [SD 4.7] Hz; $P < 0.05$; $|d| = 0.45$, medium effect). A decrease in cFFF was observed from 608 kPa (6 bar) to the 132 kPa (1.3 bar) reading during decompression (43.5 [SD 4.6] Hz vs. 42.1 [SD 4.2] Hz; $P < 0.05$; $|d| = 0.32$, small effect). The normobaric (101.3 kPa) cFFF at the end of the session (following decompression) was lower compared to the beginning (45.6 [SD 4.7] Hz vs. 42.6 [SD 4.6] Hz; $P < 0.05$; $|d| = 0.65$, medium effect). The relative decreases beginning from the normobaric cFFF-value were equal to 4.6%, 7,7%, and 6.6%, respectively.

Discussion

This feasibility study aimed to determine whether the cFFF of individuals exposed to varying pressures inside a hyperbaric chamber can be reliably assessed from outside the chamber through the porthole in the chamber door. The results indicate that this experimental setup is both feasible and capable of yielding plausible data.

The observed decrease in cFFF under hyperbaric conditions (608 kPa [6 bar]) aligns with previous reports suggesting that elevated partial pressures of nitrogen can induce narcosis[9,17] and can transiently impair cortical processing.[5,6]

The modest yet consistent decline in cFFF during decompression in this study may indicate a lingering effect of gas narcosis, fatigue, or altered neural excitability. The final normobaric value being lower than the pre-dive baseline suggests that even short exposures to hyperbaric conditions can produce temporary alterations in central nervous system function detectable by cFFF.[10,18]

Importantly, the effect sizes observed in this study ranging from small to large, highlight the sensitivity of cFFF to changes in ambient pressure and support its use as a non-invasive, easy-to-use, real-time marker of cortical arousal during and after hyperbaric exposures.

When conducting studies at elevated pressures, observers themselves are also at risk of experiencing the effects of nitrogen narcosis. This poses a significant challenge for cFFF-based assessments of CNS function under different breathing gas mixtures, as reliable measurements are difficult to obtain, if the observer is impaired by nitrogen narcosis. Therefore, it is desirable for the observer to be able to conduct these measurements from outside the chamber.

The cFFF is primarily determined by the spatial distribution of photoreceptors (cones and rods) on the retina. When a light stimulus flickers, the flicker is perceived more intensely if it is projected directly onto the fovea than when viewed peripherally. Consequently, the highest cFFF values are observed when the stimulus strikes the fovea.[19] Therefore, it was essential to maintain a consistent distance between the light source and the participant's eyes as well as a constant visual angle throughout the protocol.

This consideration is particularly important, as deviations in the relative positioning of the light source and the eyes can influence the outcome. For example, in underwater diving research, measurements are often performed both at the surface and at depth, where maintaining fixed positions is inherently more difficult.

Additionally, any movement of the observer or the light source can affect cFFF values. A particularly notable source of error is eye movement, especially saccades, which can significantly distort results. During saccadic eye movements, humans have been shown to perceive flicker frequencies as high as 2,000 Hz.[20]

LIMITATIONS

This study's focus on experienced male divers may limit generalisability, but this cohort was deliberately chosen to reduce variability from confounding factors such as experience, age, and sex.

Another limitation is the interpretation that reduced cFFF reflects inert gas narcosis. While this is plausible, confirmatory measures like EEG or cognitive testing were beyond the scope of this feasibility study, which aimed primarily to assess protocol viability.

Lastly, cFFF values measured in the hyperbaric chamber were 13% higher than outside, potentially affecting comparability with other studies. However, normalising baseline values to 100% a method supported by previous research, can mitigate this issue.[6,10]

Conclusions

This feasibility study demonstrated that cFFF measurements can be successfully conducted from outside a hyperbaric chamber and can yield valid and interpretable results. The observed cFFF reduction is consistent with the effects of nitrogen narcosis under elevated ambient pressure, which appear to persist beyond the period of exposure. At the same time, the increase in cFFF due to refraction through the chamber window highlights a methodological issue in field studies where flickering light passes through different media such as water, glass, and air, potentially altering measurement outcomes.

The novel approach to cFFF measurement conducted externally through the chamber porthole may support future investigations into the effects of breathing gases under elevated pressure by eliminating the risk of observer impairment. For example, it is conceivable that a transient euphoric phase, possibly related to elevated oxygen partial pressure precedes the onset of nitrogen narcosis, making the temporal dynamics of excitation and narcosis a particularly interesting subject for further study.

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Case reports

Hyperbaric oxygen therapy as salvage treatment for post-traumatic sudden sensorineural hearing loss: a case report

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Keywords
Deafness; Hyperbaric medicine; Temporal bone fracture; Trauma

Abstract
(Baptista LA, Guincho J, Donato M, Araújo P, Amaro CE, Escada PA. Hyperbaric oxygen therapy as salvage treatment for post-traumatic sudden sensorineural hearing loss: a case report. Diving and Hyperbaric Medicine. 2025 December 20;55(4):423–426. doi: 10.28920/dhm55.4.423-426. PMID: 41364867.)
Introduction: We report the first case of hyperbaric oxygen therapy used to treat sensorineural hearing loss in a child after head trauma.
Case report: A 13-year-old boy with no relevant past medical history presented to the emergency department with tinnitus and hypoacusia following head trauma. An ear computed tomography scan showed a right longitudinal temporal fracture sparing the otic capsule, and the audiogram identified a moderate sensorineural hearing loss in the right ear involving frequencies between 2,000 and 8,000 Hz. He was treated with corticosteroids and betahistine for an acute audiovestibular loss with resolution of the vestibular symptoms. At three months post-trauma the sensorineural hearing loss persisted. The patient started treatment with hyperbaric oxygen therapy with complete resolution of the hearing loss after 11 sessions.
Conclusions: This case identifies potential benefit from salvage hyperbaric oxygen therapy in the treatment of sudden sensorineural hearing loss of traumatic etiology.

Introduction

Post-traumatic sudden sensorineural hearing loss (SSNHL) accounts for approximately 4.2% of all SSNHL cases, with causes including head trauma, barotrauma, and acoustic trauma.[1] In cases of head trauma, SSNHL is mostly attributed to temporal bone fractures, which cause indirect damage to the cochlea or cochlear nerve. However, other mechanisms, such as labyrinthine concussion or direct damage to the central auditory pathways, have also been identified.[2] Despite advances in understanding the pathophysiology of post-traumatic SSNHL, specific treatment guidelines remain unclear, and management is often individualised.

Hyperbaric oxygen therapy (HBOT) is a well-established treatment for sudden sensorineural hearing loss (SSNHL), especially in cases refractory to corticosteroid therapy.[3] However, its role in post-traumatic SSNHL remains poorly defined, particularly when temporal bone fractures are absent or when the otic capsule is spared. This case report explores the first use of HBOT as a salvage treatment for post-traumatic SSNHL in a paediatric patient, providing valuable insights into the potential efficacy of this therapeutic modality for trauma-induced hearing loss.

Case report

The patient's legal guardian gave written consent for publication of this case history.

A 13-year-old male was admitted to the emergency department of a tertiary hospital after a right temporal head trauma with loss of consciousness during a football game. Upon regaining consciousness, he reported tinnitus and hypoacusia in the right ear, along with severe holocranial headache. He had no relevant past medical history, with no known drug allergies and up-to-date immunisations.

On admission, he was haemodynamically stable, afebrile, Glasgow Coma Score (GCS) 15, and had no neurological deficits on examination. Physical examination revealed erythema in the right periauricular area and haemotympanum

with an intact tympanic membrane on the right. Acumetric testing with 256 Hz and 1,024 Hz tuning fork revealed a Weber test lateralising to the right and an absent Rinne on the same side. Audiological testing was not performed at admission.

A computed tomography (CT) scan of the ear (Figure 1) showed a right longitudinal temporal fracture extending superiorly to the external auditory canal and temporal squama (without misalignment). The fracture crossed the mastoid cells and extended to the epitympanic lateral wall, sparing the otic capsule. A tissue opacity was observed in the ipsilateral posterior and inferior mastoid cells, as well as in the recesses of the posterior wall of the tympanic cavity and adjacent to the oval window, which was interpreted as bleeding.

The patient was admitted to the paediatric special care unit and treated with fluids, analgesics, and intravenous amoxicillin-clavulanic acid (50 $mg \cdot kg^{-1} \cdot dose^{-1}$). Forty-eight hours later, he developed rotational vertigo accompanied by nausea and vomiting, along with subjective worsening of the right-sided hypoacusia. Examination revealed a grade II horizontal nystagmus to the left and a pathological head impulse test (HIT) to the right. Follow-up CT scan showed near-complete reabsorption of blood in the middle ear and the audiogram demonstrated moderate sensorineural hearing loss (SNHL) on the right side, involving frequencies between 2,000–8,000 Hz (Figure 2). Prednisolone (1 $mg \cdot kg^{-1}$ for 15 days) and betahistine (24 mg every 12 hours for one month) were prescribed for a presumed post-traumatic inner ear irritative process. The patient was discharged three days later with resolution of the vertigo but no improvement in hearing.

At three months follow-up, the patient continued to report right-sided hypoacusia. The audiogram did not show significant changes (Figure 3) and videonystagmography testing was normal. HBOT was recommended and initiated three months post-trauma, consisting of 11 sessions at 253 kPa (2.5 atmospheres absolute) with 100% oxygen for approximately 70 minutes per session. Following HBOT, the patient reported complete symptom resolution, and an audiogram revealed complete recovery of SNHL (Figure 4).

Figure 1
Axial CT scan image demonstrating a right longitudinal temporal bone fracture (white arrow)

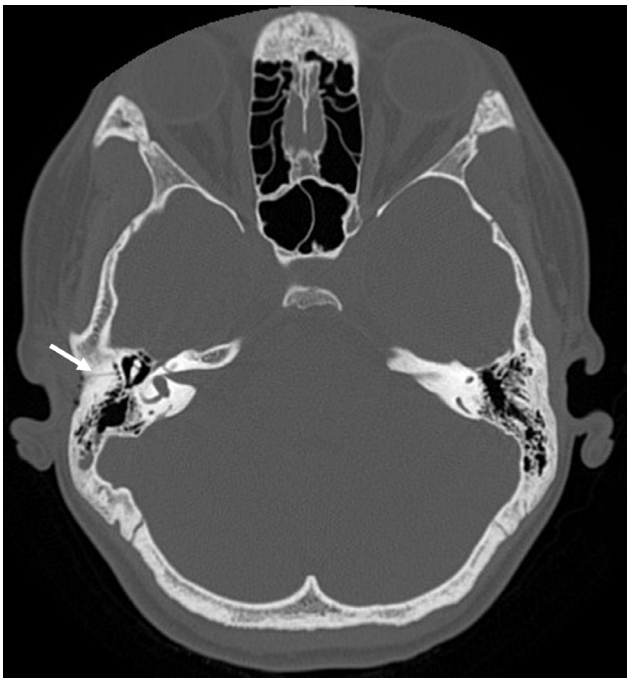
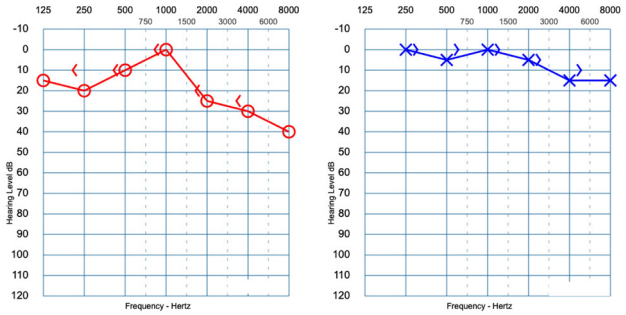


Figure 2
Pure tone audiometry showing moderate sensorineural hearing loss in the right ear (red, open circles)



Discussion

We describe a case of post-traumatic sudden sensorineural hearing loss (SSNHL) in which complete recovery after a three-month period with no improvement was associated with HBOT. To the best of our knowledge, this is the first report demonstrating potential efficacy of HBOT for sensorineural hearing loss of traumatic etiology.

Clinical studies have shown that head trauma can cause hearing loss in 15% to 66% of adults.[4] Temporal bone trauma resulting from head injury can lead to hearing loss with or without the presence of a temporal bone fracture. Since the 1940s, such fractures have been classified based on their angle relative to the petrous ridge into longitudinal, transversal or mixed (comminuted or oblique) types.[5] Longitudinal fractures tend to be associated with ossicular chain disruption and subsequent conductive hearing loss, whereas transverse fractures more commonly led to sensorineural hearing loss due to trauma to the labyrinth.[6,7] More recently, classification into otic capsule violating versus otic capsule sparing fractures has proven to be more informative in terms of prognosis, with capsule violating fractures linked to higher incidence of SSNHL, nerve disruption, cerebrospinal fluid fistula, facial nerve paralysis and intracranial complications.[8–10]

Our patient had an otic capsule sparing longitudinal fracture, which likely accounted for the immediate hearing loss (conductive, as indicated by acumetric testing), most probably caused by the haemotympanum. However, it

Figure 3
Pure tone audiometry three months after the event, showing no improvement in the moderate sensorineural hearing loss in the right ear (red, open circles)

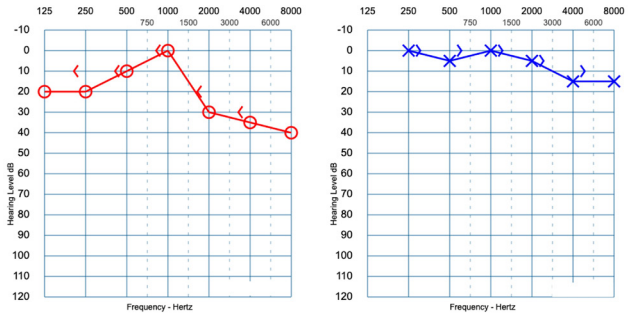
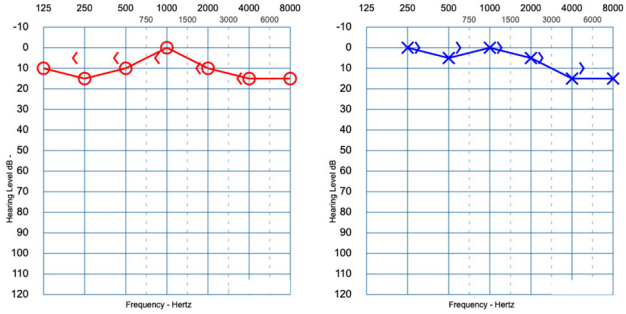


Figure 4
Pure tone audiometry showing complete recovery of sensorineural hearing loss after HBOT



is noteworthy that vertigo and worsened hearing loss (sensorineural, as confirmed on the audiogram) developed 48 hours post-injury, rather than immediately. This delayed onset can be explained by secondary audiovestibular loss, likely due to a labyrinthine concussion mechanism. Haemorrhage caused by a microfracture of the bony labyrinth could infiltrate the peri or endolymphatic spaces, leading to neuroepithelial degeneration and causing a labyrinthine concussion. In such cases, vestibular symptoms tend to cease over days as anatomic lesions heal and central balance mechanisms are reestablished, while hearing loss often persists.[11,12] Another possible explanation for this case would be a traumatic perilymphatic fistula, where tension-induced pressure transmission through the cochlear aqueduct or internal auditory canal would lead to oval window rupture. In such cases, diagnostic testing often appears normal, and a definitive diagnosis requires surgical exploration of the middle ear.[13]

Regarding treatment, initial management with corticosteroids and antibiotics followed common clinical practice. The use of HBOT as salvage therapy was proposed after persistent hearing loss at three months. The risks discussed with the patient and family included middle ear barotrauma, sinus and paranasal sinus barotrauma, ocular side effects, oxygen-induced seizures and claustrophobia.[14]

Clinical HBOT is defined as placing a patient in an increased pressure environment and having them inhale 100% oxygen for a defined period per treatment.[15] Studies have proved the efficacy of HBOT in treating idiopathic SSNHL, especially when combined with steroids, showing better outcomes in patients with severe or profound hearing loss (≥ 70 dB).[16,17] Furthermore, a 40-year review of research provided strong evidence supporting the use of HBOT for acute severe traumatic brain injury by reducing brain ischaemia protecting the neurovascular system.[18] However, its role in treating post-traumatic SSNHL remains less defined, particularly in instances where temporal bone fractures are absent or the otic capsule is spared.

Moreover, HBOT's efficacy is time-sensitive, with results diminishing as treatment is delayed, making it recommended to be started within 48 hours of diagnosis in idiopathic SSNHL.[19] Our case suggests a potentially wider therapeutic window for post-traumatic cases, possibly benefiting patients unable to receive early treatment, such as those admitted to intensive care units.

It is important to highlight that, given the natural course of sensorineural hearing loss, spontaneous recovery cannot be ruled out as the cause of hearing improvement in this case. Studies suggest that approximately 32% to 65% of patients with sudden SSNHL experience spontaneous recovery, with the majority of these improvements occurring within the first two weeks following onset.[20] Therefore, the causal relationship between HBOT and hearing recovery remains speculative and cannot be definitively proven. Only prospective randomised controlled trials with adequate sample sizes can confirm the efficacy of HBOT in post-traumatic SSNHL.

Conclusions

This case highlights the potential of HBOT as an effective salvage treatment for post-traumatic SSNHL. Additionally, it challenges the previously assumed strict time window for HBOT, suggesting that delayed intervention may still provide meaningful benefits. Future studies are necessary to establish clear treatment protocols and expand the understanding of HBOT's therapeutical potential in managing trauma-induced hearing loss.

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Cutaneous decompression sickness after an air dive with oxygen breathed during decompression in a commercial diver with a persistent foramen ovale

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Bubbles; Echocardiography; Occupational diving; Oxygen decompression; PFO; Transfer under pressure

Abstract

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A 43-year-old commercial diver had cutaneous decompression sickness after a dive to 17 metres of seawater for 160 minutes breathing air with transfer under pressure and oxygen breathed during decompression in a dry chamber. He had worked as a commercial diver for 16 years without previous problems. A bubble contrast transthoracic echocardiogram showed a large atrial right-to-left shunt. His persistent foramen ovale (PFO) was closed using a transcatheter technique and he has returned to commercial diving. As far as we are aware, shunt-mediated decompression sickness has not been reported previously after a shallow air dive with oxygen breathed during decompression. The findings in this diver adds to the observation of occurrence of three episodes of cutaneous decompression sickness after dry hyperbaric exposure breathing air and decompression whilst breathing oxygen in two individuals with atrial right-to-left shunts.

Introduction

The incidence of some forms of decompression sickness (DCS), particularly cutaneous, neurological and cochlear-vestibular DCS, is increased in divers with a clinically significant right-to-left shunt.[1–5] After many innocuous dives, small numbers of venous bubbles are liberated but, during passage through the pulmonary capillaries, the gas diffuses from the bubbles into the pulmonary alveoli and bubbles do not reach the systemic circulation. A right-to-left shunt, which allows paradoxical bubble embolism, increases the risk of DCS after dive profiles with a theoretically low risk.[2,6] It is believed that when paradoxical bubble embolism enables bubble emboli to invade tissues supersaturated with inert gas (usually nitrogen) after a dive, the bubbles are amplified as the dissolved gas in the tissue passes down the pressure gradient from the tissue into the bubbles, which increase in size to cause DCS.[6,7]

Similar degrees of pressure reductions in dry conditions can also cause DCS, but it is rare when modern commercial decompression profiles are employed. A review of the small number of reported cases in which appropriate investigations were performed suggests that a right-to-left shunt is present in most cases when DCS occurs after low risk decompression profiles in non-divers, such as hyperbaric tunnel workers and hyperbaric chamber attendants.[8] The review also suggests that the manifestations of shunt-mediated DCS were predominantly neurological in those who decompressed whilst breathing air and always cutaneous in those who decompressed whilst breathing oxygen to reduce tissue nitrogen load.[8] As far as we are aware, there are no comparable observations of DCS when oxygen decompression is used for shallow air dives.

We describe a commercial diver who had cutaneous DCS after a dive breathing air at a depth of 17 metres of seawater (msw) for 160 minutes with transfer under pressure and oxygen breathed during decompression in a dry chamber. He was found to have a large right-to-left shunt across a persistent foramen ovale (PFO). After the PFO was closed using a transcatheter technique he has been permitted to return to commercial diving.

Case report

The diver has seen this report of his history, confirmed its accuracy and has given written consent to its publication.

A male diver age 43-year-old, height 170 cm and weight 81 kg, had occasional migraine but he never had aura. There was no other significant medical history. He had smoked intermittently since his teens, but had not smoked for some months before his DCS.

During the incident dive in July 2024, he wore a suit heated with hot water. At the end of the dive for 160 minutes at 17 msw breathing air, he returned to a diving bell at a depth of 12 msw. He transferred under pressure to a dry chamber where he decompressed breathing 100% oxygen. Decompression was with the French number 5 (air/oxygen 12 m table) decompression table for an 18 msw dive for 180 minutes, which consisted of breathing 100% oxygen for 10 minutes at 12 msw, 15 minutes at 9 msw and 15 minutes at 6 msw.[9] After the dive he did not undertake unusual or strenuous activity. It was his practise of not showering for three hours after surfacing – so he did not shower in the one-hour period between finishing decompression and onset of symptoms.

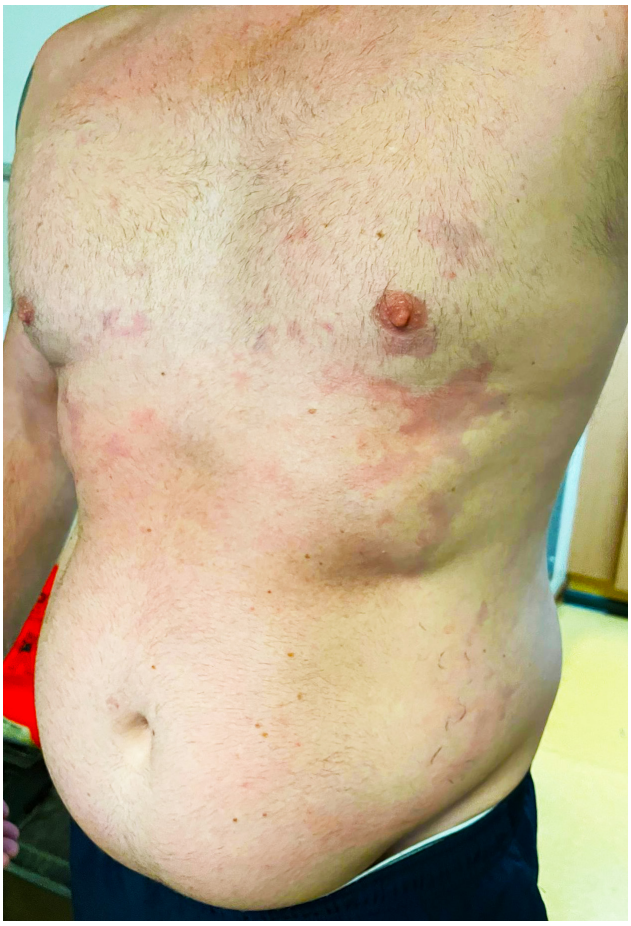
One hour after surfacing, he developed a pruritic erythematous marbled rash over the upper anterior abdomen which spread to his lower chest bilaterally associated with mild discomfort (Figure 1). There were no other abnormal findings and specifically neurological examination was normal. His rash and the associated discomfort completely resolved with a single recompression using US Navy Treatment Table 6. The other diver on the dive had no symptoms.

In the previous 16 years, the diver had performed more than 1,000 dives in the UK and abroad without any problems. During the first 10 years of his career, he had performed many relatively shallow air dives with long bottom times. During the next 18 months he performed more than 100 dives to depths of 30–40 msw with air as the breathing gas and surface decompression. The next period in his career involved dives used air or nitrox as the breathing gas, which only occasionally required in-water decompression stops, but when required the stops were performed using the same breathing gas. The exception during that period was a single 28-day trimix saturation dive with a storage depth of 60 m and maximum depth of 80 m. Thermal insulation on the dives consisted of dry suits, wet suits, water-heated suits or simple coveralls depending on the water temperature.

In 2024 he started a contract that required transfer under pressure with oxygen decompression. He did a total of ten dives. There were six days with one dive per day followed by one dive-free day. Then he did three dives in three days before the incident dive. All dives were similar to the incident dive, being at about the same depth as the incident dive with the transfer bell at 12 msw.

A transthoracic echocardiogram showed a structurally normal heart with a mobile interatrial septum. Bubble contrast injection when breathing normally showed a right-to-left atrial shunt with between 25 and 50 bubbles seen in the left heart on the stop-frame with the greatest number of bubbles. The shunt was slightly larger on a second bubble contrast injection with a sniff. On the third injection of bubble contrast with release of a Valsalva manoeuvre, the shunt was much larger with bubbles causing complete opacification of the left heart chambers. The patient was counselled about the implications of the finding and the options for management in accordance with the joint position statement on atrial shunts and diving.[10]

Figure 1
The rash of cutaneous decompression sickness in the diver



The diver had transcatheter closure of his PFO under fluoroscopy and transoesophageal echocardiography guidance using a 25 mm Amplatzer Talisman PFO Occluder (Abbott). Following the closure procedure, he took clopidogrel 75 mg daily for three months.

Ten weeks after the closure procedure transthoracic echocardiography showed that the alignment of the occlusion device was good. Six bubble contrast injections were given (one with normal breathing, two with sniffing and three with release of Valsalva manoeuvres). There was no evidence of a significant shunt. There were very occasional single bubbles seen in the left heart which might have been through pulmonary transit.

After cessation of clopidogrel, he was approved to return to diving. Since then he has performed 21 dives using air and nitrox as breathing gases. All were shallow and did not require decompression stops.

Discussion

The great majority of divers who have shunt-mediated DCS have followed a low-risk decompression profile that either required no stage decompression or required in-water decompression stops, which were performed correctly.[2,6]

The diver described in this report suffered cutaneous DCS after an uneventful dive that was within the limits of an accepted low risk decompression table. He was found to have a large right-to-left shunt across a PFO. He differed from the majority of divers who have shunt-mediated DCS because he transferred under pressure and performed the required decompression stops in dry conditions whilst breathing 100% oxygen. As far as we are aware this is the first report of shunt-mediated DCS after oxygen decompression in a diver. Though he performed a wet dive, his decompression was analogous to that of a hyperbaric tunnel worked or an attendant in a therapeutic hyperbaric chamber.

A recent case report of DCS in a hyperbaric worker that included a review of four additional cases in the literature found that shunt-mediated DCS is an important occupational risk for individuals with a large right-to-left shunt when working in hyperbaric air, but the manifestations of DCS differ in those who decompress whilst breathing oxygen compared with those who decompress whilst breathing air.[8] Prolonged oxygen breathing during decompression does not entirely eliminate venous bubbles in every decompression in every person and, in the two hyperbaric workers described, it was associated with cutaneous DCS on three occasions but not neurological DCS.[8] Decompression whilst breathing oxygen can be associated with migraine aura, which can be triggered by arterial bubbles without decompression.[11] In contrast, DCS was usually neurological when it occurred after dry decompression whilst breathing air.[8]

If further observations confirm that breathing oxygen during decompression protects against neurological DCS but not cutaneous DCS, it may provide a mechanism for reducing the more serious forms of DCS. It will also support the hypothesis that DCS resulting from paradoxical gas embolism is critically dependent on the peripheral amplification of bubble emboli in supersaturated tissues with a high nitrogen load, because the duration of oxygen breathing would have cleared nitrogen from tissues with rapid elimination half-lives such as neurological tissues, but not from tissues with slow elimination half-lives such as subcutaneous tissues.[6,7]

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Hyperbaric oxygen therapy for hypoglycaemic encephalopathy due to insulinoma: a case report

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Brain injury; Blood sugar level; Hyperbaric medicine; Hypoglycemia; Outcome

Abstract
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Introduction: Hypoglycaemic encephalopathy is a potentially life-threatening condition which can present with seizures, altered mental status or focal neurologic deficits. Therapeutic options are limited and the overall prognosis is poor. Among previously reported cases, the maximum time for patients to recover consciousness after hypoglycaemic encephalopathy was 14 days. So far, no studies have reported that hyperbaric oxygen therapy (HBOT) can improve the consciousness disorder of hypoglycaemic encephalopathy.
Case report: We report a case of hypoglycaemic encephalopathy caused by insulinoma who had a refractory consciousness disorder for 90 days and whose recovery was temporally related to institution of HBOT, suggesting that HBOT is a possible treatment for hypoglycaemic encephalopathy.
Conclusions: Hyperbaric oxygen therapy can be considered in hypoglycaemic encephalopathy when the hypoglycaemia has been corrected but patients still have reduced consciousness.

Introduction

Hypoglycaemic encephalopathy (HE) manifests as one of the prominent clinical features of severe hypoglycaemia. HE can present with seizures, coma or focal neurologic deficits and frequently be mistaken as stroke or other neurologic disorders.[1] HE due to insulinoma is rare and difficult to diagnose and treat.

There is controversy around the role of hyperbaric oxygen treatment (HBOT) in neurological disorders, with some evidence of benefit in acute and chronic traumatic brain injury,[2] but to date these problems do not appear on the Undersea and Hyperbaric Medicine Society list of approved indications.[3] Whether HBOT can treat the consciousness disorder caused by hypoglycaemia or insulinoma has not been reported. We report a case of a 65-year-old woman with HE caused by insulinoma whose recovery was temporally related to institution of HBOT. We highlight the potential for misdiagnosis and missed diagnosis in HE, and identify a new potential treatment option for refractory consciousness disorder caused by hypoglycaemia or insulinoma.

Case report

Written informed consent for publication of the clinical details, laboratory tests and clinical images was obtained from the guardian of the patient.

A 65-year-old woman was admitted to the emergency department because of paroxysmal dizziness and episodic cognitive impairment for two years and coma for two days. She had no significant past medical history and family history. She was free from drug or alcohol abuse. Her episodic cognitive impairment resulted in occasionally arriving at the wrong place and failing to find her way home. Events lasted for a few hours, and occurred once or twice a month. During this period the patient had been admitted to the emergency room and the blood glucose was normal. She had been hospitalised in the department of neurology and diagnosed as transient ischemic attack (TIA).

Two days before the admission described here, the patient suddenly appeared unconscious and unresponsive. She was unconscious at home for two days and medical treatment was not sought. On the third day, she was admitted to the

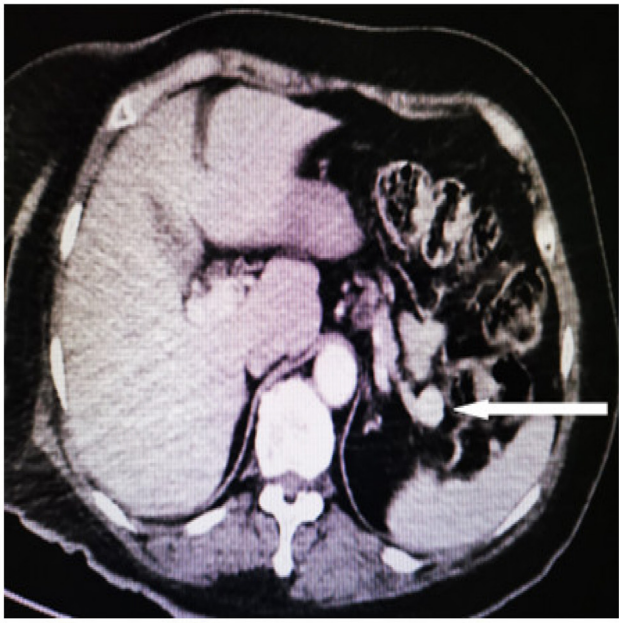
emergency room in a comatose state. Her blood glucose was undetectable (less than 1 mmol·L^{-1}) on arrival. Although hypoglycaemia was corrected after intravenous glucose injection, her consciousness did not recover. Laboratory serum tests demonstrated increased proinsulin, insulin and C peptide levels, implying inappropriate endogenous insulin production (Table 1). Pancreatic enhanced computed tomography was performed to confirm the diagnosis and location, and it revealed a mass in the tail of the pancreas (Figure 1). Combined with the patient's clinical manifestations, a diagnosis of insulinoma was finally made and surgical resection of tumor was performed. However, the patient's consciousness disorder did not improve after the operation with a Glasgow Coma Scale Score of 7 (E2V2M3).

Three months after the episode of coma, the patient was admitted to our department of hyperbaric oxygen medicine because of a refractory consciousness disorder unimproved over three months. The Glasgow Coma Scale Score was still 7 (E2V2M3), the same as previously. Brain magnetic resonance imaging (MRI) (Figure 2A) showed extensive bilateral lesions in the cerebral cortex, basal ganglia and periventricular areas. Electroencephalogram (EEG) showed many 10–40 uV and 3–7 c·s^{-1} δθ waves. She was diagnosed with HE and HBOT (253 kPa [2.5 atmospheres absolute], 60 min, once a day) was started. Surprisingly, after only three sessions the patient's consciousness was improved, showing a response to verbal stimuli and being able to speak inappropriate words. After 10 sessions the patient's cognitive function was further improved. She could open her eyes spontaneously and localise pain stimuli. After 30 sessions of HBOT, the patient's cognitive function was significantly improved. She could communicate in short sentences, eat by herself, stand and walk slowly with others' help (outcome scores are shown in Table 2). She tolerated HBOT well without any adverse effects. Re-examination of the brain MRI showed that the lesion area was smaller than that before HBOT (Figure 2B). Re-examination of the EEG showed that δθ waves were less than that before HBOT. The patient was satisfied with the treatment effect and discharged from the hospital. After half a year's follow-up, there was no significant change in patients' consciousness compared with that at discharge.

Table 1
Values of laboratory serum tests obtained during hypoglycaemic episode

Test	Value	Reference range
Blood glucose	< 1.0	4.1−5.9 (mmol·L^{-1})
Proinsulin	1750.40	30−180 (pg·mL^{-1})
Insulin	98.27	4.03−23.46 (mIU·L^{-1})
C peptide	3907.20	99.9–1242.1 (pmol·L^{-1})

Figure 1
Pancreatic enhanced computed tomography image outlining suspected pancreatic mass. Area in question is a 1.6 x 1.5cm mass in the tail of pancreas (highlighted by arrow)



Discussion

HE is a potentially life-threatening manifestation of hypoglycaemia, which can present with seizures, altered mental status or focal neurologic deficits.[1] HE due to insulinoma is rare and difficult to diagnose as it mimics a great variety of neurological conditions. In addition, as in an earlier presentation of this case, some patients' blood glucose levels have returned to normal when they arrive at hospital, which increases the difficulty of diagnosis and easily leads to missed diagnosis and misdiagnosis.[4] In this case, the patient suffered from dizziness and cognitive impairment repeatedly in the early stage of the disease, but her blood glucose level was normal in several visits to the hospital. So, the possibility of hypoglycaemia was missed, and she was diagnosed as TIA. Therefore, for patients with recurrent dizziness and cognitive impairment, the blood glucose should be measured repeatedly to avoid delay in diagnosis and treatment of insulinoma.

In the last episode of hypoglycaemia, the patient appeared unconscious, but her family thought it was still a TIA attack and observed her at home for two days without seeing a doctor, resulting in prolonged hypoglycaemia and severe brain damage. Even though the hypoglycaemia was corrected and the tumor was removed, the patient's consciousness disorder had not improved. Although the new definition of TIA does not take into account the duration of symptoms, symptoms of TIA generally do not exceed 24 hours, and most TIAs are considered to last less than 1 or 2 hours.[5] This suggests that it is necessary to carry out adequate education for the patient's family members, that is, if the patient's symptoms continue to be unrelieved, they should seek medical treatment as soon as possible.

Figure 2

Brain MRI images; (A) before HBOT, showing extensive bilateral lesions in the cerebral cortex, basal ganglia and periventricular areas; (B) after 30 HBOT sessions, the lesion area was substantially reduced

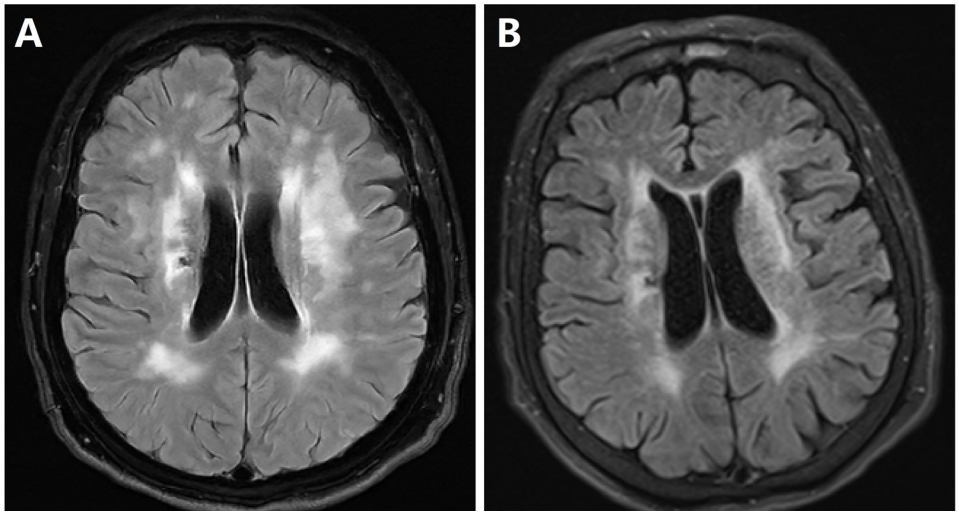


Table 2

Outcome scores; outcome was assessed using the Glasgow coma scale (GCS), coma recovery scale-revised (CRS-R) score, functional independence measure (FIM) and Barthel index (BI) before hyperbaric oxygen treatment (HBOT), after three sessions of HBOT, after 10 sessions of HBOT and after 30 sessions of HBOT

Parameter	Before HBOT	After 3 HBOT	After 10 HBOT	After 30 HBOT
GCS	E2V2M3	E3V3M4	E4V4M5	E4V5M6
CRS-R	1	4	9	15
FIM	18	18	24	55
BI	0	0	20	60

HBOT is considered to be safe and well-tolerated, and although there is controversy about efficacy in brain injury, there is some evidence for benefit following brain trauma and stroke. However, whether HBO can treat the consciousness disorder caused by hypoglycaemia / insulinoma has not been reported. In addition, it has been suggested that HBOT is unlikely to revert severe brain injury with major neuron loss and therefore should be considered mainly at the early stage of relevant disorders, when only minimal cognitive deficiency was detected.[2] Among previously reported cases, the maximum time for patients to recover consciousness after HE was 11 days,[6] 13.3 days[7] and 14 days[8] respectively. However, in this case, the patient's consciousness had been severely disturbed for 90 days, and improved after only three sessions of HBOT, suggesting that improvement of consciousness could occur even after 90 days after HE, and that HBOT is a potential treatment even if it is not given in the early stage of the disease. Thirty sessions of HBOT was associated with significant improvement of the patient's cognitive function, suggesting that for cognitive impairment caused by hypoglycaemia, if the patient's neurological function failed to meet expectations, HBOT should be continued for at least 30 sessions. However, no firm conclusions can be based on a case report and further observational reports in more patients are needed.

Conclusions

For patients presenting with recurrent episodic dizziness and cognitive impairment, blood glucose should be measured repeatedly to avoid delay in diagnosis and treatment of insulinoma. In patients who have suffered HE, HBOT can be considered when the hypoglycaemia has been corrected but patients still exhibit a consciousness disorder, even if it is not in the early stage of the disease.

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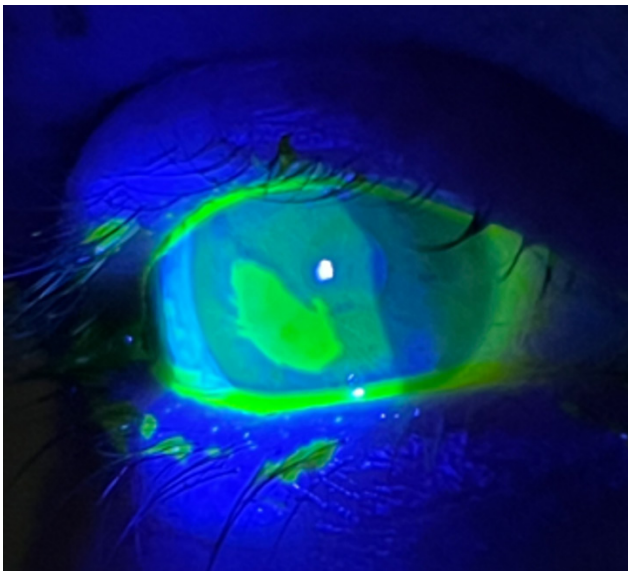
Images in diving and hyperbaric medicine

Blurred vision after diving, what's your diagnosis?

Case and image

We present the case of a 50-year-old woman with no notable antecedents who has been diving for several years without any particular problems (Advanced Open Water Diver, 80 dives in total). She is not currently undergoing any treatment and has no known allergies. In August on the French Mediterranean coast, she participated in a week-long diving trip in the 20-metre zone, with one dive per day. She is not fatigued, is hydrating regularly, is not having trouble sleeping, and is not ill. Her last dive, to a maximum depth of 18 m for a total duration of 44 minutes, proceeded without incident. The ascent speed was correct, and the decompression procedure was respected. On the surface, she immediately noticed blurred vision in her right eye. Once on the boat, she reported her symptoms and was given oxygen at 15 L·min^{-1} before being evacuated to the hyperbaric centre. On admission 1.5 hours after exiting the water, she still had blurred vision, now associated with a burning sensation in her right eye. She described no foreign body event, and did not need to clear her mask during the dive. Examination revealed a white eye with no visible foreign body and normal ocular movement. The right eye is slightly watery with blurred central vision. The remainder of the clinical examination was otherwise normal. The fluorescein examination is shown in Figure 1.

Figure 1
Fluorescein examination of the right eye



What is your diagnosis?

1. Neurological decompression illnesss
2. Ocular barotrauma due to mask squeeze
3. Keratitis due to jellyfish sting
4. Toxic keratitis
5. Herpetic keratitis

Discussion and multiple choice question result

In the presence of visual disturbances on exiting the water, neurological cerebral decompression illness should be the first consideration, as this is an emergency requiring hyperbaric admission as soon as possible.[1] Visual disturbances such as diplopia, visual field changes or, less commonly, cortical blindness may be observed. Decompression sickness is more likely after a dive to a depth greater than 30 meters, or long, prolonged dives requiring decompression stops, repetitive dives, or dives with yo-yos. Rapid ascent with expiratory blockade can also be responsible for pulmonary barotrauma with cerebral air embolism, which can also lead to visual disturbances.[1]

The described case does not correspond to this situation and the history now includes unilateral ocular burning, suggesting a local ocular cause. Examination with fluorescein blue light revealed corneal damage. The etiologic search for a keratitis is then in the foreground.

In novice divers (which is not the case here), failure to compensate for the negative pressure of the mask during descent may result in benign subconjunctival hemorrhage that resolves in a few days, but this form of barotrauma does not manifest as keratitis.[1]

The most common causes of keratitis are traumatic, infectious or allergic. Questioning in this case appears to rule out trauma. Infectious and allergic causes most often result in keratoconjunctivitis, which is not the case here as the conjunctiva is spared. Herpetic keratitis can present as unilateral keratitis, but the most common presentation is that of a corneal ulcer with an arborescent or dendritic appearance. The patient has no history of oral or genital herpes. Keratitis associated with soft contact lens infection could also be considered, as in a case of *Pseudomonas aeruginosa* infection associated with scuba diving,[2] but in our case the patient does not wear contact lenses.

In addition to diving, there may be an environmental cause related to exposure to marine organisms that can cause skin or eye stings. Cases of keratitis have been described after exposure to sea anemones[3] or red coral.[4] In the latter case, ocular damage is triggered by red coral nematocysts, organelles that release toxins. In the Mediterranean, the most likely exposure is to jellyfish or floating debris of jellyfish filaments. In this case, however, facial burns would probably be associated. Furthermore, the absence of mask clearing during the dive rules out this hypothesis.

Upon further questioning, the patient reveals that she had been using a special anti-fog spray to prevent her mask from fogging up. Toxic keratitis associated with the use of anti-fogging agents is a relatively rare cause, but one that we have seen in our hyperbaric center. Mask fogging is a common problem in scuba diving. Applying saliva to the inside of the mask lens limits fogging, but does not always eliminate it. On the other hand, the application of saliva can lead to hygiene problems. This has led to the development of commercial anti-fog products designed for use on diving masks. These products may contain volatile compounds that are potentially toxic to the corneal epithelium, such as glycols, alcohols, surfactants and phenol derivatives.[1] Exposure to these compounds may cause blurred vision, photophobia, lacrimation, and blepharospasm that manifests shortly after diving. Fluorescein examination usually reveals diffuse superficial punctate keratopathy. This complication usually results from improper use of the anti-fogging agent, either by over-application of the product or by failure to rinse the mask prior to application to the face. The instructions for use for anti-fog products clearly state that the mask should be rinsed or wiped thoroughly after application of the spray. However, it has been found that this recommendation is not always followed, and in this case, rinsing was not performed. Wright described two cases of toxic keratitis in recreational divers with symptoms of corneal lesions appearing one to three hours after exposure to these anti-fogging agents.[5] In both cases, corneal lesions were limited to diffuse superficial punctate keratopathy that resolved within 24 hours without scarring or permanent damage. A more recent case describes the occurrence of unilateral keratitis with stromal edema after massive spraying of an anti-fogging agent on swimming goggles.[6] Topical corticosteroids, antibiotic eye drops, and oral tetracycline were initiated. Epithelial involvement and diffuse stromal opacity resolved rapidly, but white subepithelial plaques were observed in the central cornea at the one-year follow-up. These plaques disappeared completely after one month of treatment with topical corticosteroids.

In our case, the involvement was limited to superficial epithelial keratitis. Treatment with antibiotics and vitamin A ointment was initiated, with consultation of an ophthalmologist to monitor progress.

After considering the different diagnoses, the correct answer to the multiple choice question is 4.

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Blindness, sudden; Decompression illness; Keratitis; Ophthalmology; Scuba diving

Letter to the Editor

Commentary on the article by Stevens G, Smart DR. The influence of wetsuit thickness (≥ 7 mm) on lung volumes in scuba divers

We read with interest the recent article by Stevens and Smart "*The influence of wetsuit stiffness on lung volumes*".[1] The substantial number of participants and the diversity of their diving backgrounds allowed the authors to draw interesting comparisons of wetsuit effects between occupational and recreational divers, as well as between women and men. These add valuable data to the understanding of physiological demands in various diving conditions.

Since we have also investigated the side effects of wetsuits, we would like to offer some additional observations.

Wet suits are designed to minimise water movement between the garment and the diver's skin. To achieve a close fit without loose areas, they are made from elastic materials, predominantly neoprene. Such material can vary in thickness and stiffness, but in all cases its elastic properties generate a recoil force when stretched. Around cylindrical body segments such as the arms and legs, this recoil produces an inward centripetal pressure that can be measured with small flat balloons used as pressure transducers. In one study assessing the effects of wetsuits effect on fluid balance and urine flow, a 5 mm neoprene suit exerted a limb compression of approximately 25.8 mmHg, similar to class II-III 18 therapeutic stockings.[2] In another study, a custom-made neoprene suit comprising two layers (3.5 mm and 5.5 mm) for a total thickness of 9 mm produced a compression of 30 mmHg on the skin.[3]

This recoil pressure also applies on the thoracic and abdominal walls, tending to reduce circumferences and to limit the amplitude of ventilatory volume changes. During expiratory manoeuvres either slow or forced this elastic restriction facilitates exhalation. Accordingly, the greater reductions in forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1) observed with thicker suits appear to be a logical consequence of the higher pressure generated by thicker materials.[1] During immersion and diving, the hydrostatic pressure adds a fluid-redistributing effect to that of the wetsuit, as evidenced in the enhanced diuretic response.[2]

A reduction in lung compliance is an additional consequence of the decreased gaseous lung volume, which in turn makes lung inflation more difficult. In intensive care settings, patient positioning is known to influence regional lung ventilation and is a key factor in optimising ventilatory support.[4] Hydrostatic pressure has been shown to produce similar effects during surface immersion in healthy individuals.[5]

Both the elastic restriction of lung volumes and the stiffness of the material therefore contribute to an increased inspiratory work of breathing and its associated cardiovascular consequences.[6,7] An increase in the work of breathing can, by itself, provoke unpleasant dyspnoea,[8] which may be hazardous during diving.[9] On average, the work of breathing rises more rapidly in women than in men, due to smaller lung volumes and airway calibres. Moreover, increased inspiratory work is a key contributor to immersion pulmonary oedema, especially during exercise.[7,10,11] Indeed a higher inspiratory work due to a reduced lung or thoracic wall compliance decreases the gaseous lung volume, lowers the inspiratory pleural and mediastinal pressure[5,12] and concomitantly reinforces the preload[7] and in turn the congestion of the pulmonary and bronchial vessels. Even a thin neoprene vest has been shown to aggravate dyspnoea and haemodynamic impairment during land-base cycling exercise.[13]

If the authors were to undertake a similar study under immersion, as they seem to suggest, we would respectfully propose that it would be of interest to measure the inspiratory capacity and the inspiratory pressures both during spontaneous breathing and during maximal inspiration, without a suit and with a suit, both on land and while immersed.

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Notices and news

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

President's report

Bengüsu Mirasoğlu

This is my first time writing as President, I would like to take this opportunity to express how privileged I feel to serve this society. Years ago, when I attended my first meeting in Aberdeen, certainly with no idea that I could one day become President, I was deeply impressed by the scientific work being presented and by how warmly I was welcomed as a new and young diving and hyperbaric physician. I had just completed my residency and was trying to find my path. EUBS made me wonder, question, search for answers, and ultimately research and ask even more questions. This society has played an important role in shaping my professional journey, offering mentorship, scientific inspiration, and a community of colleagues who continually push the boundaries of medical knowledge. I am truly grateful for all it has contributed to my development.

Over the years, our society has stood as a beacon of scientific rigor, collaboration, and innovation. We will continue to support the development of scientific knowledge, encourage the exchange of ideas across borders, and create spaces where young researchers and experienced experts alike can thrive. In this way, we will keep moving our field forward.

Just a few months ago, we had the pleasure of meeting in Helsinki for our annual scientific meeting. It was a highly successful event with engaging workshops, thought-provoking keynote lectures, fruitful presentations and exciting social programme. I would like to thank to Dr Anne Räisänen-Sokolowski and the entire organising committee, as well as to all speakers and contributors who helped make this meeting a true scientific feast. We are already looking forward to gathering again next year in Geneva.

As we welcome a new year, I extend my warmest wishes to all members of the European Underwater and Baromedical Society (EUBS), the South Pacific Underwater and Hyperbaric Medical Society (SPUMS), as well as the broader community of diving professionals and medical practitioners around the world. May the year ahead bring health, inspiration, and renewed energy for the work we are called to do. Wishing you a joyful holiday season and a bright, fulfilling New Year.

Bengüsu Mirasoğlu
President EUBS

The EUBS website is at
http://www.eubs.org/
Members are encouraged to log in and keep their personal details up to date.

The latest issues of *Diving and Hyperbaric Medicine* are via your society website login.

EUBS Notices and news

EUBS 2026 Annual Scientific Meeting

After the great success of the 49th Annual Scientific Meeting in Helsinki, Finland, with 318 registered participants from 46 countries, EUBS will move to Geneva, Switzerland for its 50th (anniversary) meeting, which will take place from 14–18 September 2026. It will be a full week of science and important decisions, as it will also be the occasion for the next major Consensus Conference on the Indications for Hyperbaric Oxygen Therapy, organided by the ECHM (European Committee for Hyperbaric Medicine). EUBS fully supports this conference both logistically and financially and cordially invites you to be a part of this important update of the good practice of our clinical practice.

The congress website, www.eubs2026.com, gives you all details and information on this joint EUBS-ECHM meeting as it becomes available. A plea for all who consider attending please do take advantage of the early-bird registration rates (until 26 April 2026) and book your spot early.

EUBS will be delighted to welcome you to this meeting and help contribute to its success.

EUBS Annual General Assembly

Our annual EUBS General Assembly took place on 5 September 2025, in Helsinki, traditionally on the last day of the EUBS Annual Scientific Meeting.

The report of the General Assembly, as well as the supporting documents (financial report, results of ExCom elections) is available for our members via the 'Member area' on the EUBS website.

It has been decided that the membership fees for the next year will remain the same as last year. Also, the price for the 'print option' for the DHM Journal will remain at €50, which, considering the increased printing costs and a higher number of pages per issue, is still a bargain.

EUBS ExCom expresses their appreciation and thanks to our Corporate Members, as well as to our 10 'Affiliated Societies' – national scientific societies and organisations supporting and promoting EUBS among their members, who benefit from a 10% reduction in EUBS membership fee.

EUBS Executive committee

This year, we have had to elect a new Vice-President. Indeed, Jean-Eric Blatteau has reached the end of his term as President of our Society and is replaced by Bengüsu Mirasoğlu. There was one candidate for this position, Anders Kjellberg, who was elected with almost unanimity. To replace Evangelos Papoutsidakis from Barcelona (Spain) after serving a four-year term, as well as Anders Kjellberg (our Member-at-Large 2024), we have elected two new Members-at-Large. There were three candidates: Pieter Bothma (United Kingdom), Gamze Sümen (Turkey) and Benoît Desgraz (Switzerland).

After a very close race, Pieter Bothma was elected as the Member-at-Large 2024 (for a three year term, replacing Anders Kjellberg) and Benoit Desgraz was elected as Member-at-Large 2025 for a four-year term. Thanks to Vangelis for his service to the Society, and of course to Jean-Eric, who will continue on the ExCom as Immediate Past President. This unfortunately also means that Jacek Kot, our Past President, leaves ExCom after having dedicated in total more than 12 years for EUBS.

The Executive committee wish to express their thanks to all candidates and Members-at-Large for their willingness to help our Society move forward and their contributions to the ExCom activities. The composition of the new ExCom can be found on the EUBS website, with contact information for each member.

EUBS Social media

All EUBS members are reminded to bookmark and follow our Social Media channels:
Facebook: https://www.facebook.com/European-Underwater-and-Baromedical-Society-283981285037017/
Twitter by X: @eubsofficial
Instagram: @eubsofficial

While the 'EUBS website news' email messages are a way to communicate important information directly to our EUBS members, Facebook, Twitter and Instagram will be used to keep also non-members updated and interested in our Society. The EUBS social media is managed by Bengüsu Mirasoğlu (bengusu.mirasoglu@eubs.org).

EUBS membership

Remember to renew your EUBS membership. If your membership has expired, you will see a message when trying to log in on the EUBS website. You can then immediately renew it online.

EUBS membership gives you significant advantages, such as immediate access to the most recent issues of the DHM Journal, (if selected) a print copy of the eJournal for your convenience, reduced registration fee at our Annual Scientific Meeting (this benefit alone already covers the cost of your membership), reduced membership fees at selected Affiliate Societies, access to the GTUEM database of non-indexed scientific literature, searchable membership database, etc.

Members of Affiliate Societies benefit from a 10% discount on the EUBS membership fee. When applying for or renewing your membership, select your Affiliate Society from the drop-down list and the reduction in membership fee will be automatically applied.

In case you have difficulties renewing or accessing your Membership Area, please contact us at secretary@eubs.org. Please do note that payment by PayPal is by far the easiest and also cheapest way to pay your membership fee.

You can also pay by bank transfer, but please note that you have to pay the banking costs for international money transfers (EUBS is registered in the UK, which is now outside of Europe). You have to make sure to select this ("*all banking costs carried by the sender*") when you make the transfer. If not, our bank will refuse your payment.

Also, the money transfer may take up to one week and may fail for some obscure reason.

Finally, ensure you write your name (the EUBS member whose membership you are renewing) as the ONLY information in the message attached to the payment, or we cannot identify the payment and your membership may not be renewed as expected.

Therefore, unless you are in the UK, we do not recommend this payment option. Using Wise (formerly 'Transferwise') is another option to reduce or avoid banking costs and have a faster and secure transfer of your membership fee.

EUBS website

Visit our EUBS website for the latest news, conferences and meetings, endorsed documents and courses. You can also find information on travel and research grants, employment opportunities, research projects looking for multicentric collaboration, and much more.

The OXYNET database, previously managed by the European Committee for Hyperbaric Medicine (ECHM) is now an integral part of the EUBS website, and can be consulted through a Europe (and World) Map interface, through the Menu item 'OXYNET Map' (sounds logical) or directly at www.eubs.org/oxynet (or http://www.eubs.org/?page_id=1366)

Have a look at the 'EUBS History' section which has been added under the Menu item 'The Society'. There is still some information missing in the list of EUBS Meetings, Presidents and Members-at-Large – please dig into your memories and help us complete this list.

Please also have a look at our Corporate members – societies and companies who support the EUBS by their membership. Their logos and contact information can be found at the Corporate members page (http://www.eubs.org/?page_id=91).

In case you have any suggestions for adding or correcting the info posted, please contact us at webmaster@eubs.org.

Merger of the European Committee for Hyperbaric Medicine (ECHM) with EUBS

European Committee for Hyperbaric Medicine

In 2025, the ECHM and the EUBS finally agreed to merge. This step will allow both organisations to strengthen their joint message on safety standards in hyperbaric oxygen therapy, the training of medical personnel, the preparation of indications for hyperbaric treatment, and the planning of scientific research in the field of diving and hyperbaric medicine.

The decision will come into effect in 2026. Its first outcome will be the ECHM–EUBS Conference in Geneva (14–18 September 2026), where, on the opening day, an invited expert panel will hold a consensus session presenting a new list of indications for HBOT based on an analysis of clinical evidence.

Further details will be provided in subsequent communications.

Jacek Kot
President, ECHM

SPUMS
South Pacific Underwater Medicine Society

Notices and news

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

SPUMS President's report

Neil Banham

Preparations for the SPUMS 54th Annual Scientific Meeting (ASM) in 2026 to be held at the Palasia Hotel in Palau next May are well under way, with about 120 registrants so far. It promises to be a great meeting, with a diverse programme and fabulous diving.

2026 SPUMS ASM
Date: 10–15 May 2026
Venue: Palasia Hotel, Palau
Theme: *Free diving*

Qantas fly to Koror, Palau, departing Brisbane on Saturday mornings and returning Sunday morning. Flights to Koror may also be via Taipei or Tokyo (Narita). Please contact spumstravel@diveplanit.com if you would like assistance with flights.

Registration, then booking of accommodation, diving and a pre or post conference liveaboard diving trip (via Diveplanit) are now available via the SPUMS website. South Pacific Underwater Medicine Society - SPUMS-ASM.

The **2027 SPUMS 55th ASM** has been confirmed as follows:
Date: 16–22 May 2027
Venue: Atmosphere Resort, Dumaguete, Philippines
Theme: *Innovation in Diving and Hyperbaric Medicine*
Convenors: Cathy Meehan and Lizzie Elliott

Register your interest now for a pre-ASM liveaboard trip to Tubbataha. Diveplanit has optioned a full charter on the Philippines Aggressor II for the 8–15 May trip which fits well with the conference dates. https://diveplanit.wetravel.com/trips/spums-2027-philippines-tubbataha-liveaboard-add-on-diveplanit-travel-pty-ltd-99120653. More information will be available soon, save the date! Options for our 2028 ASM are being considered – further ideas are welcome.

The SPUMS Position Statement regarding paediatric and adolescent diving, published in the December 2024 issue of *Diving and Hyperbaric Medicine*, has just been made into an educational video by Richard 'Harry' Harris, co-hosted with Lizzie Elliott and dive instructor Charlotte Barbosa. Thank you to John Lippmann and the Australasian Diving Safety Foundation (ADSF) for funding this and making it happen. The video aims to educate parents, dive instructors, medical professionals and the prospective divers. It is available here https://www.adsf.org.au/education and will be available via the SPUMS website.

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

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

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

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

The Mike Bennett Scholarship applications for 2026 close on the 31 December 2025. The successful applicant will be funded to attend a Scientific Meeting of relevance to diving and hyperbaric medicine. Full details can be found here: South Pacific Underwater Medicine Society - SPUMS - Mike Bennett Scholarship.

On behalf of ExCom, I would like to wish all members and their loved ones a safe and happy Christmas and New Year and a fabulous 2026.

Dr Neil Banham
SPUMS President

Mike Bennett Scholarship

Dr Sue Pugh, the wife of the late Professor Mike Bennett AM (a past SPUMS President and mentor to many), has bequeathed funds to create a Scholarship ('The Mike Bennett Scholarship') to fund the successful applicant to attend a Scientific Meeting of relevance to diving and hyperbaric medicine.

Suitable meetings may include (but are not limited to) the Annual Scientific Meeting (ASM) of South Pacific Underwater Medicine Society (SPUMS), Undersea and Hyperbaric Medical Society (UHMS), European Underwater and Baromedical Society (EUBS), Hyperbaric Technicians and Nurses Association (HTNA), British Hyperbaric Association (BHA).

The Mike Bennett Scholarship will be offered annually with one successful applicant chosen if they are considered to meet the selection criteria. The Scholarship may not be awarded in any given year if the applications received are not deemed suitable by the Selection Panel.

The Mike Bennett Scholarship is open to anyone working in the field of diving and hyperbaric medicine, including doctors, technical staff, nurses and those performing research in the field. Applications from those from Pacific nations who might not otherwise have the opportunity to attend an international scientific meeting are also encouraged.

Selection of the successful applicant will be overseen by a SPUMS Selection Panel comprising:

Dr Sue Pugh
SPUMS President (currently Dr Neil Banham)
SPUMS Immediate Past President (currently Professor David Smart)
SPUMS Education Officer (currently Dr David Cooper)
Diving and Hyperbaric Medicine Journal Editor (currently Professor Simon Mitchell)

The successful applicant for The Mike Bennett Scholarship will have the actual costs of ASM Registration, travel and accommodation funded to a maximum of AUD $10,000. However, the applicant will be responsible for all other expenses incurred.

There are no rigidly defined selection criteria, however, preference will be given to the following:

- SPUMS members
- Presenting at the ASM:
  - (1) A diving or hyperbaric medicine presentation
  - (2) An evidence-based medicine presentation
- Those who have previously made a significant contribution to SPUMS.

Applications should include a brief synopsis (1–2 pages) of the project and be submitted to president@spums.org.au.

Closing date: 31 December 2025

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

SPUMS Diploma in Diving and Hyperbaric Medicine

(Updated June 2025)

Requirements for candidates

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

- be medically qualified;
- remain a current financial member of the Society for the duration of their candidacy for the Diploma;
- pay such administrative fees and charges (e.g., candidate registration fee) as may, from time-to-time, be approved by the Society's Executive;
- supply evidence of satisfactory completion of an examined two-week fulltime course in Diving and Hyperbaric Medicine at an approved facility. The list of such facilities may be found on the SPUMS website;
- have completed the equivalent (as determined by the Education Officer) of at least six months' fulltime clinical training in an approved Hyperbaric Medicine Unit;
- submit a written proposal for research in an area of relevance to underwater or hyperbaric medicine, in a standard format, for approval *before* commencing their research project;
- produce, to the satisfaction of the Academic Board, a written report on the approved research project, in the form of a scientific paper suitable for publication. Accompanying this written report should be a request to be considered for the SPUMS Diploma and supporting documentation for 1–5 above.

In the absence of documentation otherwise, it will be assumed that the paper is to be submitted for publication in *Diving and Hyperbaric Medicine*. As such, the structure of the paper should broadly comply with the 'Instructions for authors' available on the SPUMS website www.spums.org.au or at South Pacific Underwater Medicine Society - Submitting to DHM.

The paper may be submitted to journals other than *Diving and Hyperbaric Medicine*; however, even if published in another journal, the completed paper must be submitted to the Education Officer for assessment as a diploma paper. If the paper has been accepted for publication or published in another journal, then evidence of this should be provided.

The diploma paper will be assessed, and changes may be requested, before it is regarded to be of the standard required for award of the Diploma. Once completed to the reviewers' satisfaction, papers not already submitted to, or accepted by, other journals should be forwarded to the Editor of *Diving and Hyperbaric Medicine* for consideration. At this point the Diploma will be awarded, provided all other requirements are satisfied. Diploma projects submitted to *Diving and Hyperbaric Medicine* for consideration of publication will be subject to the Journal's own, independent peer review process.

Additional information – prospective approval of projects is required

The candidate must contact the Education Officer in writing (email is acceptable) to advise of their intended candidacy, and to discuss the proposed topic of their research. A written research proposal must be submitted before commencing the research project.

All research reports must clearly test a hypothesis. Original basic or clinical research is acceptable. Case series reports may be acceptable if thoroughly documented, subject to quantitative analysis, and the subject is extensively researched and discussed in detail. Reports of a single case are insufficient. Review articles may be acceptable if the international literature is thoroughly analysed and discussed, and the subject has not recently been similarly reviewed. Previously published material will not be considered. It is expected that the research project and the written report will be primarily the work of the candidate, and that the candidate is the first author where there are more than one. Evidence of each author's specific contributions should be provided in the case of multi-author papers.

The preferred format for submission of the final project is as a single file (Word or unlocked pdf), 1.5-line spaced, Times New Roman 12-point font, unformatted, with all figures and tables embedded in the document at an appropriate location.

It is expected that all research will be conducted in accordance with the joint NHMRC/AVCC statement and guidelines on research practice, available at: http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/r39.pdf. or the equivalent requirement of the country in which the research is conducted. All research involving humans or animals must be accompanied by documentary evidence of approval by an appropriate research Ethics Committee. Human studies must comply with the Declaration of Helsinki (1975, revised 2013). Clinical trials commenced after 2011 must have been registered at a recognised trial registry site such as the Australia and New Zealand Clinical Trials Registry http://www.anzctr.org.au/ and details of the registration provided in the accompanying letter. Studies using animals must comply with National Health and Medical Research Council Guidelines or their equivalent in the country in which the work was conducted.

The SPUMS Diploma will not be awarded until all requirements are completed. The individual components do not necessarily need to be completed in the order outlined

above. However, it is mandatory that the research project is approved prior to commencing research.

As of 01 July 2025, projects will be deemed to have lapsed if:
(1) The project is inactive for a period of three years, or
(2) The candidate fails to renew SPUMS Membership in any year after their Diploma project is registered (but not completed).

For unforeseen delays where the project will exceed three years, candidates must advise the Education Officer in writing if they wish their Diploma project to remain active, and a three-year extension may be approved. If there are extenuating circumstances why a candidate is unable to maintain financial membership, then these must be advised in writing to the Education Officer for consideration by the SPUMS Executive. If a project has lapsed, then the candidate must submit a new application as per these guidelines.

Fees and charges: From 01 January 2026 a one-off Registration Fee of AUD $250.00 will be payable at the time of enrolment for the Diploma. This is in addition to the annual Society Membership Fee.

The Academic Board reserves the right to modify any of these requirements from time to time.

As of June 2025, the SPUMS Academic Board consists of:
Dr David Cooper, Education Officer
Associate Professor Simon Mitchell.

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

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

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HBOE
HBOEvidence

HBOEvidence

HBOEvidence is seeking an interested person/group to continue the HBOEvidence site. The database of randomised controlled trials in diving and hyperbaric medicine: hboevidence wikis.unsw.edu.au. The HBOEvidence site in the process of being integrated into the SPUMS website.

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

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 are as below.

Dates: 16–27 February 2026
Venue: Hougoumont Hotel, Fremantle, Western Australia
Cost: AUD $3,300.00 (inclusive of GST) for two weeks

Successful completion of this course will allow the doctor to perform Recreational and Occupational (as per AS/ NZS 2299.1) fitness for diving medicals and be listed for such on the SPUMS Diving Doctors List (provided that they continue to be a financial SPUMS member).

The course content includes:

- History of diving medicine and hyperbaric oxygen treatment
- Physics and physiology of diving and compressed gases
- Presentation, diagnosis and management of diving injuries
- Assessment of fitness to dive
- Visit to RFDS base for flying and diving workshop
- Accepted indications for hyperbaric oxygen treatment
- Hyperbaric oxygen evidence based medicine
- Wound management and transcutaneous oximetry
- In water rescue and management of a seriously ill diver
- Visit to HMAS Stirling
- Practical workshops
- Marine Envenomation

Contact for information:
Sam Swale, Course Administrator
Phone:*+61-(0)8-6152-5222*
Fax:*+61-(0)8-6152-4943*
Email: *fsh.hyperbaric@health.wa.gov.au*
Accommodation information can be provided on request.

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.

Royal Australian Navy Medical Officers' Underwater Medicine Course

Dates: 9–20 March 2026, 19–30 October 2026
8–19 March 2027, 11–22 October 2027

Venue: HMAS Penguin, Sydney

The MOUM course seeks to provide the medical practitioner with an understanding of the range of potential medical problems faced by divers. Emphasis is placed on the contraindications to diving and the diving medical assessment, together with the pathophysiology, diagnosis and management of common diving-related illnesses. The course includes scenario-based simulation focusing on the management of diving emergencies and workshops covering the key components of the diving medical.

Cost: The course cost is AUD $2,332.00 (excl GST) but is subject to change.

Successful completion of this course will allow the doctor to perform Recreational and Occupational (as per AS/ NZS 2299.1) fitness for diving medicals and be listed for such on the SPUMS Diving Doctors List (provided that they continue to be a financial SPUMS member).

For information and application forms contact:
Rajeev Karekar, for Officer in Charge
Submarine and Underwater Medicine Unit
HMAS Penguin
Middle Head Rd, Mosman
NSW 2088, Australia
Phone: *+61 (0)2-9494-7292*
Email: *rajeev.karekar@defence.gov.au*

SPUMS Facebook page
Find us at:
SPUMS on Facebook

SPUMS

South Pacific Underwater Medicine Society

54TH ANNUAL SCIENTIFIC MEETING

Palasia Hotel, Palau

10–15 May 2026

Theme: "Free diving"

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

Courses and meetings

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/

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

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

The Science of Diving

Support EUBS by buying the PHYPODE book '*The science of diving*'. Written for anyone with an interest in the latest research in diving physiology and pathology. The royalties from this book are being donated to the EUBS.
Available from:
Morebooks
https://www.morebooks.de/store/gb/book/the-science-of-diving/isbn/978-3-659-66233-1

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DHM Journal Facebook

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

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 33 years, attended by more than 5700 medical doctors (MD).

In 2026 the Scott Haldane Foundation (SHF) celebrates its 50th anniversary. Of course SHF will, during its jubilee year, continue to organize courses and events for MD's interested diving and hyperbaric medicine.

Currently the program is still preliminary. As soon as the dates and locations are fixed, the schedule will be published on www.scotthaldane.org.

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).

Back issues of DHM

After a one-year embargo, individual articles from *Diving and Hyperbaric Medicine* are freely available on our website https://www.dhmjournal.com/index.php/full-journals-embargoed/full-journals

They are also available on PubMed Central as full articles after one year embargo dating back to 2017. These are searchable via their doi, PMID or PMCID number.

Embargoed articles are available via the DHM website for single use purchase.
Please follow the link below if you would like more information:
https://www.dhmjournal.com/index.php/purchase-single-articles

or email Nicky Telles our Editorial Manager:
editorialassist@dhmjournal.com

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
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***Editorial Manager*:** editorialassist@dhmjournal.com
***Journal information*:** info@dhmjournal.com

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

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

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

Original articles, Technical reports and Case series: up to 3,000 words is preferred, and no more than 30 references (excluded from word count). Longer articles may be considered at the editor's discretion. These articles should be subdivided into the following sections: an **Abstract** (subdivided into Introduction, Methods, Results and Conclusions) of no more than 250 words (excluded from word count), **Introduction, Methods, Results, Discussion, Conclusions, References, Acknowledgements**, **Funding** sources and any **Conflicts of interest**. **Legends/captions** for illustrations, figures and tables should be placed at the end of the text file.

Review articles: up to 5,000 words is preferred and a maximum of 50 references (excluded from the word count); include an informative **Abstract** of no more than 300 words (excluded from the total word count); structure of the article and abstract is at the author(s)' discretion.

Case reports, Short communications and **Work in progress reports:** maximum 1,500 words, and 20 references (excluded from the word count); include an informative **Abstract** (structure at author's discretion) of no more than 200 words (excluded from the word count).

Educational articles, Commentaries and **Consensus reports** for occasional sections may vary in format and length but should generally be a maximum of 2,000 words and 15 references (excluded from word count); include an informative **Abstract** of no more than 200 words (excluded from word count).

Letters to the Editor: maximum 600 words, plus one figure or table and five references.

The journal occasionally runs **'World as it is'** articles; a category into which articles of general interest, perhaps to divers rather than (or in addition to) physicians or scientists, may fall. This is particularly so if the article reports an investigation that is semi-scientific; that is, based on methodology that would not necessarily justify publication as an original study. Such articles should follow the length and reference count recommendations for an original article. The structure of such articles is flexible. The submission of an abstract is encouraged.

Supplements to a particular issue are occasionally published for purposes deemed appropriate by the editor. These may accommodate articles / treatises that are too long for the main journal or collections of articles on thematic areas. There is no open portal for submission of such material and any plans or suggestions for supplements should be discussed with the Editor before writing.

Formatting of manuscripts: All submissions must comply with the following requirements. **Manuscripts not complying with these instructions will be suspended** and returned to the author for correction before consideration. Guidance on structure for the different types of articles is given in the full version of these instructions.

Documents on DHM website https://www.dhmjournal.com/index.php/author-instructions

The following pdf files are available on the DHM website to assist authors in preparing their submission:

Instructions for authors (full version 2024 – this document)
DHM Keywords 2023
DHM Mandatory submission form 2024
Trial design analysis and presentation
Conflict of interest statement
English as a second language
Guideline to authorship in DHM 2015
Samples of formatted references for authors of journal articles (last reviewed 2024)
Recommendations for the conduct, reporting, editing, and publication of scholarly work in medical journals 2024
Helsinki Declaration revised 2013
Is ethics approval needed?

IN THE EVENT OF A LIFE THREATENING EMERGENCY PLEASE CALL YOUR LOCAL EMERGENCY SERVICES FIRST

For an accident in Australia, call the nearest public hospital with a Hyperbaric Unit and ask for the Duty Hyperbaric Doctor – see list below:

New South Wales/ACT **(02) 9382 2222** (Prince of Wales Hospital)
Northern Territory **(08) 8922 8888** (Royal Darwin Hospital)
Queensland **(07) 3646 8111** (Royal Brisbane Hospital) **(07) 4433 1111** (Townsville Hospital)
South Australia **(08) 7074 0000** (Royal Adelaide Hospital)
Tasmania **(03) 6166 8308** (Royal Hobart Hospital)
Victoria **(03) 9076 2000** (The Alfred)
Western Australia **(08) 6152 2222** (Fiona Stanley Hospital)

If you have a diver emergency **OUTSIDE AUSTRALIA**, please use one of the contact numbers below:

New Zealand from within New Zealand:
0800-4DES 111
(Diving Emergency Service)
New Zealand from overseas:
+64 9 445 8454
Asia, Pacific Islands **+618-8212 9242** (DAN World)
Americas **+1-919-684 9111** (DAN)
Europe **+39-06-4211 8685** (DAN EUROPE)
Southern Africa **+27-10-209 8112** (DAN SOUTHERN AFRICA)

Scholarships for Diving Medical Training for Doctors

The Australasian Diving Safety Foundation is proud to offer a series of annual Diving Medical Training scholarships. We are offering these scholarships to qualified medical doctors to increase their knowledge of diving medicine by participating in an approved diving medicine training programme. These scholarships are mainly available to doctors who reside in Australia. However, exceptions may be considered for regional overseas residents, especially in places frequented by Australian divers. The awarding of such a scholarship will be at the sole discretion of the ADSF. It will be based on a variety of criteria such as the location of the applicant, their working environment, financial need and the perception of where and how the training would likely be utilised to reduce diving morbidity and mortality. Each scholarship is to the value of AUD $5,000.00.

ADSF
AUSTRALASIAN DIVING SAFETY FOUNDATION

There are two categories of scholarships:

1. ADSF scholarships for any approved diving medical training program such as the annual ANZHMG course at Fiona Stanley Hospital in Perth, Western Australia.
2. The Carl Edmonds Memorial Diving Medicine Scholarship specifically for training at the Royal Australian Navy Medical Officers' Underwater Medicine Course, HMAS Penguin, Sydney, Australia.

Interested persons should first enrol in the chosen course, then complete the relevant ADSF Scholarship application form available at: https://www.adsf.org.au/r/diving-medical-training-scholarships and send it by email to John Lippmann at johnl@adsf.org.au.
