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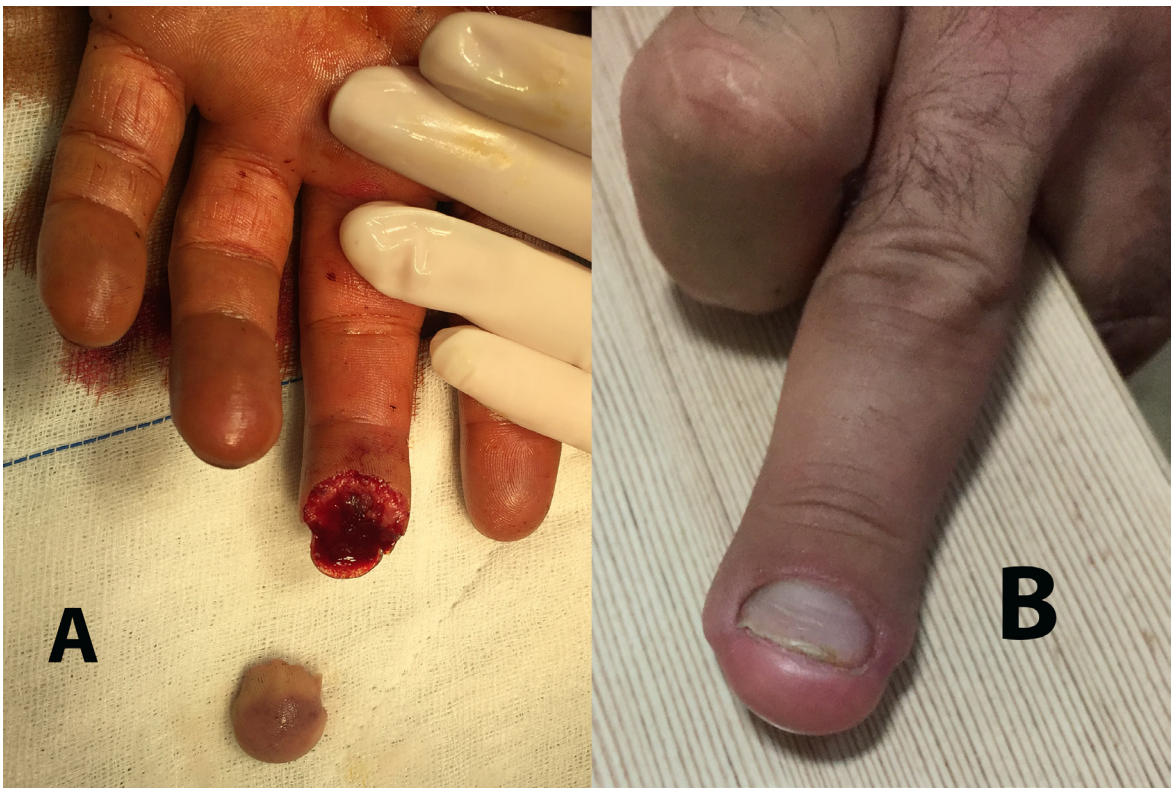

HBOT in Tamai zone 1 replantation

DAN emergency calls involving children

Oxygen saturation in repeated freedives

Longitudinal variability in Eustachian tube function

Decompression illness in breath-hold divers

Intravenous pumps in hyperbaric chambers

Validation of a hyperbaric sham treatment

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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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DIVING AND HYPERBARIC MEDICINE

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Editor's offering

We will probably remember 2023 as the first year that the world truly began to open up after the COVID-19 pandemic. There is much evidence of this in my own activities with many exciting meetings and events planned. I have just returned from a diving expedition, delayed by several years, to the Pearse Resurgence cave here in New Zealand where Richard Harris and Craig Challen performed the first deep dive (230 m) using hydrogen as a breathing gas for over 30 years. Next month I head to Malta for the Rebreather Forum 4 meeting, and both of the journal's parent societies will hold face to face meetings this year: SPUMS in Cairns, Australia; and EUBS in Porto, Portugal. There's also the UHMS meeting in San Diego, AHDMA in Kota Kinabalu, and the Diving Talks meeting in Portugal. Exciting times.

This first issue of DHM in 2023 contains a balance of articles pertaining to diving and hyperbaric medicine.

Yavuz Tuluy and colleagues from Turkey report a series of patients with Tamai zone 1 digital amputations who were treated with hyperbaric oxygen as an adjunct to microsurgery. This was associated with a high proportion of successful outcomes and importantly, the protocol incorporating hyperbaric oxygen allowed a shortened hospital stay for these patients. I'm sure members of our societies will join with me in remembering the terrible earthquake in Turkey earlier this year which resulted in catastrophic loss of life, and in acknowledging our Turkish colleagues in their roles in treating countless patients with traumatic injuries as a result.

Elizabeth Helfrich and colleagues from DAN America report a fascinating review of emergency call records involving children in diving accidents. Although most of the adult callers were concerned that the child involved might have been suffering decompression sickness, most calls with diagnoses that actually proved diving-related pertained to ear or sinus problems, and pulmonary barotrauma was more common than decompression sickness. Anxiety and panic were features of many cases.

Eric Mulder and colleagues report an important study of rate of arterial oxygen desaturation (measured using peripheral oximetry) across repetitive freedives. Despite the dives being separated by recommended surface intervals there was an increasing rate of desaturation across the three dives performed which the authors speculatively attribute to oxygen debt that is not 'repaid' during the time between dives. This paper has important implications for freedivers indulging in repetitive dive activities like spearfishing, and opens up some great opportunities for further work to better characterise optimal surface intervals.

Nele Peters and colleagues report an innovative methodology to measure Eustachian tube function over time (three one-week intervals in this study). They showed low week-to-week intraindividual variability in function. Not only is this paper of interest to diving physicians, but it is also likely of interest from a methodologic perspective within the wider field of otology.

Lesley Blogg and colleagues present a systematic review of reports of decompression illness (DCI) in breath-hold divers. It is important in reading to their paper to appreciate that DCI is a collective term which embraces both decompression sickness (DCS) caused by bubble formation from dissolved gas and arterial gas embolism (AGE), the latter due to introduction of bubbles to the arterial circulation by pulmonary barotrauma.[1] Both potentially present with symptoms of cerebral involvement and can be difficult to tell apart (which was the principal original reason for proposing the collective 'DCI terminology'). Even though both pathologies should be uncommon in the absence of breathing compressed gas, it seems both can occur in breath-hold diving though DCS is more common.

Aisha Al Balushi and David Smart present a review of the literature pertaining to safety and performance of intravenous infusion devices in hyperbaric environments. This is clearly an important topic in relation to managing patients requiring intensive care during hyperbaric oxygen treatment. They found that although infusion accuracy is typically well documented, safety evaluations are rarely compliant with recommended guidelines.

Finally, Pierre Louge and colleagues report on a randomised blinded study that aimed to determine a minimum compression pressure and other additional blinding strategies that resulted in reliable blinding of subjects to sham (low pressure) versus typical hyperbaric treatment pressure exposures. Their results will no doubt rekindle the debate over whether a very low-pressure air exposure is truly an inert sham, but those claim it's not are yet to explain why a hyperbaric treatment would be necessary if the desired clinical benefit can be obtained with an inspired PO_2 that can be achieved using supplemental oxygen at atmospheric pressure.

Reference

1 Mitchell SJ. DCS or DCI? The difference and why it matters. Diving Hyperb Med. 2019;49:152–3. doi: 10.28920/dhm49.3.152-153. PMID: 31523788. PMCID: PMC6881199.

Prof Simon Mitchell
Editor, Diving and Hyperbaric Medicine Journal

Cover photo: Finger tip (Tamai zone 1) amputation and successful reimplantation involving treatment with hyperbaric oxygen (from Tuluy, et al. in this issue).

Original articles

Effects of external bleeding and hyperbaric oxygen treatment on Tamai zone 1 replantation

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Keywords
Finger; Microsurgery; Reperfusion; Surgery

Abstract
(Tuluy Y, Aksoy A, Sir E. Effects of external bleeding and hyperbaric oxygen treatment on Tamai zone 1 replantation. Diving and Hyperbaric Medicine. 2023 March 31;53(1):2–6. doi: 10.28920/dhm53.1.2-6. PMID: 36966516.)
Introduction: Tamai zone 1 replantation poses a challenge due to the very small size of the vascular structures; often there is no vein for anastomosis. Replantation may have to be done with only an arterial anastomosis. In our study, we aimed to evaluate the success of replantation by combining external bleeding and hyperbaric oxygen treatment (HBOT) in Tamai zone 1 replantation.
Methods: Between January 2017 and October 2021, 17 finger replantation patients who underwent artery-only anastomosis due to Tamai zone 1 amputation received 20 sessions of HBOT with external bleeding after the 24th postoperative hour. Finger viability was assessed at the end of treatment. A retrospective review of outcomes was performed.
Results: Seventeen clean-cut finger amputation patients were operated on under digital block anaesthesia with a finger tourniquet. No blood transfusion was required. In one patient, complete necrosis developed and stump closure was performed. Partial necrosis was observed in three patients and healed secondarily. Replantation in the remaining patients was successful.
Conclusions: Vein anastomosis is not always possible in fingertip replantation. In Tamai zone 1 replantation with artery-only anastomosis, post-operative HBOT with induced external bleeding appeared to shorten the hospital stay and was associated with a high proportion of successful outcomes.

Introduction

After the successful digital artery repair in 1965 and the first successful total amputated thumb replantation in 1968, fingertip replantation began to be performed with the development of microsurgical techniques and instruments.[1,2] Replantation is the gold standard in finger amputations. The best aesthetic and functional results can be achieved with successful replantation. Fingertip injuries pose a challenge due to the very small size of the vascular structures and often there is no vein for anastomosis. Tamai zone 1 replantation refers to the replantation level distal to the base of the nail, and artery-only replantation is predominantly performed.[3] Successful artery-only replantations were reported in 1972 and 1973.[1,4] Numerous techniques have been described to provide venous flow in patients undergoing artery-only replantation. These techniques use medical or mechanical leeches, partial or total nail bed removal, and/or fish-mouth incision.[5] With these techniques, however, the hospital stay of the patients is prolonged.[6–8] Improved approaches would shorten recovery and accelerate the return of the patients to work.

After artery-only replantation, ischaemia-reperfusion (IR) injury is inevitable. This can cause tissue microcirculation insufficiency and necrosis after prolonged ischaemia, which adversely affects the viability of the amputated finger and may lead to partial or total necrosis.[9] In addition to the damage caused by ischaemia in the amputated tissue, tissue damage can continue after reperfusion. Tissue microcirculation insufficiency and necrosis due to reperfusion after prolonged ischaemia is defined as 'IR injury'. In Tamai zone 1 replantation, both IR injury and inability to perform venous anastomosis negatively affect the success of replantation. Hyperbaric oxygen treatment (HBOT) has been used in replantation of the extremities and has been found to be effective.[9,10] Therefore, in our study, we aimed to evaluate the success of replantation by combining external bleeding and HBOT in Tamai zone 1 replantation.

Methods

Senior hospital management have confirmed that a retrospective case series of this nature is out of scope for

ethics committee review in this jurisdiction. All patients gave permission for their data to be reported.

Records were reviewed for patients who underwent finger replantation with artery only anastomosis after Tamai zone 1 amputation and who received post-operative HBOT. Between January 2017 and October 2021, 17 finger replantation patients were managed in this way. Patients who underwent multiple finger replantation, had diabetes, additional trauma, and did not complete HBOT sessions were excluded from the review. Since venous anastomosis could not be performed, external bleeding was performed with an incision made from the pulp. Patients received HBOT on the 1st postoperative day since the surgery time and recovery after anesthesia were long. Patients were discharged on the first postoperative day but returned for daily HBOT and induced external bleeding. Hyperbaric oxygen treatment (100% oxygen at 243 kPa [2.4 atmospheres absolute] for 90 minutes) was administered over 20 sessions. The demographic characteristics, amputated finger and complication data of the patients were noted. This was not a prospective study and there was no control group. To mitigate this limitation we compared outcomes with case series in the literature in which artery-only anastomosis was performed and HBOT was not administered. All patients reviewed received 20 sessions of HBOT and there were no patients with missing sessions.

SURGICAL PROCEDURE

The amputated finger was wrapped with saline-soaked gauze and kept in appropriately cool conditions (approximately +4°C) until the patient was taken into surgery. Replantation was performed under digital block anaesthesia. After a finger tourniquet was applied, the wound was evaluated under loupe magnification. Adequate debridement was performed, and blood vessels were dissected. The tourniquet was then released, and the blood flow of the digital arteries was checked. The amputated finger was prepared for anastomosis. Bony fixation was performed by using one or two Kirschner wires in all patients. These were removed in all patients at postoperative 4th week after evaluation by hand radiography. Tendon repair was not required, because all the injuries were Tamai zone 1 amputation. Central digital artery and digital nerve anastomosis were performed with 11-0 nylon sutures. The skin edges were loosely sutured to allow bleeding and prevent venous congestion. During the operation, 5,000 IU heparin was given and 100 mg acetylsalicylic acid was given in the follow-ups. Oral antibiotics were given to the patients for one week in the postoperative period.

Table 1
Numbers of patients by affected finger and hand

Hand / finger	2nd	3rd	4th	5th
Right hand	2	4	3	1
Left hand	1	3	2	1

Results

Seventeen clean-cut finger amputation patients were operated on under digital block anesthesia with a finger tourniquet. The numbers of patients by digit and hand are reported in Table 1. Eleven patients were male and six were female. The mean age was 31.2 (range 19–46) years. No blood transfusion was needed, bleeding was controlled after each application of HBOT. The estimated amount of bleeding was 200–400 ml, with an average of 270 ml. At the end of 20 sessions, the patients were examined and subsequently followed up for an average of 21 months (range 8–33). In one patient, complete necrosis developed and stump closure was performed. Partial necrosis was observed in three patients which healed secondarily. All other patients had successful replantation examples of which are shown in Figures 1 and 2. No soft tissue infection or osteomyelitis was

Figure 1
Left hand, second finger Tamai zone 1 amputation (A) and the same patient at the fourth postoperative month (B)

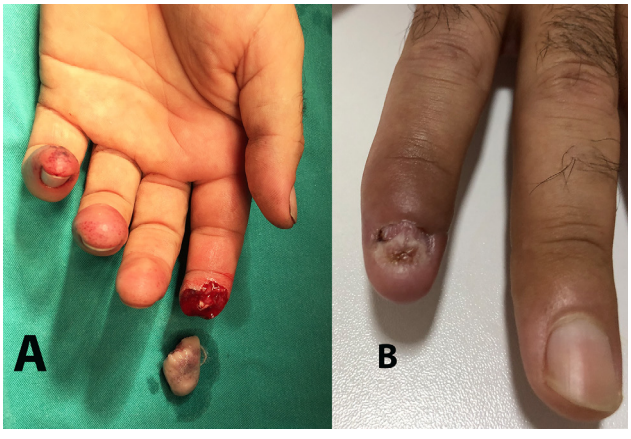
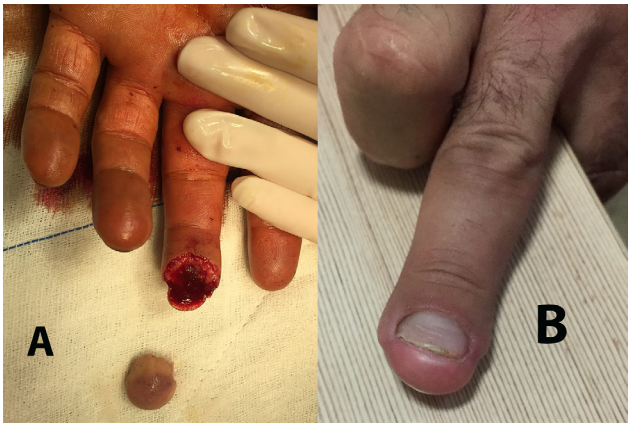


Figure 2
Left hand, third finger Tamai zone 1 amputation (A) and the same patient at the eighth postoperative month (B)



observed. Since the amputation was distal from the distal interphalangeal joint, the range of motion for functional evaluation was not evaluated. Fingertip sensation began to return from the sixth month. No loss of sensation was observed in long-term follow-ups.

Discussion

In fingertip amputations, apart from replantation, reconstruction can be performed with composite grafts, and local or free flaps.[11,12] The aim in fingertip amputation surgery is to preserve the length and sensory innervation of the finger and to ensure its function. Although replantation is the best option in fingertip amputations, reconstructive surgeons may avoid replantation due to the difficulty of the technique and postoperative follow-up. Vessels are small, and the surgical field is narrow and deep.[13] Vascular repair can be performed with the dorsal or volar approach.[14]

In artery-only replantation, venous insufficiency is inevitable and venous flow must be provided with different techniques. In one study where artery-only anastomosis was performed, heparinized saline was applied topically to the gap in the Tamai zone 1 suture line. The authors reported that 93% of the replanted fingertips survived.[15] Nevertheless, they recommended performing vein anastomosis if possible. In another study in patients who underwent distal phalanges replantation, adequate vein width was obtained 8–12 hours after arterial anastomosis and vein anastomosis was performed with a second operation.[16] The disadvantage of this technique is that it is a two-stage procedure and if adequate arterial flow is not achieved in the first operation, vein anastomosis may not be achieved in the second operation.

Mechanical and medical leech therapy can also be applied. In medical leech treatment, the risk of wound infection increases and the patient or family members may have difficulty during the application.[17] In the mechanical leech technique, an angio-needle is anastomosed to the branch of the central digital artery. The catheter is removed gently after the circulation of the replanted finger is established. A chemical leech procedure can also be used. After making a 2 mm incision over the fingertip, heparin is injected subcutaneously and systemic heparin and dextran-40 are given.[7] A high success rate is obtained using this technique. Adequate bleeding is also obtained by nail plate removal. The nail plate can be removed totally or partially.[6,8,18] In these studies, the length of hospital stay was extended up to 14 days, and in our study, the patients were discharged on the first postoperative day. The shortening of the hospitalisation period has positive effects both in terms of health expenses and patient psychology.

In a study by Han et al. the average duration of neovascularization was 7.6 days in patients who underwent artery-only anastomosis and postoperative external bleeding.[19] They also observed that the duration of external bleeding was shorter in young and less injured patients.
In the anatomical study by Nam et al. the central artery was divided into three types. In type 1, only one dominant artery branched off from the distal transverse palmar arch. If the fingertip had two dominant arteries, it was classified as type 2 and if the fingertip had three and more branches, it was classified as type 3.[20] They observed that type 3 was the most common type. If there are two or more arteries one can be used for venous anastomosis. After the digital artery anastomosis, another digital artery can be used for anastomosis to the vein of the proximal stump, if it is possible. We performed digital nerve anastomosis in all cases. Since the neural network of the region is dense in fingertip replantation, adequate innervation is provided even if nerve anastomosis is not performed.[21]

Ischaemia-reperfusion injury is one of the major problems in replantation surgery. Many different mechanisms play a role in IR injury. Reactive oxygen species (ROS) such as superoxide and hydroxyl radicals are thought to be the main mediator of cell damage. These cause DNA damage, lipid peroxidation and cell membrane damage.[22] There are protective antioxidants such as glutathione, superoxide dismutase (SOD) and catalase. However, if ROS production is too high these antioxidants may be overwhelmed. Xanthine oxidase and neutrophils are sources of ROS production.[23] Reactive oxygen species trigger migration of neutrophils to the injury zone and initiate inflammation. After reperfusion, migrating neutrophils synthesize greater quantities of ROS, contributing to reperfusion injury. Apoptosis is also seen in IR injury. Nitric oxide (NO) competes with oxygen for binding to cytochrome c oxidase which has higher affinity for NO.[23] This pathway induces apoptosis. In IR injury, vasoactive substances are released from the cells and disrupt the circulation of the injured tissue.

Hyperbaric oxygen treatment increases SOD activity and antioxidant gene expression.[24,25] It may also reduce neutrophil adhesion by reducing adhesion molecule expression,[26] and increase angiogenesis by promoting vascular endothelial growth factor transcription.[27] There have been no comparative studies to determine an optimal HBOT protocol in related injuries. In one study of injured hands HBOT was given five days a week beginning 24 hours after replantation with improved survival of the compromised tissues.[9] In another study of finger replantation HBOT was started on the first postoperative day,[10] while others have started HBOT within the first 24 hours after replantation in hand injuries. We started HBOT on the first postoperative day, and utilised 20 treatments; a higher number than some studies which we nevertheless considered appropriate because no vein anastomosis was performed. Ideally, studies comparing different HBOT regimens could be conducted to derive an optimal protocol. Care should be taken about complications during HBOT. Possible complications include seizure, reversible myopia and barotrauma to ears, sinuses or lungs.[9,28]

This is the first publication describing HBOT in fingertip amputations. In our study, hospital stay was shortened after HBOT was combined with induced external bleeding, and replantation was successful in 16 (94%) of the patients. Total necrosis was observed in only one patient. In our previous study comparing patients with and without vein anastomosis in fingertip replantation, the success rate was 77.3% (41/53) in patients who did not have vein anastomosis.[29] Given the observational nature of the present report, no firm conclusions can be drawn from this comparison. However, use of HBOT seemed associated with shorter hospital stay and better graft survival in the present study. In other approaches without HBOT, it is not possible to discharge the patients on the first postoperative day and the surgeon needs to allocate more time to the patient. This time is of great importance for microsurgeons who are busy performing long and challenging surgical procedures.

We acknowledge the lack of a contemporaneous control group or randomisation, and the small cohort of patients, as limitations of the study.

Conclusions

Vein anastomosis is not always possible in fingertip replantation. In our clinical experience it appears that HBOT with induced external bleeding shortens the hospital stay and increases the success of replantation In Tamai zone 1 replantation with artery-only anastomosis.

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A review of 149 Divers Alert Network emergency call records involving diving minors

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Keywords
Arterial gas embolism; Children; Decompression illness; Pulmonary barotrauma; Scuba diving

Abstract
(Helfrich ET, Saraiva CM, Chimiak JM, Nochetto M. A review of 149 Divers Alert Network emergency call records involving diving minors. Diving and Hyperbaric Medicine. 2023 March 31;53(1):7–15. doi: 10.28920/dhm53.1.7-15. PMID: 36966517.)
Introduction: Minors have been scuba diving for decades, and while the initial concerns about potential long-term complications related to bone development appear to be unfounded, the incidence of scuba diving injuries among them has been poorly studied.
Methods: We reviewed 10,159 cases recorded in the DAN Medical Services call center database from 2014 through 2016 and identified 149 cases of injured divers younger than 18 years. Records were analysed for case categorisation on the most common dive injuries. Information about demographics, level of training, risk factors, and relevant behavioural aspects were collected when available.
Results: While the most common reason for the call was to rule out decompression sickness, the majority of cases pertained to ear and sinus issues. However, 15% of the dive-related injuries involving minors had a final diagnosis of pulmonary barotrauma (PBt). While no reliable data is available on the incidence of PBt in adult divers, the authors' impression based on personal experience suggests that the number of cases of PBt in minors trends higher than in the general diving population. The narratives on some relevant records describe unmanageable levels of anxiety leading to panic.
Conclusions: Based on the results and narratives on these cases, it is reasonable to infer that psychological immaturity, suboptimal management of adverse situations, and inadequate supervision might have led to severe injuries among these minor divers.

Introduction

Minors between 8 and 18 years old have been scuba diving for decades, but data on participation numbers and incidence of injuries are unavailable. When recreational scuba diving began in the 1950s, it was primarily reserved for relatively young, physically fit men. Over the following decades, recreational scuba expanded to include more women with both sexes of varying ages and fitness and, more recently, children. Training agencies have developed programs issuing special certifications for children as young as ten. Once the junior diver turns 15 years old, upgrading to a full certification is an administrative process. Some agencies have gone beyond, developing programs that allow participants as young as eight years old to breathe from a compressed gas source, although these usually involve only surface water activities and close supervision.

Most outdoor recreational activities involve managing inherent risks. Scuba diving requires special equipment and training to survive a hostile environment and physical and mental capacity to manage its risks. Diving can be psychologically stressful for those new to the sport or practicing it in challenging conditions. The most common cause of death in child divers is drowning, whilst the major contributor is panic.[1]

During childhood, dramatic changes happen in the brain. The prefrontal cortex and amygdala mature, giving us tools to perfect decision-making processes, regulate emotions, detect threats, and activate appropriate fear-related behaviours in response to threatening or dangerous stimuli.[2] Psychological immaturity can prevent minors from reacting to emergencies underwater with the same capacity as adults.[3] Panic can lead to uncontrolled rapid ascents, increasing the risk of pulmonary barotrauma.[4] Alternatively, children can lose focus within routine dives and make mistakes such as holding their breath or losing buoyancy control, similarly leading to an increased probability of injury.[5] Scuba diving requires a specific set of skills and physical coordination that may be poorly developed in minors. Demonstration of these skills in a highly controlled environment such as a swimming pool may not readily transfer to the open water environment.[6]

There have been concerns about the potentially harmful effects of compressed gas diving on growth rates. Epiphyseal growth plates have an increased blood supply, and there

was concern that under these unique conditions, scuba diving could impose a higher inert gas load and higher decompression stress than on most other body compartments, potentially impairing long-bone development. However, after decades of extensive diving by minors, including long-term follow-ups on cases of the bends, there does not seem to be any evidence to support this theory.[7,8]

Patent foramen ovale (PFO) is more common in children and can be found in up to 36% of individuals.[9] However, the incidence of decompression sickness does not seem to be higher than adults, possibly due to the depth restrictions commonly imposed on young divers.

Asthma with associated bronchoconstriction, air trapping phenomena, and reduced exercise tolerance is frequent in children. Its prevalence diminishes with age, demonstrating that the respiratory system is often still developing until teenagers become young adults. A child breathing from a compressed gas source in a swimming pool only two metres deep may not be at risk of decompression sickness (DCS), but is certainly at risk of pulmonary barotrauma (PBt) and arterial gas embolism (AGE).[10]

The Eustachian tube is not fully developed until approximately 12–13 years of age. The shorter and horizontalised Eustachian tubes, and often hypertrophic adenoids, tend to hinder normal middle ear ventilation, possibly making them prone to middle ear infections. These characteristics expose children to a higher risk of otic barotrauma.[11–13] Adults often experience difficulty with the concept of ear equalisation techniques, and instructors and parents must be convinced the minor comprehends the importance of this skill and is physically capable of performing these techniques effectively and efficiently without hurting themselves.

Temperature loss is higher in children due to the higher body surface area to mass ratio. Poor fit of neoprene wetsuits can increase conductive and convective heat loss, increasing the risk of hypothermia and forcing a high metabolic compensation to mitigate the energy consumption.

The World Recreational Scuba Training Council (WRSTC), a self-governing body with the primary goal of developing minimum standards for training recreational diving worldwide, has determined that a minor may not be deemed fully certified as an autonomous entry-level diver until age 15.[14] The council, however, does not define the minimum criteria to enrol a candidate in training. The council's standards also state that "*students under the minimum age may qualify for a special certification that allows them to dive under the supervision of an adult who has, as a minimum, an entry-level scuba certification*."[14]

While the incidence of diving injuries among recreational divers has been the focus of many studies, very few investigations focus on diving injuries of minors.[15,16] One retrospective study described 22 dive accidents in minors who were treated for AGE (six cases) and DCS (16 cases).[17]

Our study represents a retrospective analysis of diving injuries involving minors assisted through the emergency line of DAN's Medical Services Call Centre (MSCC) and recorded in a database between January 2014 and December 2016. The MSCC is open to all divers in need and professionals managing diving injuries. In general, more than half of users are non-DAN members and thus MSCC data provides a snapshot of injuries in the entire population of active recreational scuba divers.

Methods

The study was approved by the Institutional Review Board at the Divers Alert Network (Approval 020-15020).

This was a retrospective analysis of records stored in MSCC database, in the period from 2014 to 2016 inclusive.

SOURCE OF DATA

MSCC digital records contain case notes of incidents reported to DAN. Some records contain audio files and documents shared by the calling party, like medical notes, diagnostic studies, images, and dive profile logs.

A record usually starts as a transcribed narrative of the first interaction between a caller to the DAN Hotline and the DAN hotline agent (a diver medic, a nurse, or a physician) on call who interviewed the caller. The agent leads the call, gathering the minimally necessary information before recommending the best course of action.

The caller is interrogated about the reason for the call (usually documented as a chief complaint), the events leading to the injury or incident, and the subjective description of symptoms. Recommendations are offered once the hotline agent has enough information to have a reasonably good idea of what might be going on. After the initial contact, DAN Medics remain engaged with the case as it progresses through all phases: during case management while the diver is in the field, offering expert consultation once the injured diver is admitted to a medical facility, and monitoring the patient's progress with frequent follow-up calls after the diver is discharged. The result is usually a reasonably good recollection of events and outcomes.

IDENTIFIYNG CASES INVOLVING MINORS

In the observed period, there were 10,159 emergency case records involving divers of all ages. The injured diver's age or date of birth was not being explicitly recorded or provided by callers. Thus, we used a query written in Transact Structured Query Language (T-SQL) SQL Server Management Studio (SSMS) to search through four text

fields (chief complaint, history of present illness, initial assessment, recommendations) for the keywords suggesting the victim was a minor (e.g., identifiers for ages < 18 years old, child, daughter, son, boy, girl, etc.).

After identifying 2,269 cases, further review rejected 2,020 false-positive cases not involving minors. Out of the remaining 249 cases, 35 were not diving-related. Of the remaining 214 cases, only 106 cases had explicitly stated the diver's age; these cases were confirmed to fit the study criteria. One hundred and eight cases were ambiguous and required further investigation.

For an ambiguous case to be included, there had to be an abundance of indicators that the case referred to involved a minor. Some of the indicators of a minor were a parent calling on behalf of their child, the injured diver being a member of Boy Scouts, or enrolment in high school. Exclusion information for potential adults included enrolment in college or university, completing a divemaster course, or being in military service. With further review, 65 records were excluded as the divers were positively identified as older than 18 years of age at the time of the incident. From the remaining 43 records, 34 divers were positively identified as minors with a confirmed age, and nine divers were confirmed minors at the time of the incident but did not have confirmed ages. All 43 cases, in addition to the previous 106 cases, were included in the 149 cases in this study.

CASE CATEGORISATION

The included cases were classified using a protocol with inclusion criteria for each category. The categories, based on the most common dive injuries, were as follows: arterial gas embolism (AGE); anxiety; decompression sickness (DCS); ear, nose and throat injuries (ENT); hazardous marine life injury (HMLI); immersion pulmonary oedema (IPO); musculoskeletal; other; pulmonary barotrauma (PBt); caller 'uncertain'; unrelated; and unrelated infectious gastroenteritis. Each case was categorised twice, once for 'reason for call' and once for 'final diagnosis category'. The reason for call was determined from the caller's concerns or chief complaint (see Table 1). The final diagnosis category used for this study was determined by a senior physician at DAN after reviewing all case notes (see Table 2). When available, a treating physician diagnosis (TMD) was cross-matched with DAN's final diagnosis category.

SUBSAMPLE VALIDATION

The final sample of cases was validated with subsample validation. Three hundred and fifty cases were randomly selected from the original 2,269 cases and independently verified by a second reviewer with 100% match for both confirmed and suspected minors.

The categorisation of 'reason for call' and 'final diagnosis category' was also confirmed via subsample validation

Table 1

Reason for call classification descriptions; AGE – arterial gas embolism; DCS – decompression sickness; HMLI – hazardous marine life injury

Reason for call	Description
Arterial gas embolism	Caller suspected AGE. Chief complaint was acute onset of focal neurological deficit, severe headaches associated with vomiting and seeking hyperbaric treatment options.
Anxiety	Caller's chief complaint was minor's anxiety.
Decompression sickness	Caller suspected DCS. Chief complaint related to joint pains, fatigue, decompression injuries.
Ear nose and throat	Complaints related to ears and sinuses including problems equalising and headaches.
HMLI	Injury from hazardous marine life.
Other dive related	Non-emergent dive-related injury including 'fin foot', trauma, and eye irritation.
Respiratory	Complaints related to respiratory system including shortness of breath, difficulty breathing, non-cardiac chest pain, dyspnoea, coughing, suspected pulmonary barotrauma including pneumothorax, immersion pulmonary oedema.
Uncertain	Caller suspected something was wrong with child but was 'uncertain' regarding cause. Caller was seeking any connection between child's complaints and diving.

Table 2
Final diagnosis classification descriptions; DCS – decompression sickness; HMLI – hazardous marine life injury

Final diagnosis category	Description
Arterial gas embolism	Confirmed focal neurological deficit in direct association with a dive exposure. Unlikely if onset greater than 15 minutes after surfacing.
Anxiety	Treating physician diagnosed minor with anxiety or anxiety-related issue; no other injuries.
Decompression sickness	Signs and symptoms highly compatible with DCS in association with a moderate to significant dive exposure. No other compelling explanation for symptoms. Recompression therapy, normobaric oxygen, or simply time (mild/marginal cases) resolved the case. Unlikely if symptom onset more than 6 hours after surfacing. Very unlikely if symptom onset more than 24 hours after surfacing.
Ear nose and throat	Signs and symptoms compatible with ear or sinus barotrauma, and/or ear infection (troubles equalising, dizziness, nausea, vomiting, headaches, pain in sinus regions, or as diagnosed by a physician).
HMLI	Injury reported as being caused by direct or indirect contact with hazardous marine life species.
Musculoskeletal	Musculoskeletal ailments not compatible with DCS. Symptoms can often be explained by recent, normal movements associated with diving such as carrying a tank.
Other, dive related	Non-emergent dive-related injuries. Included fin foot, dehydration, trauma, exhaustion, suit squeeze, contact dermatitis, unknown rash (non-HMLI and non-DCS), eye irritation, and swallowed water.
Pulmonary barotrauma	Signs and symptoms compatible with a form of extra-alveolar air (pneumothorax, pneumomediastinum, subcutaneous emphysema).
Non-diving related	Signs and symptoms, or symptom latency is incompatible with a diving injury; or examining physician ruled out a diving injury. Patient had unrelated illness or infectious gastroenteritis that is not a result of diving.

with a second independent reviewer, with 100% match of categorisation following criteria in Tables 1 and 2.

Results

Among 10,152 cases in the database for the observed period, we positively identified 149 records (1.5%) involving minors with a suspected diving injury, 100 of which were finally diagnosed with dive-related injuries.

A concern about DCS was the primary reason for calls involving minors, accounting for 38% of all calls, followed by ENT-related complaints (26%). Pulmonary barotrauma was suspected in 12 cases (8%), and AGE was suspected in six cases (4%). However, the final diagnosis was more often ENT-related with 32% of all injuries, 15% musculoskeletal issues, 12% gastrointestinal issues, 9% PBt without AGE, 1% PBt with AGE, and 6% DCS, with other diving and non-dive related cases accounting for 25% of all calls (see Table 3).

DECOMPRESSION SICKNESS

Decompression sickness was indicated as a reason for a call in 56 cases. However, the diagnosis was confirmed in only nine, representing 16% of suspected DCS cases, 6% of all calls, and 9% of all diving injuries. Based on manifestations, four cases were neurological DCS, four were mild DCS (three with musculoskeletal pain and one with rash), and one case was inner ear decompression sickness (IEDCS). Only one minor diagnosed with DCS reported having decompression obligations during the dive.

Of the remaining 47 cases initially suspected to be DCS, the final diagnosis was musculoskeletal issues in 22 cases; gastrointestinal issues in 12 cases; four cases of ENT barotrauma; one case of PBt, one anxiety, and one HMLI. Five cases were classified as 'other, dive related', two being dehydration, one physical exhaustion, one case of contact dermatitis, one fin-foot, and one suit squeeze.

EAR AND SINUS BAROTRAUMA

Similarly to adults, ENT issues were minors' most common diving-related injuries (n = 47, 32%). Ear nose and throat injuries were suspected early on 39 calls, and in all those cases, ENT injuries were confirmed, validating the general assumption that ENT barotraumas can usually be self-diagnosed by laypeople. Of the remaining eight cases, ENT issues were not initially suspected. Four called for concerns

Table 3

Reason for call vs final diagnosis category; AGE – arterial gas embolism; DCS – decompression sickness; ENT – ear, nose and throat; GI – gastrointestinal issues; HMLI – hazardous marine life injury; IPO – immersion pulmonary oedema; PBt – pulmonary barotrauma

Condition	Reason for call		Final diagnosis		
	Cases (*n*)	% of all calls	Cases (*n*)	% of all calls	% of diving diagnoses
DCS	56	38%	9	6%	9%
Respiratory (unspecified)	12	8%	–	–	–
PBt (without AGE)	–	–	13	9%	13%
PBt and AGE	6	4%	2	1%	2%
Anxiety	1	1%	3	2%	3%
ENT	39	26%	47	32%	47%
HMLI	12	8%	12	8%	12%
IPO	1	1%	0	0%	0%
Other dive related	5	3%	14	9%	14%
Caller uncertain	17	11%	–	–	–
Sub-total diving	149	100%	100	68%	100%
GI issues	–		18	12%	–
Musculoskeletal	–		23	15%	–
Other non-diving	–		8	5%	–
Sub-total non-diving	**–**		**49**	**32%**	**–**
Total:	**–**		**149**	**100%**	**–**

about DCS, two called suspecting AGE, one called for PBt, and one caller was 'uncertain' about what was going on with the minor but knew there was something wrong. Eleven minors with ENT injuries were relatively inexperienced divers, 10 being entry-level students.

PULMONARY BAROTRAUMA AND ARTERIAL GAS EMBOLISM

Fifteen minors were diagnosed with PBt, 13 without neurological findings, and two exhibited signs compatible with AGE. Concerns about PBt was a reason for the call on 12 occasions, and among those the diagnosis was confirmed in eight instances. Of the remaining four, two were diagnosed with non-dive-related issues, one with an ENT barotrauma and one with a musculoskeletal ailment. Of those finally diagnosed with PBt, only one caller was initially concerned about AGE with PBt being the culprit. Three callers did not seem to have any red flags about the type of injury possibly sustained, and one called for concerns about DCS.

Possibly contributing factors associated with PBT were identified in 11 cases (73%); there was insufficient evidence to determine a cause of PBt in the remaining four cases (27%). In seven cases (64%), there were confirmed reports of rapid ascents; of these seven cases, six (86%) had rapid ascents due to confirmed or highly suspected anxiety. One child became anxious after practicing a controlled emergency swimming ascent (CESA) during training; another reported an anxiety attack underwater that led to breath-hold and a rapid ascent. A child freediver planned a dive to 15 feet (4.6 m), then extended to 35 feet (10.7 m) for unknown reasons. This child then had 'seizure-like' activity underwater, right leg weakness upon surfacing, and a final diagnosis of AGE from the treating physician. It is unreported if the child breathed from compressed air at depth, although likely given the symptomology and treating physician diagnosis. Three more minors likely became anxious at depth, leading to rapid, unplanned ascents and consequent PBt. The final case with rapid ascent did not confirm or deny any anxiety from the child, although both the child and their dive buddy were diagnosed with PBt by their physician.

Of the remaining four instances of PBt (36%), an event happened at depth that likely led to accidental breath-hold and PBt. Two of these cases (50%) were caused by issues with equipment; one child reported a free-flowing regulator, while another reported being overweighted. It is likely this diver attempted to assist ascent by increasing lung volumes with deep inspiration and breath-holding. Of the other two cases, one diver had an 'enormous belch' during ascent, which suggested considerable aerophagia. In the final case, the minor stated they simply laughed 'uncontrollably' underwater. Also of interest is two young divers in this cohort (13%) who noticed chest pain after the first dive but continued to dive for the day. It is unclear whether that might have contributed to the severity of the initial injury.

Concerns have been raised about weight belts slipping off and causing uncontrolled ascents,[2] but incidents of this nature were not seen in this group. Regarding their level of training, five of the injured children were students completing a junior open water diver or open water diver program. Level of training was not available in the remaining cases.

ANXIETY

Anxiety seems to have played a significant role as the trigger in at least one third of the cases of PBt, but anxiety was also the final diagnosis in three minors (2%) with post-dive symptoms. Only one caller considered anxiety as the potential culprit for the child's manifestations. In this case, the treating physician considered anxiety the most likely cause of symptoms. Still, there was a discrepancy between treating physicians and DAN regarding whether recompression and hyperbaric oxygen treatment (HBOT) would be recommended as a precautionary approach. In another case, the Coast Guard evacuated a minor for suspected DCS, but the final diagnosis was 'hyperventilation syndrome'. In the third case, the initial working diagnosis was 'possible IPO' due to an unprovoked sudden onset of coughing while at depth. However, a timely medical evaluation did not reveal any objective findings or abnormalities to substantiate this suspicion and diagnosed the case as being due to a panic attack. Later, the novice diver admitted to feeling too anxious underwater due to limited visibility and wanting to end the dive.

Discussion

The number of injured minors recorded in the MSCC represents only 1.5% of all reports. The most remarkable finding of this study is that despite having significantly more calls for suspicion of DCS, pulmonary barotrauma was more common. The low number of DCS cases is possibly the result of less provocative dives being done by minors.

Although anxiety was rarely the reason for a call or a final diagnosis, different levels of anxiety are woven throughout various case narratives. Panic is a known trigger leading to dangerous scenarios in diving.[18] A recent study suggests a major difference between minor and adult divers is developmental difference in executive function, leading to issues with response inhibition, sustained attention and cognitive flexibility.[19] This conclusion is in line with our observations; in one third of the cases of PBt, narratives describe high levels of anxiety and even panic. These minors were accompanied by an adult diver, which might have prevented an even more severe outcome.

Physiological, psychological, and behavioural differences between minors and adults support the notion that their challenges also differ. A difference in diving injuries, especially between instances of DCS and PBt, is also consistent with DAN's observations with adult callers on the DAN Hotline – a future extension on this study would compare injury incidence between adults and minors.

ASSESSMENT OF MEDICAL, PHYSICAL, AND PSYCHOLOGICAL FITNESS TO DIVE

Minor diver candidates are often referred to physicians for assessments for fitness to dive. Medical fitness to dive is well-covered on dedicated forms available online on the World Recreational Scuba Training Council's (WRSTC) website, on the Undersea and Hyperbaric Medical Society's (UHMS) Recreational Diving Medical Screening System, and by the South Pacific Underwater Medicine Society's (SPUMS) Diving Medical.[14,20,21] Physical and psychological fitness can be challenging to assess and are often left to the discretion of a clinician who may not have training in diving medicine or any diving experience. However, such assessments have limitations even when conducted by appropriately trained physicians. Provided there are no medical or physical contraindications, psychological fitness might be better gauged as a candid discussion between the physician, the candidate's legal guardians, and the scuba instructor,[22] with the candidate present if deemed appropriate.

Assuming the minor has the body mass and strength to cope with and overcome potentially adverse situations (currents, drifting, or rescue of an adult-sized diver) and has no medical contraindications, perhaps the most critical aspect of diving fitness is reviewing the candidate's psychological maturity, as this is the most important factor in accepting and managing unseen risks, and predicting behaviour in adverse circumstances. Children often have a well-developed sense of adventure and a poorly developed sense of mortality.[5] Chronological age is a poor predictor of maturity in minors. Albeit more cryptic and admittedly rather impractical, perhaps reflection on the intersection between biological, psychological, and social age could more accurately predict the physiological and psychological response of a person making use of life-support equipment to survive a hostile environment.

Remarks for clinicians

A physician assessing fitness to dive should only do so if being fully cognisant of the nature of the activity, the type of equipment to be used, the environment in which it is to take place, and the physiological and psychological effects of the underwater environment on the diver.

When assessing a minor's fitness to dive, clinicians must remember that the candidate's guardians and dive instructor might offer a valuable perspective on the candidate's psychological maturity.

Diving can impose a wide variety of challenges on those practicing it. As an aquatic recreational activity, diving can lead to musculoskeletal strain, which can be, and often is, misdiagnosed as DCS. While some risks are inherent to diving physiology, others might be related to psychological stress, practicing physical activity, or traveling (for example, fatigue, gastrointestinal issues, dehydration, among others). Divers often experience non-diving injuries during or around the diving activity and are misdiagnosed due to a recent history of diving. Conversely, dive-related injuries can often be missed by a clinician without knowledge of diving medicine. If a physician is unsure of the proper course of action, the DAN hotline is available 24/7 for consultations in diving medicine.

Remarks for the industry

Minor divers are a special population. These individuals face different challenges than adult divers and pose different challenges for dive professionals.

When training individuals in vulnerable populations, no other group generates more polarisation than young divers. Those with a favourable view are often seduced by the child's joy, lack of fear, and the rewarding feeling of witnessing the development of aquaticity in a short time. Those sceptical often base their position on mental maturity, rudimentary understanding of physics, anatomy, and physiology necessary to understand mechanisms of injury and accept risks, or simply question the physical strength of a young diver to rescue an adult in an adverse situation. Solid arguments can be made both against and in favour.

Perhaps dive professionals should have specialised training to teach young divers and lead them during open water dives. Such training should focus on their individual needs and unique behavioural aspects that seem to make them more prone to incidents and injuries. Minor divers should always be at an arms-length distance from an adult diver who needs to monitor them closely, especially with regards to comfort and air consumption. As the diver matures emotionally and their response to stress becomes more predictable, this distance could be gradually relaxed.

Safety enhancements can also be made regarding the standard operating procedures in open water dives. Recreational scuba divers are encouraged to adopt the 'buddy system' for mutual support and monitoring stress reactions in adverse circumstances. Under this arrangement, two individuals are paired and operate together as a single unit throughout the dive. They are encouraged to stay close and communicate regularly. The more they dive together, the more they know each other, and the more efficient they become. The concept has proven to work well in many disciplines, including diving. However, minor divers may not be reliable dive buddies due to their smaller body size, reduced strength, lesser maturity, and often unpredictable response to threats. WRSTC standards state that "*students under the minimum age may qualify for a special certification that allows them to dive under the supervision of an adult who has, as a minimum, an entry-level scuba certification*".[14] An argument could be made that when the team involves pairing an adult and minor where there is a significant body size or strength dissymmetry, the safety of both could be unacceptably compromised if the adult is depending on the minor for assistance in an emergency. In these cases, a team of three seems to be a more prudent minimum.

Modifying the dive plan and standard safety protocols is logical when leading dive groups containing young divers with inherent limitations. In the same way that a dive professional must hold certifications in wreck diving to teach wreck diving or to lead a group on a wreck, specific dive training should be available for dive professionals to teach and guide minor divers before working with them. This training should include an extensive explanation of minors' different challenges and how to manage them.

Instructors should be knowledgeable about the signs of anxiety and be confident in recommending to the candidate and their parents that the candidate might not be yet mentally mature enough to be a safe diver without fear of being accused of discrimination. Each minor is physically and psychologically different, and pre-existing physical, behavioural, and emotional limitations can compound the anxiety minors are more likely to experience.[23]

LIMITATIONS

This study has limitations worth emphasising. As with most epidemiological studies in diving medicine, the primary limitation to the generalisation of results is the lack of a denominator. This limitation, and a relatively small sample size, prevents us from inferring the possible prevalence of diving injuries among minors and making sensible comparisons with adult diving populations. A future study design would be to look at adult diver injury incidence from the DAN Hotline and compare to minor divers.

Another limitation concerns the methodology used. As this retrospective study was waived from consent, we did not contact any party to gather more information about these incidents. Instead, we worked with the documentation available: written narratives, call recordings, medical records shared by calling parties, and any public domain records when available. Re-interviewing the parties involved in these cases could provide much more data than we could gather by retrospectively reviewing existing records.

The role of anxiety as a trigger and root cause of an injury is likely underrepresented. This could be partly due to the subjective nature of anxiety, a possible behavioural bias from

minors not always accepting and verbalising their fears, and inherent defects in the quality of the data captured and its completeness.

Due to inherent limitations in telecommunications, the final diagnosis category of 'anxiety' as the explanation of all manifestations was at the discretion of the evaluating physician and could be underestimated as well. We have since updated our standard operating procedures and tools to better capture anxiety as a differential diagnosis and risk factor.

Conclusions

Dive-related emergencies involving minor divers are rare. Our data suggest that lung over-expansion injuries seem more common than DCS. During the three years we analysed, 10% of the calls involving a minor with a suspected diving injury had a final diagnosis of PBt, making up to 15 percent of the diving injuries in minors during that period.

Factors contributing to PBt in minors might be associated with fitness and immaturity. Qualified fitness to dive evaluation, improved training, and closer adult supervision might help mitigate the risks of injuries in minor divers.

Dealing with diving accidents is extremely stressful at the best of times, but it is far more so if the victim is a minor, due to the additional emotional pressures, which should not be underestimated.

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Underwater pulse oximetry reveals increased rate of arterial oxygen desaturation across repeated freedives to 11 metres of freshwater

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Breath-hold diving; Diving reflex; Hypoxia; Oxygen consumption; Safety; Unconsciousness

Abstract
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Introduction: Recreational freedivers typically perform repeated dives to moderate depths with short recovery intervals. According to freediving standards, these recovery intervals should be twice the dive duration; however, this has yet to be supported by scientific evidence.
Methods: Six recreational freedivers performed three freedives to 11 metres of freshwater (mfw), separated by 2 min 30 s recovery intervals, while an underwater pulse oximeter measured peripheral oxygen saturation (SpO_2) and heart rate (HR).
Results: Median dive durations were 54.0 s, 103.0 s and 75.5 s (all dives median 81.5 s). Median baseline HR was 76.0 beats per minute (bpm), which decreased during dives to 48.0 bpm in dive one, 40.5 bpm in dive two and 48.5 bpm in dive three (all $P < 0.05$ from baseline). Median pre-dive baseline SpO_2 was 99.5%. SpO_2 remained similar to baseline for the first half of the dives, after which the rate of desaturation increased during the second half of the dives with each subsequent dive. Lowest median SpO_2 after dive one was 97.0%, after dive two 83.5% ($P < 0.05$ from baseline) and after dive three 82.5% ($P < 0.01$ from baseline). SpO_2 had returned to baseline within 20 s after all dives.
Conclusions: We speculate that the enhanced rate of arterial oxygen desaturation across the serial dives may be attributed to a remaining 'oxygen debt', leading to progressively increased oxygen extraction by desaturated muscles. Despite being twice the dive duration, the recovery period may be too short to allow full recovery and to sustain prolonged serial diving, thus does not guarantee safe diving.

Introduction

Breath-hold diving, or 'freediving' is an underwater activity practiced for competition, recreation or as a profession. In competitive freediving, athletes aim to reach a maximum time, distance or depth underwater in one dive.[1–4] The growth in popularity of the competitive sport has led to a rapid increase also of recreational freedivers world-wide.[5] All freediving activity is done on one breath of air only, and divers must rely on physiological responses to conserve oxygen to sustain brain function and work capacity, and if they overestimate their ability they are at risk of hypoxic syncope,[6,7] also called 'blackout'. While competitive freediving is made relatively safe via the presence of safety divers who can bring the diver back to the surface should blackout occur, recreational freedivers are at greater risk; according to statistics from Diving Alert Network (DAN) an average of 51 freedivers per year drown, which may be related to blackout events.[5]

Unlike competition divers, who perform one maximal dive after which they can rest and recover for an unlimited time, recreational freedivers typically perform repeated non-maximal dives to moderate depths with short recovery intervals. But how do freedivers know how to pace their dives to allow full recovery and safe continued diving? There is a 'rule of thumb' that the diver should spend double the dive duration at the surface to fully recover between subsequent dives. However, the serial effects of repeated freedives in a field setting are in fact not well known. Most studies of freediving physiology to date have been done in the laboratory due to the challenges with underwater measurements, and studies of repeated dives with short intervals revealed some serial effects on physiological responses and peripheral oxygen saturation (SpO_2).[8,9] Could changes induced by repeated diving lead up to hypoxic loss of consciousness? To understand if incomplete recovery of oxygen stores between dives could be a factor responsible for blackout in recreational divers, it is necessary to perform research during actual freediving to depth, as the gas

exchange and physiological responses are likely affected by the changes in hydrostatic pressure.

We recently constructed an underwater pulse oximeter enabling continuous measurements of heart rate (HR) and SpO_2,[10] and monitored these variables during deep single dives in competition divers.[11] Our current aim is to use this novel technology in recreational divers to study SpO_2 and HR, to investigate any serial effects in short dives. While SpO_2 will reveal the level of oxygen remaining after each dive, the HR-recordings will show the magnitude of the 'diving response' which conserves oxygen to sustain brain function[12,13] by peripheral vasoconstriction[14,15] and bradycardia.[16,17] By studying the diving response and arterial oxygen depletion during three subsequent dives to 11 metres of fresh water (mfw), a commonly reached depth for recreational divers, we aimed to reveal whether it is true that a diver can safely continue diving as long as the recovery interval is kept double the dive time.

Methods

The study protocol was approved by the Regional Committee for Medical and Health Research Ethics in Umeå. Sweden, (Dnr 2019-05147) and the tests were conducted in accordance with the Declaration of Helsinki. All participants gave their informed, written consent to participate in the study.

PARTICIPANTS

Six male recreational freedivers with a mean and standard deviation (SD) age of 36 (8) years, height 181 (3) cm and weight 89 (13) kg from a local freediving club participated. They had trained freediving regularly for a minimum of one year, and thereby qualified in category 3 in the 5-level categorisation system used to describe training levels of freedivers.[18] All divers were accustomed to repeated freediving and had frequently been to at least 12 meters depth, and were trained in safety procedures.

PROCEDURES

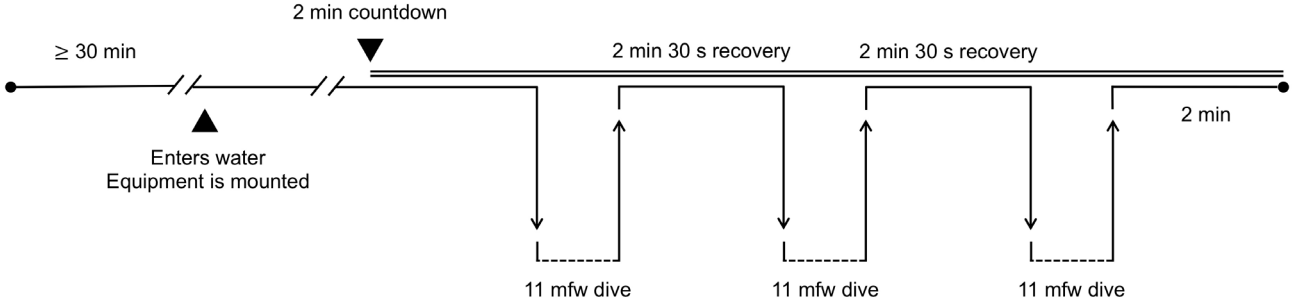
The participants reported to the 11 m deep indoor freshwater diving facility with an ambient air and water temperature of 32°C at least two hours after eating, wearing their own freediving equipment (mask, fins, wetsuit, weights). They were weighted to be neutrally buoyant at approximately 10 mfw. After 30 min of poolside rest, participants performed three consecutive freedives to 11 mfw separated by resting intervals at the surface, with countdown for the following dive. This protocol was aimed to reflect a part of their usual training routine; thus, dive durations were entirely determined by the divers, without receiving any input from the research team. Based on previous experience, the dive time was estimated to be on average about 1 min 15 s and the recovery interval, intended to be of about double the dive duration, was pre-set to 2 min 30 s (Figure 1). Participants were asked to refrain from hyperventilating and 'lung packing' manoeuvres, which are often used by competition divers to enhance dive duration.[19] Dives were conducted with established safety procedures including safety divers.[3] Following a 2-min countdown before each dive, the freediver swam down along a vertical diving line at his own pace and stayed at the bottom of the pool as long as he would do during usual training. Upon resurfacing, divers performed the recovery hook-breathing technique which is part of the safety measures frequently used by competition divers and many recreational freedivers.[19] Diving and interval durations were monitored with a stopwatch and recorded by the underwater depth-time profile of the datalogger.[20]

MEASUREMENTS

Upon entering the water, the participants were equipped with the prototype pulse oximeter for continuous measurements of SpO_2 and HR.[10] For details regarding the method and analysis see previous publications.[10,11] In short, plethysmograms were recorded at 30 Hz, and SpO_2 and HR were extracted from the plethysmograms at a window size of six seconds, and analysed internally every second. The

Figure 1

The experimental procedure consisting of three 11 metres of fresh water (mfw) freedives with 2 min 30 s recovery between the dives. Double line signifies continuous measurements of peripheral oxygen saturation (SpO_2) and heart rate (HR). Dotted line signifies bottom time, which was determined by the diver



two sensor heads were placed on both temples of the diver, and kept in place with medical tape and by the pressure of the hood of the wetsuit. Recording started two minutes before the first dive and continued until two minutes after termination of the third dive.

For the analysis, the results of both sensors were averaged for each data point to obtain one single value for SpO_2 and HR for each second.[10,11] The resulting time series for each dive was then smoothed using a 5-s moving median function. Baseline SpO_2 and HR were determined by calculating the mean over the time period of 90 to 30 s of measurements prior to the first dive.[12] The maximal arterial oxygen desaturation was calculated as the percentage change between the previously established baseline SpO_2 and the lowest SpO_2 value (SpO_{2nadir}).[21] We aimed to identify divers at risk of reaching severe hypoxia by tallying the number of divers reaching SpO_2 less than 75% in dives one to three. The magnitude of the diving bradycardia was calculated as the percent change from baseline HR, and maximal HR-reduction determined by identifying the lowest HR value (HR_{nadir}) of each dive.[21]

Heart rate data for all divers were also identified for the different diving phases for each dive, including baseline, descent (average of a 5 s window around 5 mfw during the descent), bottom (average of the entire period at the bottom), ascent (average of a 5 s window around 5 mfw during the ascent), first recovery period (average of the first 15 s directly after resurfacing) and second recovery period (average of values between 16 and 30 s after resurfacing). Because the duration of dives differed between participants, data for each dive were also expressed for subsequent timepoints representing percentages of total dive time.

STATISTICS

Due to the small sample size, a Friedman test was run to determine if there were differences in dive times and the studied physiological variables across the three dives. Pairwise comparisons were performed with Bonferroni corrections for multiple comparisons. For all data median values and interquartile range are presented, but mean values are also provided in an additional table for clarity. Additionally, a mixed model repeated measures analysis of variance (ANOVA; within factors: diving phase; between factors: dive number) were used to identify if dive number influenced the pattern of HR throughout the dive. Significant interactions or main effects were followed up with simple main effect analyses with pairwise comparisons using Bonferroni correction. For all ANOVA's, effect sizes are presented as partial eta-squared statistic (η^2_p). $P < 0.05$ was considered statistically significant.

To assess the impact of repeated dives on 1) arterial oxygen desaturation during diving and 2) reoxygenation during post-dive surface intervals, two models were constructed to assess the impact across each of three dives. The diving data were treated as the periods of diving, beginning at the onset of apnoea until the presentation of the oxygen saturation minimum at, or immediately after, the termination of diving. Surface intervals were based on the remaining data, extending from the presentation of the oxygen minima to the onset of the next dive. For each category (diving desaturation and surface reoxygenation), two models were considered. Models were constructed for each category separately. Peripheral oxygen saturation was modelled as a function of proportion of the dive (allowing comparison of variable dive durations) or surface interval (0–100%) or time since surfacing in an interaction term with dive number. In each model, the proportion of the dive was included as a continuous smooth term and dive number as a fixed factor. The framework used for the above-described analysis was generalised additive models (GAM), constructed within the 'mgcv' package in R.[22] Additive models were required to allow complex, non-linear relationships between covariates and response metrics. For each model, the final model was chosen by a model minimising the information theoretic criterion (AIC),[23] if their inclusion did not improve the model by two or more ΔAIC. Residual plots were examined for any evidence of violation of model assumptions.

Results

DIVE DURATION

All participants completed the three dives without reporting any symptoms related to hypoxic blackout, nor were there any symptoms of severe hypoxia, i.e., loss of motor control, observed by the researchers. Median (range) dive durations for dives one, two and three were 54.0 (39–83) s, 103.0 (86–113) s and 75.5 (58–122) s ($\chi^2(2) = 2.333$, $P = 0.311$). Median duration of all dives was 81.5 (51–108) s. The resulting median duration of recovery intervals was 151 (150–161) s, thus close to double the dive duration. Median (IQR) and mean (SD) values for the studied variables are presented in Table 1.

PERIPHERAL OXYGEN SATURATION

Median pre-dive baseline SpO_2 was 99.5 (99–100)%. SpO_{2nadir} occurred on average at 7.5 (2–8) s after resurfacing across all dives, thus the circulatory delay was the same in all dives. SpO_2 was significantly different across the diving series ($\chi^2(3) = 16.119$, $P = 0.001$). Post hoc analysis revealed a difference between baseline SpO_2 and SpO_{2nadir} after resurfacing from dive two (83.5 (70–87)%; $P = 0.015$) and three (82.5 (58–95)%; $P = 0.003$), but there were no significant differences between SpO_{2nadir} of dives one, two and three. The number of divers that dropped below an SpO_2 of 75% increased from none during dive one, to two in dive two, to three in dive three.

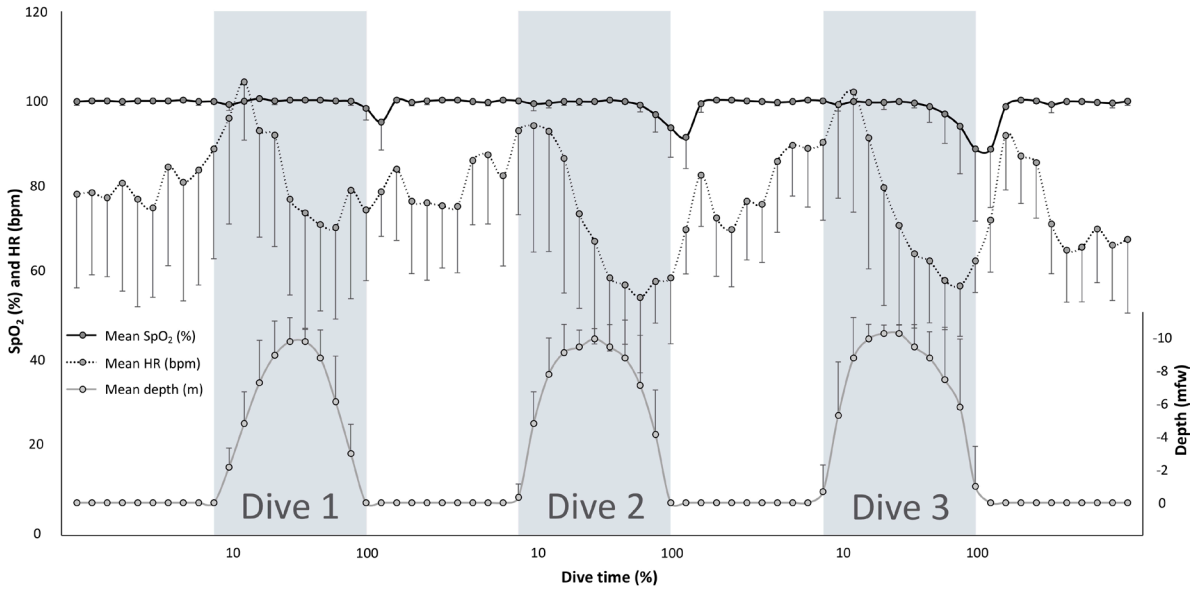
Table 1

Dive durations, pulse oximetry and heart rate results from baseline (pre-dive) and three dives to 11 metres of fresh water (mfw); HR – heart rate; HR_{nadir} – lowest heart rate in the dives; IQR – interquartile range; NA – not applicable; SpO_2 – peripheral oxygen saturation; SpO_{2nadir} – lowest peripheral oxygen saturation in the dives

Parameter	Statistic	Baseline (pre-dive)	Dive one	Dive two	Dive three
Dive duration (s)	Median (IQR)	NA	54 (39–83)	103 (86–113)	75.5 (58–122)
	Mean (SD)	NA	63 (30)	96 (28)	87 (42)
SpO_2 pre-dive SpO_{2nadir} (%)	Median (IQR)	99.5 (99–100)	97 (81–98)	83.5 (70–87)	82.5 (58–95)
	Mean (SD)	100 (1)	91 (11)	80 (14)	76 (22)
Oxygen desaturation (Δ%)	Median (IQR)	NA	3.0 (17.7–1.3)	16.1 (29.0–13.0)	17.1 (41.7–5.5)
	Mean (SD)	NA	8.7 (10.9)	19.7 (14.0)	23.7 (21.8)
HR pre-dive HR_{nadir} (bpm)	Median (IQR)	76 (61–97)	48 (41–69)	40.5 (36–48)	48.5 (36–51)
	Mean (SD)	79 (21)	55 (19)	45 (14)	44 (9)
Maximal HR reduction (Δ%)	Median (IQR)	NA	31.0 (36.8–21.4)	43.0 (48.7–39.5)	45.4 (48.3–42.8)
	Mean (SD)	NA	30 (16)	42 (12)	43 (10)

Figure 2

Mean and standard deviation (SD) heart rate (HR) in beats per minute (bpm), peripheral oxygen saturation (SpO_2 %) and dive depth profile in metres of fresh water (mfw) from six freedivers in three sequential dives to 11 mfw; values are relative to dive time; dive time is expressed as percentage to be able to compare means from all dives which were of different durations



HEART RATE

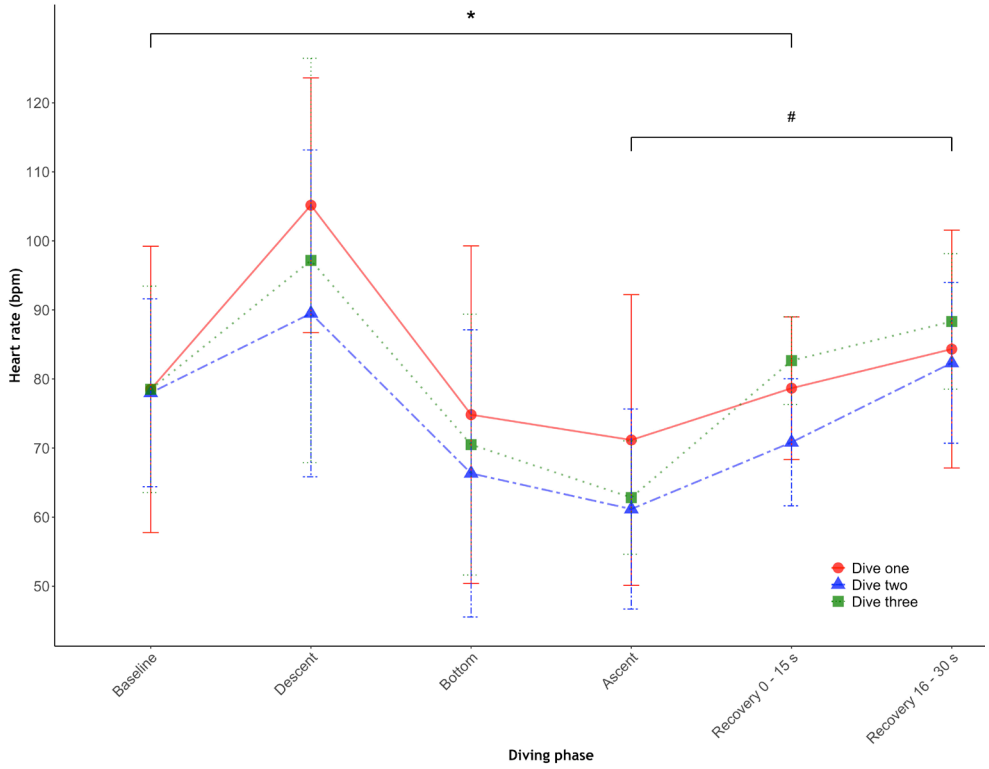
Median pre-dive baseline HR was 76.0 (61–97) bpm. HR was significantly different across the diving series, ($\chi^2(3) = 12.559$, $P = 0.006$). Post hoc analysis revealed a significant difference between baseline HR and HR_{nadir} from dive one (48.0 [41–69] bpm; $P = 0.044$), two (40.5 [36–48] bpm; $P = 0.001$) and three (48.5 [36–51] bpm; $P = 0.005$), but there were no significant differences between HR_{nadir} when the three dives were compared (Figure 2).

DIVING RESPONSE PHASES

Figure 3 displays the HR responses divided into different phases for dives one, two and three. Heart rate responses for the different diving phases shows similar patterns for all divers, regardless of dive number or individual absolute HR, i.e., there were no significant differences in each phase between the dives. There was no significant interaction between dive number and diving phase ($F_{10,75} = 0.485$, $P = 0.895$, η^2_p $P = 0.061$), and no main-effect for dive number

Figure 3

Mean (SD) heart rate in beats per minute (bpm) for divers undertaking each dive, with dives divided into phases (baseline, descent, bottom, ascent and recovery); * indicates significant difference between descent phase and all other phases except for the second recovery phase. # indicates significant difference between ascent phase and both recovery phases



($F_{2,15} = 0.524$, $P = 0.602$, η^2_p $P = 0.065$). However, there was a main effect for diving phase ($F_{5,75}$ = 14.046, $P < 0.001$, η^2_p P = 0.484). Heart rate increased from baseline during descent, when divers actively needed to overcome positive buoyancy. When divers reached the bottom of the pool, HR dropped below baseline levels, and continued to drop further during the ascent, where positive buoyancy reduced work ($P < 0.05$ between the descent phase and all other phases except for second recovery phase). Upon resurfacing, HR increased back to baseline levels ($P < 0.05$ between ascent phase and both recovery phases).

OXYGEN DESATURATION AND RESATURATION PATTERNS

The fitted model predicted that SpO_2 for the first half of the dive was similar between dives one, two and three. However, during the second half of the dives the rate of desaturation increased with each subsequent dive (Figure 4 left panel). The maximal model was retained as the best model with lowest AIC, thus, the interaction term between proportion of dive and dive number was retained allowing flexibility in the model to fit different intercepts to each dive, supporting the finding that the patterns of change in SpO_2 between dives were different. Further, the fitted model predicted that the pattern and rate of reoxygenation was similar between recovery surface intervals (Figure 4 right panel). The final model did not retain the interaction term between dive duration and dive number, supporting the observation that the patterns of change in SpO_2 during recovery intervals was not different across the dive series.

Discussion

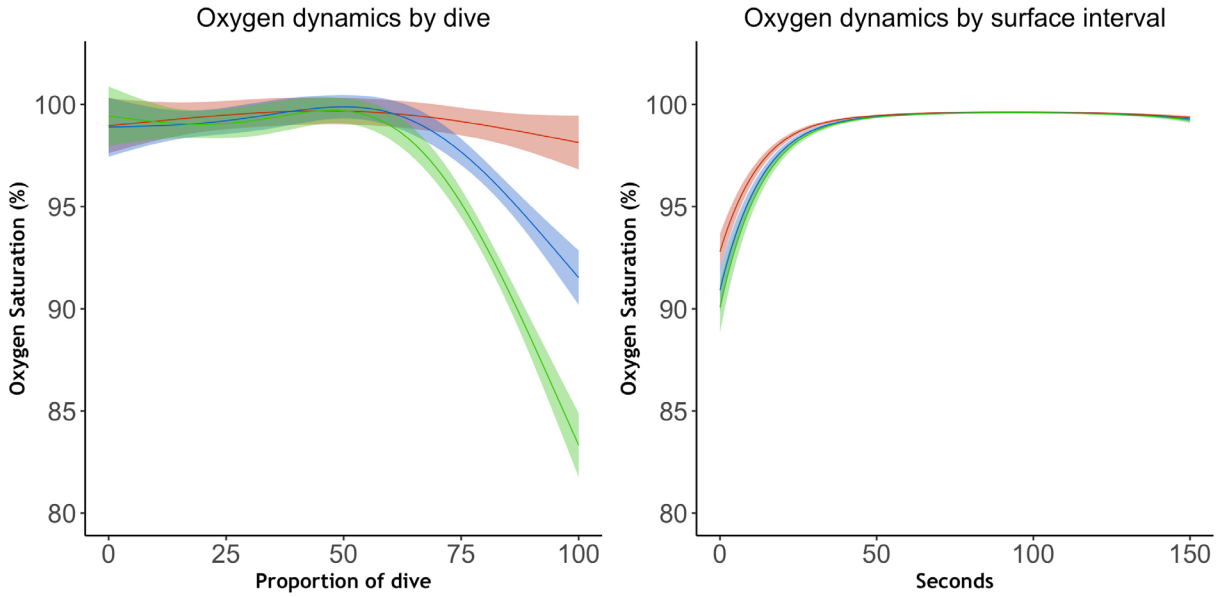
This study is, to our knowledge, the first to use underwater pulse oximetry to examine SpO_2 and HR continuously during repeated diving to moderate depths in recreational freedivers.

PERIPHERAL OXYGEN SATURATION PATTERNS

A progressively increased desaturation occurred across the dive series. Modeling of oxygen desaturation and resaturation rate showed that desaturation rate increased from the first dive through the third dive, despite similar oxygen resaturation patterns during recovery. This seems to imply that oxygen was consumed at a faster rate for each subsequent dive, despite similar magnitudes of the oxygen conserving diving response and despite similar dive durations. We are also unable to relate these differences in desaturation rate to differences in levels of exertion between dives, as depth was the same for each dive, which is also supported by similar HR-response and dive durations. We therefore speculate that the enhanced rate of peripheral oxygen desaturation across the three dives may be attributed to a subsequently increased oxygen extraction in working muscles. It is well known that oxygen uptake recovers more

Figure 4

Left panel – plots represent mean oxygen dynamics for dive one (red), dive two (blue) and dive three (green), shaded areas represent 95% confidence intervals. The changed shape and slope of the curve indicates statistically significant increased rate of oxygen desaturation for each subsequent dive. Right panel – lines represent oxygen dynamics upon resurfacing for dive one (red), dive two (blue) and dive three (green), shaded areas represent 95% confidence intervals, demonstrating similar rates of reoxygenation after all dives



slowly following muscular activity than arterial oxygen concentration.[24] The resting interval between dives was about twice the average dive duration, thus following the 'rule of thumb' taught in freediving schools, but our study shows that this may in fact be too short to allow full recovery for the divers, due to a remaining oxygen debt in their muscles.

Consequently, our findings indicate that the relatively short repetitive freedives to moderate depth, characteristic of recreational freedivers, may induce hypoxaemia at a progressively increased rate across the series. The lowest SpO_2 observed was 47% in one diver during the last dive which lasted 135 s, which is below 50%, a level considered to be associated with blackout in untrained individuals.[1] We speculate that a longer diving series may enhance the oxygen debt further which could involve a risk of blackout.

Even though we are aware of the possible margin of error in these pulse oximetry measurements, a well-known problem associated with all pulse oximetry measurements when oxygen saturation is decreased, these results are similar to data presented by others,[25] from a dive lasting 300 s by an elite diver. This shows that our recreational divers indeed approached very low levels of oxygen saturation in a shorter amount of time, and that some of our divers may have been very close to levels associated with blackout in non-elite divers.

Our data thus shows that factors other than dive duration may be important when aiming to predict oxygen management during repeated diving. These results are highly relevant from a safety perspective as recreational freedivers seem to be at particular risk for hypoxic blackout.[5] Our findings indicate that the pacing of repeated dives is essential to safety, and that the current recommendations are not sufficient.

DIVING RESPONSE PATTERNS

Heart rate traces for all divers and dives had similar features: an increase in HR from baseline levels during the descent phase, when the divers were actively swimming down while overcoming positive buoyancy. During this phase the exercise tachycardia thus dominates over the diving bradycardia, and the cardiovascular diving response is not developed. A contributing factor could be that depth was only 11 mfw, and the descent phase may have been too short to allow the drop in HR due to the diving response, typical of the passive 'free-fall' phase in deep freediving.[3,11] In studies of deeper and longer dives it appears that the two stimuli for exercise tachycardia and diving bradycardia may balance out resulting in an intermediate HR.[11]

Another factor responsible for the high HR during the descent phase in our study could be that the HR reduction, in addition to being triggered by apnoea, is influenced by facial cold-receptors stimulated by the difference between ambient air and water temperature.[26] The fact that both air and water were 32°C in our study may have resulted in a smaller stimulus than in a previous study where air was 26°C and water 22°C.[11]

When the diver reached the bottom and stopped swimming, bradycardia developed, as also observed in deeper dives.[11,27,28]

Compression of the lungs by hydrostatic pressure to half their size at the surface could also have contributed to the drop in HR at the bottom.[29]

During ascent the low HR prevailed in the positively buoyant diver, while in deep diving this phase is associated with an elevation in HR due to the intense work against negative buoyancy.[11] During the initial 15 s after surfacing, HR increased, and this recovery tachycardia continued to increase in magnitude during the following 15 s when breathing had elevated SpO_2, however, not to same extent as patterns previously noted after deep dives.[11]

The percent change from baseline to the lowest HR attained during the dives increased from 30 (16)% during the first dive to 42 (12) and 43 (10)% during dive two and three respectively, however this increase was not statistically significant. A lowest individual HR reduction of 58% was observed in one diver during the second dive. The HR reductions of the latter two dives is comparable to those found in previous studies in competition divers.[11,27]

We would also like to suggest that the tendency for longer dive durations in dives two and three may demonstrate that the initial dive acts as a warm-up, enabling the diver to produce longer apnoeic duration due to the recruitment of spleen red cell stores,[8] or enhancement of other protective reflexes. This should be examined in a larger group of divers, as the small group studied here would possibly lead to type two errors.

LIMITATIONS

A limitation of this study is the small sample size, which reduces statistical power, and only allows preliminary conclusions to be drawn from our results. Also, pulse oximetry is less reliable at low saturation levels, so absolute values in the low range can be numerically less accurate than e.g., blood gas analysis,[30] but their relative values between dives would be reliable. The observation that the response patterns were similar for all divers regardless of their differences in response magnitude could support the generalisation of these findings. Future studies should include a larger sample and more subsequent dives to further evaluate the effects of serial diving.

Conclusions

In the current group of recreational divers, a series of three dives separated by recovery periods of approximately twice the dive duration led to subsequently increased rate of arterial oxygen desaturation, despite attaining baseline SpO_2 values within 30 s of resurfacing. The fact that the rate of oxygen desaturation increased throughout the freediving session did not overtly compromise the safety of these divers, even though some individuals reached extremely low SpO_2 values. However, the combination of progressively increased rate of oxygen desaturation for longer sequential dive sessions could pose a risk of blackout, especially if dives are being prolonged. We therefore speculate that the enhanced rate of peripheral oxygen desaturation across the three dives may be attributed to an increased oxygen extraction in inadequately recovered working muscles. Indeed, in three out of six divers, this led to hypoxia below 75% SpO_2 in the last dive. Despite being twice the dive duration, the recovery period may have been too short to allow full recovery, thus an oxygen debt had accumulated. This suggests that the 'rule of thumb' – that such intervals guarantee safe freediving – may not be reliable for longer freediving sessions. Longer dive series should be studied to see if the rate of oxygen desaturation continues to increase and may cause a risk of hypoxic blackout. To evaluate our results further, it would be helpful if oxygen uptake measurements could be made across serial dives, and if venous oxygen levels could be determined.

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Intraindividual variability of the Eustachian tube function: a longitudinal study in a pressure chamber

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Abstract
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Introduction: The Eustachian tube (ET) is essential for fast and direct pressure equalisation between middle ear and ambient pressure. It is not yet known to what extent Eustachian tube function in healthy adults changes in a weekly periodicity due to internal and external factors. This question is particularly interesting with regard to scuba divers among whom there is a need to evaluate intraindividual ET function variability.

Methods: Continuous impedance measurement in a pressure chamber was performed three times at one-week intervals between measurements. Twenty healthy participants (40 ears) were enrolled. Using a monoplace hyperbaric chamber, individual subjects were exposed to a standardised pressure profile consisting of a 20 kPa decompression over 1 min, a 40 kPa compression over 2 min, and a 20 kPa decompression over 1 min. Measurements of Eustachian tube opening pressure (ETOP), opening duration (ETOD), and opening frequency (ETOF) were made. Intraindividual variability was assessed.

Results: Mean ETOD during compression (actively induced pressure equalisation) on the right side was 273.8 (SD 158.8) ms, 259.4 (157.7) ms, and 249.2 (154.1) ms (Chi-square 7.30, $P = 0.026$) across weeks 1–3. Mean ETOD for both sides was 265.6 (153.3) ms, 256.1 (154.6) ms, and 245.7 (147.8) ms (Chi-square 10.00, $P = 0.007$) across weeks 1–3. There were no other significant differences in ETOD, ETOP and ETOF across the three weekly measurements.

Conclusions: This longitudinal study suggests low week-to-week intraindividual variability of Eustachian tube function.

Introduction

The Eustachian tube (ET) is an anatomical connection between the middle ear and nasopharynx that can be subdivided into a medial cartilaginous and a lateral osseous section.[1,2] Physiologically, it has three commonly described main tasks: first, pressure equalisation between the middle ear and ambient pressure; second, protection against repercussion or pathogens from the nasopharynx; and finally, mucociliary clearance of middle ear secretions.[3,4] To balance pressure between the middle ear and the ambient pressure there slow gas exchange across the tympanic membrane as well as middle ear mucosa. In contrast, the ET periodically allows a fast and direct middle ear ventilation which is a fundamental prerequisite for pressure equalisation during flying and diving.[5] Under physiological conditions, pressure equalisation during decompression (ambient pressure decrease, e.g., airplane takeoff or diving ascent) normally happens spontaneously. During compression (ambient pressure increase, e.g., airplane landing or diving descent) pressure equalisation must be actively induced.[6]

The development of an exact, objective and sufficiently specific tool to measure the pressure equalisation function of the ET remains a challenge, even though a variety of assessments (e.g., impedance measurements, manometric, sonographic and endoscopic methods or tubomanometry) were established in recent years. Unfortunately, no single test appears to match the criteria to become a gold standard.[7] The combination of missing universal applicability as well as a lack of general informative value is the main problem in this process. On the one hand, a physiological test environment is required to obtain reliable data on ET function. On the other, most tests generate only short and non-dynamic information or use non-physiological pressure levels and/or pressure change rates.[8]

With this working group, a method was used to determine the pressure equilibration ET function involving the use of a pressure chamber. The combination of diagnostic impedance measurement and application of a defined pressure profile in a hypo- and hyperbaric pressure chamber allows a dynamic and objective analysis of ET function. Therefore, variables describing ET function were introduced and examined in healthy cohorts.[9,10] According to recent studies, the present method is also applicable if cohorts consist of patients with chronic Eustachian tube dysfunction (ETD).[11,12]

Unspecific symptoms overlapping with other middle and inner ear pathologies complicate a clear distinction of ETD. For this reason, a recent consensus agreed on the definition as being a "*syndrome with a constellation of signs and symptoms suggestive of dysfunction of the Eustachian tube*".[4] Typically, patients report symptoms like aural fullness, popping, discomfort or pain, among others.[13] Chronic ETD can lead to the development of tympanic membrane retraction and chronic otitis media with effusion, with or without cholesteatoma.[14–16] In general, ETD is presumed to have a prevalence of 0.9% among adults.[17,18] One study reported a prevalence of 4.6% among adults in the United States of America.[19] The development of new treatment strategies has amplified the need for patient selection through reliable ET function testing and objective outcome measurement to improve quality of care.[20]

The aim of this study was to characterise the intraindividual variability of ET function in healthy participants in a longitudinal study by using a continuous impedance measurement during pressure changes in a hypo- and hyperbaric pressure chamber. The impact of possible ET function-influencing factors such as nutrition, sport activity, hormonal status, nasal cycle, or mild subclinical infection on ET function has not yet been sufficiently characterised.[21–25] A further goal was to help classify ETD treatment outcome measurements in previous as well as in future studies, since periodic fluctuations of ET function may interfere with conclusions about treatment effects. As previous studies suggest that the ET opening pressure during decompression may be higher in patients with ETD and can be reduced by Eustachian tube balloon dilatation (ETBD), results of this study were compared with earlier studies.[10–12]

Methods

The study was approved by the local ethics committee of the University of Cologne medical faculty. Written and informed consent had been obtained from all participants. The study is in accordance with the latest version of the Declaration of Helsinki.

PARTICIPANTS

This prospective study included twenty healthy participants (40 ears). The mean age was 25.9 (SD 4.0) years, 60.0% were female and 40.0% male. All participants affirmed that no symptoms and/or signs of ET dysfunction were present on each date of measurement. None of the participants reported prior problems with pressure equalisation while flying or diving. A full ear, nose and throat examination including ear microscopy and endoscopy of the nose and epipharynx was conducted by an otorhinolaryngologist. The ability to perform a visible Valsalva maneuver on both ears was confirmed. Exclusion criteria were colds, any form of perforated tympanic membrane, symptoms and/or signs of active allergic rhinitis, severe septum deviation, adenoid hypertrophy, gastroesophageal reflux disease and pregnancy.

CONTINUOUS IMPEDANCE MEASUREMENT IN A HYPO- AND HYPERBARIC PRESSURE CHAMBER

A single person chamber (Haux Life Support, Karlsbad, Germany) was used to apply a defined pressure profile for ET function measurements with an interval of one week between sessions. The pressure profile has been utilised in previously published studies.[6,8–12] It consists of two decompression phases of one minute each and a compression phase of two minutes between them (see Figure 1A). For continuous measurement of tympanic impedance, a size-adjusted rubber earplug was fitted in the external ear canals on both sides. The earplug contains three channels: (1) a loudspeaker delivering a 226 Hz tone; (2) a microphone; and (3) a small tube for pressure equalisation between the chamber and the external ear canal (see Figure 2). ET function was measured by recording the reflection of the acoustic signal simultaneously with the controlled application of pressure change. The setup as a combination of pressure chamber and continuous tympanic impedance measurement allows an objective and dynamic measurement of the ET function separately for the left and right side. In this way generated curves (see example in Figure 1B) were subsequently analysed in a specific analysis program.

In case of a participant's discomfort due to any reason, measurements would have been stopped immediately. The pressure could have been manually equalised to the atmospheric pressure.

EUSTACHIAN TUBE FUNCTION IN DECOMPRESSION (PASSIVE OPENINGS) VS COMPRESSION (ACTIVELY INDUCED OPENINGS)

While participants were instructed to not induce any active pressure equalisation during phases of decompression, they were asked to actively equalise pressure either by Valsalva manoeuvre or by swallowing during compression. They were advised to perform the Valsalva manoeuvre or swallowing whenever a feeling of discomfort occurred. They were advised to not switch the chosen equalisation method for the compression phase between measurements.

Further and detailed ET function analysis was achieved by using the variables ET opening pressure (ETOP), ET opening duration (ETOD) and ET opening frequency (ETOF). All

Figure 1
A – Pressure/time profile of the chamber test runs; B – Example of the continuous impedance measurement (green curve); detailed analysis was performed with a tenfold magnification. L – left side; R – right side

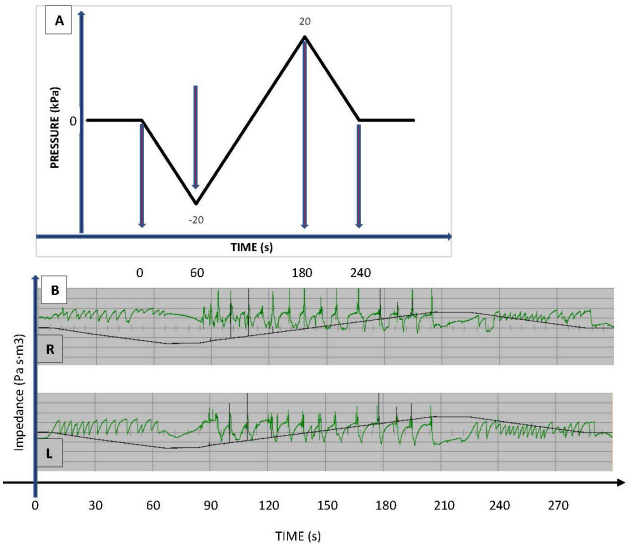


Figure 2
Setup of the ear plug arrangement inside the single person pressure chamber

three variables were calculated for decompression and compression separately as previously published.[9–12,26] The ETOP during decompression (passive pressure equalisation) was defined as the pressure difference between the initial pressure and first ET opening. Similarly, ETOP during compression (actively induced pressure equalisation) was calculated as the mean of pressure differences between minimum and maximum impedance during pressure increase. The time interval between ET opening (maximum impedance) and ET closing (minimum impedance) represented ETOD during decompression. The mean of time intervals between ET opening (maximum impedance) and ET closing (minimum impedance) was calculated to determine ETOD during compression. In contrast to phases of decompression, ETOP during compression is influenced by the participant's personal perception, as it depends on the individual decision to induce pressure equalisation. Therefore, all actively induced openings were analysed individually and afterwards an average value was calculated to represent the variable.

Since preliminary studies have shown that ETOP during decompression is the most important variable to represent Eustachian tube function, we have compared our findings with results from previous studies.

STATISTICAL ANALYSIS

Statistical analysis was performed using SPSS Statistics version 26 (IBM Corporation, Armonk, NY, USA). Data are presented as mean standard deviation (SD). Initially, the Shapiro-Wilk and Kolmogorov-Smirnov tests were used to determine whether the resulting values of the three repetitive measurements follow a normal distribution. Depending on the results, either a one-way analysis of variance (ANOVA) for repeated measurements or a Friedman test was performed to detect possible differences between measurements. To exclude a proportional bias, a final linear regression was carried out. To compare the results of ETOP during decompression with recent publications, an additional ANOVA was performed.[10–12] Null hypotheses were tested with a significance level set at $P < 0.05$.

Results

The means (SD) of all ET function-reflecting variables for each measurement are presented in Table 1. The results are shown for the right, left and the mean for both sides, respectively. Except for the ETOD during compression (actively induced pressure equalisation) on the right side and the mean for both sides, there were no significant differences calculated for variables between measurements I–III.

Linear regression revealed no evidence for any proportional bias. For ETOD, a non-parametric Friedman test of differences among repeated measurements rendered a Chi-square value of 7.30 (right) and 10.00 (mean of both sides), which was significant ($P = 0.026$ for the right side and $P = 0.007$ for the mean of both sides). Dunn-Bonferroni-adjusted post-hoc analysis revealed that measurement I and III differ from each other ($z = 2.69$, $P_{adjusted} = 0.022$ for the right side and $z = 3.16$, $P_{adjusted} = 0.005$ for the mean of both sides).

As an ANOVA appears to be quite robust against violations of the assumption of normal distribution, it was also performed

Table 1

Eustachian tube function measurements in the pressure chamber; all data are mean (standard deviation); B – both sides; ETOD – Eustachian tube opening duration; ETOF – Eustachian tube opening frequency; ETOP – Eustachian tube opening pressure (ETOP); L – left side; R – right side

Parameter	Measurement I			Measurement II			Measurement III		
	R	L	B	R	L	B	R	L	B
Pressure decrease (passive pressure equalisation)									
ETOP (kPa)	2.65 (1.08)	2.55 (0.96)	2.6 (0.95)	2.82 (1.05)	2.61 (0.90)	2.72 (0.92)	2.67 (0.97)	2.60 (0.87)	2.63 (0.89)
ETOD (ms)	806.5 (323.2)	781.0 (374.6)	793.8 (331.6)	828.8 (351.9)	752.5 (372.)	790.6 (343.2)	813.8 (342.3)	799.5 (356.1)	806.6 (337.0)
ETOF (min^{-1})	7.7 (5.0)	8.0 (5.0)	7.9 (4.7)	7.5 (6.0)	7.9 (6.2)	7.7 (5.8)	7.6 (5.5)	8.3 (5.7)	7.9 (5.3)
Pressure increase (actively induced pressure equalisation)									
ETOP (kPa)	2.90 (1.46)	2.97 (1.63)	2.94 (1.55)	2.98 (1.57)	3.11 (1.70)	3.04 (1.63)	2.87 (1.50)	2.96 (1.66)	2.92 (1.58)
ETOD (ms)	273.8 (158.8)	257.5 (153.7)	265.6 (153.3)	259.4 (157.7)	252.8 (155.7)	256.1 (154.6)	249.2 (154.1)	242.3 (143.7)	245.7 (147.8)
ETOF (min^{-1})	5.3 (2.8)	5.5 (2.9)	5.4 (2.8)	5.9 (3.2)	5.8 (3.3)	5.9 (3.2)	6.1 (3.4)	6.1 (3.5)	6.1 (3.4)

Figure 3

The mean and standard deviation of all ET function variables for each measurement (I, II and III) during compression (A–C) and decompression (D–F); aETOD – actively induced Eustachian tube opening duration; aETOF – actively induced Eustachian tube opening frequency; aETOP – actively induced Eustachian tube opening pressure; pETOD – passive Eustachian tube opening duration; pETOF – passive Eustachian tube opening frequency; pETOP – passive Eustachian tube opening pressure

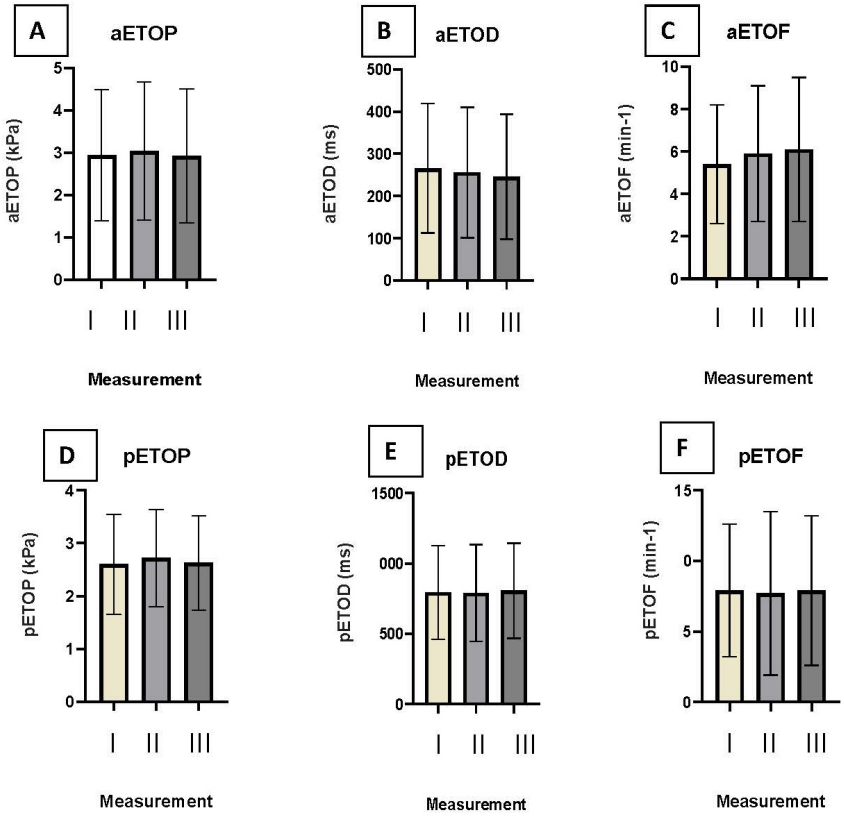
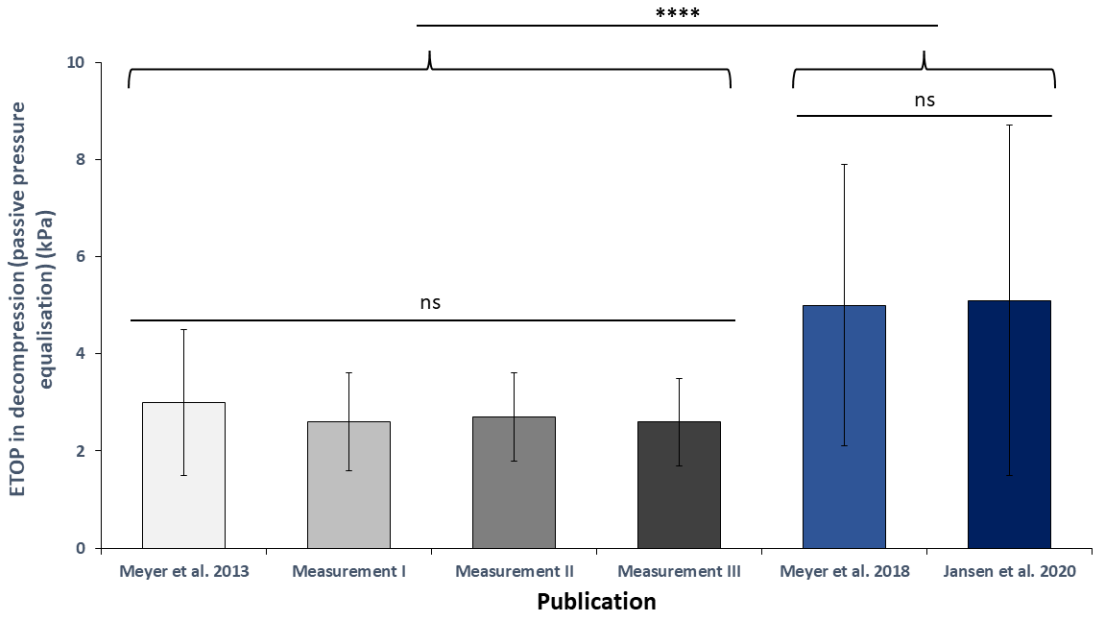


Figure 4

Comparison of mean and standard deviation ETOP during decompression (passive pressure equalisation) for healthy cohorts (Meyer, et al. 2013,[10] and present study measurements I-III), and cohorts consisting of patients with chronic ETD (Meyer, et al. 2018, Jansen, et al. 2020);[11,12] ****ETOP during decompression is significantly higher statistically in patients with chronic ETD than in healthy participants ($P < 0.0001$). ns – non-significant



and did not confirm a statistically significant difference between measurements for ETOD during compression (actively induced pressure equalisation; $F(2, 117) = 0.171$, $P = 0.843$).[27] Figure 3 gives a detailed overview.

As shown in Figure 4, an ANOVA revealed no significant differences among only healthy cohorts (measurement I-III) or among cohorts consisting only of patients with chronic ETD.[10–12] However, the ETOP during decompression (passive pressure equalisation) is significantly higher in subjects with chronic ETD than in healthy subjects ($P < 0.0001$).

Discussion

Diving results in large ambient pressure changes and there is a need for a properly functioning Eustachian tube. Probably every diver knows that there are better and worse days in terms of middle ear pressure compensation, even if the cause is not always known. Certainly, the hormonal cycle, nutritional status and colds involving the nose and nasopharynx play an important role. It would be interesting to determine whether Eustachian tube function shows differences at different times normal subjects. The test procedure used here was evaluated in preliminary studies as very reliable and dependable.[26] To our knowledge, this is the first longitudinal, dynamic and pressure chamber-based study aiming at identifying possible intraindividual ET function fluctuations in a weekly periodicity among healthy participants.

An initial question to be discussed is why ETOD during compression (actively induced pressure equalisation) differs on the right side between measurements I and III but not on the left side. If there was intraindividual variability shown for both sides, respectively, it would strongly suggest periodical fluctuations of ETOD. Considering the inconsistent results between both sides, other possible explanations are measurement inaccuracy or statistical imprecision. This hypothesis is also supported by the fact that no difference between both sides was detected for ETOF and ETOP during compression.

As stated by Tysome and Sudhoff, there is a need to enhance ET function measurement to improve the evaluation of new diagnostic approaches and therapeutic strategies for ETD.[28] For the same reasons, it is also essential to detect possible fluctuations in healthy participants. Otherwise, effects measured at a single point in time effects would be difficult to interpret due to natural range of variation in ET function. This study is a major step in the right direction, as it provides evidence that possible ET function fluctuation due to unmeasured natural factors is negligibly small in pressure chamber-based measurements. In addition, the study offers the advantage that pressure change relevant to diving can be simulated under standardised conditions, in contrast to studies in water, where a measurement under standardised conditions seems almost impossible.

Finally, there are certain limitations of this study. The interpretation of ET function-reflecting variables during

compression (actively induced pressure equalisation) continues to be challenging, as the Valsalva manoeuvre is performed heterogeneously among people.[9–12] On the other hand, on an intraindividual level the analysed curves were, subjectively speaking, strikingly similar. Apart from that, observed differences between healthy participants and patients with ETD need to be confirmed in larger cohorts and related to different subgroups. Furthermore, an interval of one or two weeks was chosen in the study. This means, for instance, that it cannot be ruled out that monthly or annual changes may occur. In addition, the individual factors influencing Eustachian tube function were not queried and analysed. For example, in further studies hormone status, weight and hydration level can be additionally examined and correlated. As pressure chamber-based measurements are not widely used, it is also essential to link the knowledge gained and findings obtained with other, more commonly available methods.

Conclusions

This study shows that in three consecutive weekly repeated measurements no relevant variations in Eustachian tube function could be detected. In fit and well subjects there was no significant week to week fluctuation in ET functions tested through continuous impedance measurements in a pressure chamber.

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Review articles

The risk of decompression illness in breath-hold divers: a systematic review

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Abstract
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Introduction: Breath-hold (BH) diving has known risks, for example drowning, pulmonary oedema of immersion and barotrauma. There is also the risk of decompression illness (DCI) from decompression sickness (DCS) and/or arterial gas embolism (AGE). The first report on DCS in repetitive freediving was published in 1958 and from then there have been multiple case reports and a few studies but no prior systematic review or meta-analysis.
Methods: We undertook a systematic literature review to identify articles available from PubMed and Google Scholar concerning breath-hold diving and DCI up to August 2021.
Results: The present study identified 17 articles (14 case reports, three experimental studies) covering 44 incidences of DCI following BH diving.
Conclusions: This review found that the literature supports both DCS and AGE as potential mechanisms for DCI in BH divers; both should be considered a risk for this cohort of divers, just as for those breathing compressed gas while underwater.

Introduction

Breath-hold (BH) diving is distinguished by the combination of immersion of the diver's face and voluntary breath-holding or 'apnoea'. It is practiced for several reasons, including the collection of food or resources (e.g., shellfish, pearls), spearfishing, recreational diving, and competitive apnoea diving. Most of these BH dives are made in relatively shallow water, although spearfishing divers and competition divers do make deeper excursions, with the world record standing at 214 metres for a 'no limits' freediving competition.[1] The Guinness world record for static apnoea (breath holding only) stands at 11:54 minutes.[2] By comparison, traditional food gatherers such as the Japanese Ama tend to make many dives in a day but to a maximal depth of only ~20 msw and for around one minute at a time.[3]

The risks of BH diving of all types include barotrauma, drowning, hypothermia, marine animal injury, hypoxia/shallow water blackout, and decompression illness (DCI) from arterial gas embolism (AGE) and/or decompression sickness (DCS). Given the short BH periods and depths involved for traditional BH diving, it was long held that DCS was unlikely to occur in these divers although this attitude has now changed, particularly as longer, deeper repetitive dives are now made more commonly.[4]

Decompression sickness arises when inert gas (for example nitrogen) is accumulated at depth and becomes supersaturated in the tissues, then forming bubbles upon ascent from the dive. Historically, it was believed that BH dives such as those made by food gatherers, were not deep or of long enough duration for inert gas tissue pressures to become raised to levels capable of causing DCS.[4] However, as early as 1958, Taravana syndrome, which is a collection of neurological symptoms such as vertigo, nausea, paralysis and unconsciousness, was noted in pearl dives who made up to 60 dives a day in French Polynesia.[5] At this time, there was some acceptance that BH dives to extreme depths, with greater frequency and duration than commonly used while food gathering could prove problematic; this acceptance is particularly pertinent for certain classes of competitive BH diving with the aim of diving as deep or for as long as possible.[4,6,7] Although Lanphier was one of those sceptical of BH diving causing DCS, in 1965 he made calculations on probable nitrogen tissue tensions for a series of BH dives using the United States Navy (USN) decompression tables. These calculations determined that the ratio between surface

interval (S) (to wash out nitrogen) and time at depth (D) regulates nitrogen accumulation.[4,6] When the ratio of these parameters (S/D) is 1, he calculated that no limits need be imposed and the dive was safe, but at a ratio of 0.5 then there was a risk of DCS in < 3 h; values below 1 implied an increasing level of risk with decreasing value. Other workers have also calculated the theoretical risk of breath-hold DCS, with one study concluding that symptomatic nitrogen supersaturation can be reached during BH diving, again with surface interval playing an integral role.[8]

Fitz-Clarke developed a model for BH DCS using documented human BH dives to 100 m or greater, which determined that predicted DCS risk is negligible in a single dive up to around 100 msw.[9] All of these findings indicate that there is more risk to a diver making multiple BH dives, but DCS has also been seen in single, deep dives at depths usually over 100 m.[9] Decompression sickness has been identified in most groups of BH divers, and it is now generally accepted that BH diving can cause neurological DCS following deep and repetitive dives, with most cases affecting the brain.[10] Certainly, competitive apnoea divers and organisations such as International Association for Development of Apnea [AIDA] or the World Underwater Federation [CMAS] are aware of the risk of DCS in their sport and offer oxygen breathing at depth (~5 m) post BH diving in competitions, in an effort to mitigate risk.

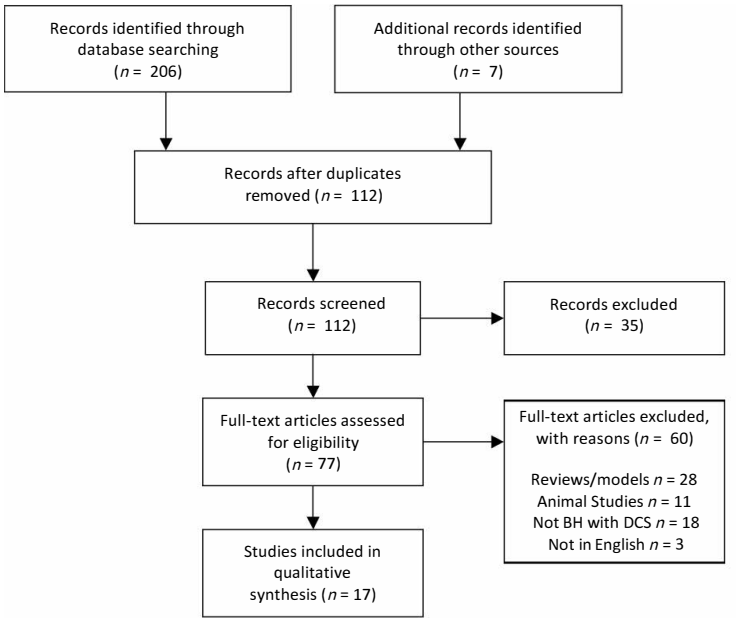
The Divers Alert Network (DAN) collects data on diving incidents across the world.[11–14] In their most recent report (2020, in press), several BH diving fatalities were reported but none of these involved DCS. However, a BH diver in the Caribbean was treated recently with hyperbaric oxygen after suffering post-dive neurological symptoms. Initially the diver was denied treatment, as attending medics did not acknowledge DCS in freedivers but after contact with hyperbaric physicians via DAN, treatment was administered (personal communication with Dr James Chimiak, DAN USA). This shows that awareness of the possibility of DCS/DCI occurring following a BH dive still needs to be improved.

Reviews on this subject have been written in the past, but to date there are no systematic reviews or meta-analysis of BH DCI cases and studies in the literature. To enable future studies elucidating the risk of AGE vs DCS in breath-hold diving and potential guidelines for safer diving we aimed to consolidate the relevant literature with this review.

Methods

A preliminary literature search was carried out (search date 23 March 2021) in PubMed to identify all terms and keywords. A systematic literature search following PRISMA Guidelines was then performed (23 August 2021). Searches were carried out on PubMed and Google Scholar. Search terms included "*breath-hold*", "*breath-hold AND diving*", "*breath-hold AND DCS*", "*apnea/apnoea AND DCS*", "*breath-hold AND diving AND DCI*", "*Taravana*", "*Taravana AND DCS*", "*breath-holding AND DCS*", "*breath-hold AND diving AND DCI*", which yielded 206 results (Figure 1). Seven additional records (excluding duplicates) were found via Google Scholar or were known personally (conference abstracts, references from references). The final selection had all duplicates removed and was limited to studies or abstracts in English. All reviews and animal studies were excluded. In total, 17 studies were found to be of relevance (Figure 1), that is they included reports of cases of breath-

Figure 1
Flow chart of the study selection process; BH – breath-hold; DCS – decompression sickness



hold diving with DCS, or experimental work investigating breath-holding and DCS.

DATA EXTRACTION

Data extracted (if present) from case reports or experimental studies identified in the search that either reported DCI in BH divers or carried out experimental trials to investigate the likelihood of DCS in BH dives included: the number of cases, the number of individuals affected, bubble grades recorded, sex and age of the diver, location, symptoms, diagnostic tools used, diagnosis (type of DCI - DCS, AGE, symptoms), treatment, dive time (period over which dives were carried out) number of dives made, maximum depth dived, maximum individual breath-hold period, and type of BH dive – if competitive BH then which category were the divers taking part in.

Results

OVERVIEW OF STUDIES

Of the 17 articles included in the final selection, 14 were case studies and three were experimental studies; two of the case studies were performed by the same group[15,16] and used the same data for analysis and thus, were treated in our analysis as a single study. The articles were published between 1965–2020, of which 15 (88%) were published in or after 2000. The data extracted from these 17 articles noted 44 cases of DCI following BH diving, 43 of these were observed in men (two studies[15,16] do not specify sex), and one case in a woman. Of the clinical case studies, 13 of the 15 detailed that they used imaging (magnetic resonance imaging [MRI] or computed tomography [CT] scans) to investigate the illness. Table 1 shows the characteristics of the studies, including subject information, dive profile and type and diagnosis made, while Table 2 lists the symptoms noted in the studies.

CASE REPORTS (SEE TABLES 1 AND 2)

Accurso et al. 2018[17] – maximum depth 40 metres of sea water (msw)

This paper describes two cases of taravana syndrome (defined in the manuscript as neurological DCS) in the same male diver, age 38 years. The diver had no existing illnesses, did not smoke, and had been spearfishing for 10 years. Further details of his dives are shown in Table 1. In both cases, CT imaging found hypodense areas, in the right internal capsule following dive 1, and in the nucleus of the right thalamus associated with oedema following dive 2. Amongst other laboratory tests, thrombophilic screening was carried out; a high level of homocysteine (28.59 $\mu mol.L^{-1}$) was found. The diver was treated with three recompression sessions on US Navy Table 6 and acetylsalicylate acid 100 mg daily was prescribed as a prophylactic treatment. He was discharged after ten days and dived after four months with no further incidents reported. The authors concluded that their findings suggested that a thrombophilic state due to hyperhomocysteinaemia might be associated with taravana syndrome in BH divers and should be investigated further.

Alaimo et al. 2010[18] – maximum depth 24 msw

This study reports the case of a 41-year-old diver who also suffered from taravana syndrome (Table 1), presenting with various neurological symptoms including (Table 2): vertigo, ringing in the left ear, confusion, paraesthesia of the right arm, and right hemiparesis. A CT scan showed four cerebral bubbles, three in the left hemisphere and one in the right at the level of the internal carotid and ophthalmic arteries. The diver was rehydrated, administered parnaparin and hyperbaric oxygen treatment (HBOT); thereafter the symptoms resolved, and a new CT scan confirmed the disappearance of the bubbles. The authors claimed that this was the first report of the documented presence of bubbles in a patient with taravana syndrome, and this supported the hypothesis that nitrogen bubbles were responsible for symptoms in these cases.

Batle 2000 a and b[15,16] – maximum depth unknown

These abstracts outline a study on 28 patients who suffered from BH diving accidents and were treated at the MEDISUB Hyperbaric Research Institute in Mallorca, Spain, between 1995–1999. The authors reported an increase in the number of incidents over that period. The divers all had neurological symptoms immediately on surfacing and once at the unit, all were given a physical examination and full clinical history taken. All were given a CT scan of the brain, the treated with USN Tables 5 and 6 until symptoms were resolved with another CT scan given several days later to confirm resolution of changes. The authors suggested that following BH diving, any resulting gas emboli would be apparent at the level of the brain. The author also went on to examine the surface intervals made by the divers in an attempt to calculate how long an interval should last in a BH diver in order to avoid the evolution of arterial bubbles but no data or results on minimum surface interval thresholds were provided.

Cortegiani et al. 2013[19] – maximum depth 30–35 msw

This report focused on a male BH diver who was 57-years-old and an experienced underwater spearfishing champion. He presented with neurological disorders including dizziness, sensory numbness, blurred vision, and left frontoparietal pain, after several dives to 30–35 msw with short surface intervals (Table 1). His symptoms spontaneously resolved but the following morning the symptoms reoccurred, so the diver returned to the hospital where he suffered a tonic-clonic seizure and lost consciousness. Magnetic resonance imaging revealed a cortical T1-weighted hypointense area in the temporal region corresponding to infarction with partial haemorrhage. He was then treated by HBOT, and the symptoms resolved swiftly. The authors concluded that

Table 1

Details of studies included in the qualitative synthesis; [*]accumulated time spent underwater across all dives made; [a]same patients as previous Batle reference; [b]mean value; [c]eight repetitive dives; [d]long BH sessions to recreate spear fishing sessions; [e]dives made over 3 days; [f]median value; [g]ratio surface to underwater time. # refers to diver number, where data for more than one diver is reported; CAGE – cerebral arterial embolism; DCI – decompression illness; DCS – decompression sickness; F – female; M – male; NS – not specified; Ref – reference; Rep – repetitive

Ref	*n*	Cases	Sex	Age (y)	Location	Clinical or exp.	Diagnosis	Total dive time (h:min)*	Number of dives	Max depth (m)	Max BH time (min)	Surface interval	Activity
17	2	2	M	38	Italy	Clinical	Both taravana (neurological DCS)	#1: 7 #2: 4	#1: 100–120 #2: 60	40	1.4	–	Spearfishing
18	1	1	M	41	Italy	Clinical	Taravana (neurological DCS)	5	60–80	24	–	–	–
29	11	0	M	–	Croatia	Exp.	–	Rep[c] 1, Deep[d] 6	Rep 8, Deep – unlimited	35 & 40	–	Rep – 3 min, Deep – not restricted	Spearfishing (simulated)
15	28	28	–	–	Spain	Clinical	Neurological DCS	–	–	–	–	–	–
16	28	28[a]	–	–	Spain	Clinical	Neurological DCS	3–8	~15·h^{-1}	63	2	–	Spearfishing, often with scooters
30	10	0	M	29[b]	Spain	Exp.	–	4:03[b] (2–6)	–	35[b] (24–40)	–	–	Deep harpoon fishing event
32	1	0	M	53	Italy	Exp.	–	–	–	40.2[f] (6.2–41.7)	140 s (± 42)	9.8 min (± 9) 3:4	Spearfishing
19	1	1	M	57	Italy	Clinical	Taravana (neurological DCS)	2:30	19	30–35	2:50	1–1:30 min	Spearfishing
20	1	1	M	21	France	Clinical	Neurological DCS	1–1:30	10–12	18	2	5–6 min	BH prep for ships diving course
21	1	1	M	39	Portugal	Clinical	Neurological DCS? Left frontal white matter lesion	5	–	30	2	“Short”	Sports?
22	2	2	M	33, 39	Japan	Clinical	Neurological DCS	~5–6	–	#1: 22, #2: 23	#1: ~1–1:30, #2:	Both ~ 1 min	Ama – food gathering, weight assisted

Table 1 continued

23	1	1	M	65	Japan	Clinical	CAGE (lung overexpansion) leading to cerebral DCI	~3	–	20	–	–	Ama – food gathering, weight assisted
24	2	2	M	-	Japan	Clinical	Cerebral DCS	#1: rep dives #2: 6	–	#1: 20, #2: 30	–	–	Free divers
25	1	1	M	-	Norway	Clinical	Limb/chest/abdominal pain and neurological DCS (paraesthesia)	5	60	20	2	Few seconds to 1–2 min	Submarine escape training – 'bottom drop'
26	1	1	M	34	Japan	Clinical	Neurological DCS	–	–	25	1:20	20–30 s	Ama – food gathering, weight assisted
27	1	1	M	31	Germany	Clinical	CAGE/stroke	–	3	100	4	15 min	Competitive free diver
28	1	0	F	74	Japan	Clinical	DCS (endogenous cerebral ischaemia)	3	–	5	–	–	Ama diver

the diver had suffered from taravana syndrome, although the reappearance of symptoms after the initial resolution was atypical.

Gempp et al. 2006[20] – maximum depth 18 msw

This case reported a 21-year-old man who complained of various neurological symptoms two hours after a series of BH dives made over approximately one hour. The diver was a sailor in the French navy, was healthy, did not smoke, and had been practicing BH training for the previous three months. Symptoms included: dizziness, visual disturbance, tightness of the chest accompanied by dyspnoea, flushed face, and numbness of all limbs involving the right side of the face. Before transferal to a HBOT unit, the diver was administered normobaric oxygen and on arrival the symptoms had resolved. The patient was treated for neurological DCI using HBOT, and given intravenous rehydration (Ringer lactate, 1 L), aspirin (250 mg), and buflomedil (400 mg) orally. Contrast transcranial Doppler ultrasound revealed appearance of bubbles, suggesting that the patient had a large right-to-left shunt most likely due to a patent foramen ovale, although a pulmonary shunt remained a possible cause. No lesions were detected in the brain upon MRI. The patient was advised to stop scuba diving and to avoid repetitive deep dives with short surface intervals. The authors advised that anyone who experiences unusual symptoms after BH diving should seek immediate medical attention due to the risk of DCI.

Guerreiro et al. 2018[21] – maximum depth 30 msw

In this case, a 39-year-old male who had performed 30 extreme BH dives (two minutes in duration to approximately 30 metres over five hours with short surface intervals) developed transient expressive aphasia and headache. On examination with MRI, he exhibited a left frontal white matter lesion. He was given three sessions of HBOT and at one month follow-up, the patient was asymptomatic, and MRI was clear. The authors noted that accumulation of nitrogen in blood and tissues after repetitive BH diving has been suggested to cause endothelial dysfunction and disruption of the blood-brain barrier, with subsequent hyperpermeability in microvasculature and vasogenic oedema.

Kohshi et al. 2000[22] – maximum depth 22 and 23 msw

This paper reports on two cases, both professional (Ama) male divers of 33 and 39-years-old who presented with neurological symptoms following repetitive BH dives (Tables 1 and 2). Both were healthy but smoked. Diver One had a sudden onset of dizziness and blurred vision; MRI of the brain on the 4th day after the onset showed two hyperintense cerebral lesions in the left occipital lobe and right basal ganglia. He was treated with dexamethasone (12 mg i.v. daily for three days), followed by drug tapering of 4 mg daily over three days. His vision improved over

Table 2

Symptoms and findings reported following breath-hold dives in the 18 studies included in the review; CT – computed tomography scan; EB – Eftedal-Brubakk bubble grade; MRI –magnetic resonance imaging scan; NA – not available

Symptoms	Cases	Reference(s)	Time of symptom onset (by reference number)
Neurological symptoms (not specified)	28	15	15, NA
Vertigo/dizziness	9	18–20; 22; 24–26	18 NA; 19 while diving; 20, two hours after diving; 22, while diving; 24, after diving; 25, while diving; 26, immediately after diving
Limb paraesthesia	6	17; 18; 20; 23; 25; 26	17, immediately after diving; 18 NA; 20, 2 h after diving; 23, immediately after diving; 25, 2 h after diving; 26, while diving
Visual disturbances	6	19; 20; 22; 24–26	19, while diving; 20, two hours after diving; 22, while diving; 24, after diving; 25, 2 h after diving; 26, immediately after diving
Facial paraesthesia	5	17; 20; 26	17, immediately after diving; 20, 2 h after diving; 26, NA
Hemiparesis/hemiplegia	5	18; 22; 27; 28	18 NA; 22 NA; 27, 3 h after surfacing; 28, while surfacing
Non-specified paraesthesia/sensory disturbance	4	19; 22; 24; 27	19, while diving; 22, while diving; 24, after diving; 27, 3 h after diving
Gait disturbances	4	23; 24; 26	23, 1–2 h after diving; 24, after diving; 26, immediately after diving
Asthenia (weakness)	4	17; 22; 25; 27	17, immediately after diving; 22, while diving; 29, within 30 mins of diving; 27, upon surfacing
Nausea	3	24–26	24, after diving; 25, while diving; 26, immediately after diving
Dyspnoea/ chest tightness or pain	3	20; 24; 25	20, 2 h after diving; 24, after diving; 25, 2 h after diving
Speech impairments/aphasia	2	23; 27	23, immediately after diving; 27, upon surfacing
Buccal rhyme deviation	2	17	17, 24 h after diving
Ringing in ears	1	18	18 NA
Confusion	1	18	18 NA
Headache	1	19	19, while diving
Tingling	1	17	17, immediately after diving
Limb pain	1	25	25, 30 mins after diving
Seizure	1	19	19, day after diving
Cerebral palsy	1	27	27, 3 h after surfacing

Diagnostic/laboratory test results			Imaging		
Abnormal heart rate	1	19		Cases	Reference(s)
Abnormal blood count workup	1	17	MRI – lesions in the brain	10	17; 19; 21–24; 26; 27
Abnormal Glasgow coma scale	1	19	CT – brain, hypodense areas	4	17–19; 27
Abnormal oxygen saturation	1	19	CT – pulmonary, ground glass pattern	1	19
Abnormal blood gases	1	19	Echocardiogram (cardiac bubbles)	1 (EB grade 4 max)	32

the next three weeks, but he still had residual right lower quadrantanopsia. At four weeks, his MRI was normal. Diver Two was admitted with moderate right hemiparesis and a hemisensory disturbance; upon MRI examination on the third day after onset three hyperintense cerebral lesions were observed. This patient received daily HBOT for four days with signs of improvement, and MRI made two weeks later showed a reduction of the hyperintensities in the left parietal and basal ganglia but not the lesion in the right frontal lobe. Upon discharge, he retained some numbness in his right upper arm. The authors concluded that diving accidents such as those reported here had not occurred in Japan before this date (2000) and this may be due to transience of symptoms in many cases or the possibility that the Japanese Ama communities keep such accidents secret.

Kohshi et al. 2020[23] – maximum depth 20 msw

This report focused on a 65-year-old male Ama fisherman who was in good health and had started diving at the age of 30. At the end of his morning dive shift, the diver experienced slurred speech and right-handed paraesthesia, and he was unable to walk properly. The diver received an MRI within two hours of the accident; hyperintense areas on fluid-attenuated inversion recovery (FLAIR) and diffusion weighted imaging were shown in the pons and right-sided parietal lobe. Diagnosis was given as hyperacute pontine ischaemia caused by AGE and a subacute ischaemic lesion in the parietal white matter. The diver was given HBOT in a monoplace chamber and received intravenous rehydration (Ringer's lactate, 1,000 mL). His gait disturbance resolved the next day and HBOT was continued over the next seven days; he was discharged with some residual numbness in his hand, which had resolved on follow-up at five months. A follow-up MRI showed a reduction of the pontine hyperintensity area but no dramatic change in the right parietal lesion. The authors concluded that repetitive BH dives tend to induce stroke-like neurological disorders which are occasionally serious.

Matuso et al. 2014[24] – maximum depth 20 and 30 msw

This paper reported two cases of BH divers presenting with cerebral DCS, who were then investigated with MRI to demonstrate distinctive characteristics of this condition. The first diver presented with right hemiparesthesia, diplopia, and gait disturbance after BH diving to a depth of 20 msw. Neurological exam revealed: left abducens nerve palsy, right-sided sensory disturbance, dysmetria, and ataxic gait. Upon MRI, multiple hyperintense lesions in the right frontal lobe, bilateral thalamus, pons, and right cerebellar hemisphere were observed and the diver was treated with HBOT. Two weeks later, all symptoms had improved, and the MRI findings were attenuated. The second patient had made BH dives to 25–30 msw over a period of six hours. He presented with left quadrant hemianopia and an unstable gait. Upon MRI, hyperintense areas in the bilateral occipito-parietal lobes were seen. This patient also received HBOT, and after three weeks his neurological symptoms disappeared and multiple hyperintense lesions on MRI were attenuated. The authors concluded that in these cases, vasogenic oedema had caused cerebral DCS and that MRI is often more useful compared with other imaging modalities for the examination of patients with DCI. In particular, they noted that AGE can be differentiated from DCS with the use of diffusion weighted imaging and apparent diffusion coefficient mapping.

Paulev 1965[25] – maximum depth 20 metres of fresh water (mfw)

In this paper, Poul-Erik Paulev reported on his own experience following repeated BH dives made to 15–20 m in the Norwegian Navy escape-training tank. Paulev made BH dives over a total period of about five hours; during the last two hours of diving, he experienced some nausea, dizziness, and eructation. Thirty-minutes after the last dive he presented with pain in the hip, right knee, and general fatigue and weakness in the whole right side of his body. Two hours after surfacing he also experienced severe chest pain, paraesthesia of the right hand and blurred vision; one hour later a colleague found him overtly pale and weak, and he was recompressed on US Navy Table 3. Following this treatment, all symptoms resolved bar some residual weakness in his right hand. Paulev reported that there were three similar cases experienced by the training tank training staff within a year prior to his accident. In each, neurological symptoms were present and were relieved by HBOT. Paulev concluded that there was little reason to question that these cases were anything other than DCS.

Tamaki et al. 2010[26] – maximum depth 25 msw

This case study reports on a 34-year-old Japanese Ama diver who had developed neurological symptoms during repetitive dives to 22 msw. His diving pattern generally involved dives of 40–80 s duration with 20–30 s surface intervals over a period of around six hours. After around two hours in the water, he noticed paraesthesia in his right hand, which did not worsen. At the end of his shift, he reported symptoms of nausea, dizziness, and double vision, with some disturbances in gait. Arriving home, he had paraesthesia in his cheek and toe on the right side of his body. With no abatement of symptoms, the next morning he went to hospital. Upon MRI, five hyperintense lesions were found in the right and left basal ganglia, right frontal lobe, pons, and right cerebellar hemispheres. He was treated with HBOT on US Navy Table 6 with no resolution of symptoms; he was treated daily thereafter for ten days. His symptoms gradually resolved, though the residual numbness in his hand persisted for nearly a year. The authors recommended that as the Ama divers tend to harvest the deep ocean floor, longer surface intervals should be taken to prevent DCS; they noted that in this case the diver tended to perform shorter surface intervals

than most Ama divers (20–30 s), although his dive depth and duration were similar.

Tetzlaff et al. 2016[27] – maximum depth 100 msw

In this case, a healthy, non-smoker, 31-year-old competitive BH diver was treated for motor weakness in his right arm and difficulty speaking following a 100 msw training dive; this was the third of three dives with a BH time of four minutes for each. He was administered normobaric oxygen en-route to the hospital, where right-sided hemiplegia, hypesthesia, cerebral palsy and aphasia were noted. Upon CT imaging, an ischaemic lesion in the left frontotemporal region was observed. Heparin was given along with HBOT on US Navy Table 6, with a second session performed the following day with no noticeable improvement. After repatriation, HBOT continued over seven sessions; MRI findings noted a large ischaemic lesion in the area of the left medial cerebral artery. Following treatment, and rehabilitation over several months, paresis of his right leg and hypesthesia improved but he could not return to his profession as a wood worker. The authors concluded that this case provided evidence that serious neurological injury after deep BH dives may occur more often than previously thought.

Yanagawa et al. 2018[28] – maximum depth 5 msw

This case focused on a 74-year-old Japanese female Ama diver who developed hemiparesis on ascent from a BH dive to 5 msw, following multiple dives made over three hours. She had hypertension that was medicated. On examination with echocardiography one hour after her accident, no bubbles could be observed in her inferior vena cava and she was diagnosed with endogenous cerebral ischaemia, not induced by DCS or AGE. On arrival at hospital various tests including MRI confirmed the initial diagnosis, and she received antiplatelet therapy. The authors comment on the usefulness of ultrasound for on-site differential diagnosis.

EXPERIMENTAL STUDIES (SEE TABLES 1 AND 2)

Barak et al. 2020[29] – maximum depth 35 and 40 msw

This study investigated the multifactorial nature of DCS specifically in BH diving, investigating causes other than bubbles. In protocol one, eleven BH divers performed eight deep (35 msw) dives with surface intervals of three minutes and cumulative BH time of 12 minutes. In protocol two, the same divers participated in a six-hour BH session, diving multiple times throughout to depths between 15–40 msw; surface intervals were not dictated. Endothelium-dependent vasodilation of the brachial artery, via flow-mediated dilation (FMD), and the number of microparticles (MPs) were assessed before and after each protocol. Absolute FMD was reduced following both diving protocols ($P < 0.001$), and there was a difference in the number of MPs produced between protocols ($P = 0.007$), with both increasing post-dive. The authors concluded that both protocols, which represented deep or repeated BH dives, seemed to cause endothelial dysfunction that may play an important role in neurological DCS (in particular, stroke-like symptoms) in addition to bubbles.

Boussuges et al. 1997[30] – mean depth 35 msw

This study had the simple aim to detect any circulating bubbles after BH diving in spearfishermen. Ten BH divers took part while participating at a deep harpoon fishing event in Minorca, Spain, in 1995. Bubbles were monitored with continuous wave Doppler over the left chest immediately after the last dive and around 30 minutes post-dive. Two-dimensional echocardiography was also used, obtaining images from the parasternal view, again immediately after diving. The mean maximum depth achieved by the ten divers was 35 msw (range 24–40 msw) for a mean duration of 4 h 03 min (2–6 h). No evidence of circulating bubbles was found with either technique, despite the relatively 'aggressive dives'. However, the authors noted that their study was limited both by the number of subjects, and that their measurements may not have been comprehensive enough to detect any bubbles that evolved. They noted that dives published in the literature suggested the onset of neurological DCI after BH diving often involved underwater scooters, delivering divers to deeper depths with longer bottom times due to the swift movement up and down of the divers to their target.[31]

Cialoni et al. 2016[32] – median depth 40.25 mfw

This study focused on one 53-year-old male spearfishing BH diver to investigate bubbles loads following BH dives. Transthoracic echocardiography was performed on the diver 15 minutes before diving and at 15-minute intervals for 90 minutes after diving in a 42-meter-deep pool, who determined his own diving pattern. Median depth was 40.25 mfw (range 6.2–41.7 mfw), mean diving time was 140.9 (SD 42.1) s and mean surface interval was 593.8 (SD 540) s, with a surface interval ratio of 3:4. Over the first 45 min post-dive, high bubbles loads were observed in all images at grade 4 Eftedal Brubakk (Scale from 1–5; 1 equates to a very low bubble load in the heart, 5 is a very high load with the heart obscured by bubbles in the field). At 60 mins to 90 mins, the load gradually decreased to Eftedal Brubakk grade 1. The authors concluded that despite the limitation of investigating one diver only, high bubble grades can occur after BH diving and that ordinary methods to predict inert gas supersaturation may not be able to predict taravana syndrome cases.

Discussion

Upon systematic review of the literature for studies reporting a convergence of breath-hold diving and DCI, only 14 clinical and three experimental papers of relevance were

found. The majority (10) of these reports related to spear fishing and food gathering, which prior to the advent of competitive freediving would naturally make up the bulk of this type of diving. As reports are few, it was interesting that in the case study reported by Kohshi,[22] the authors noted that diving accidents involving taravana syndrome / cerebral DCI had not occurred in Japan before 2000 to their knowledge; they explained this may be due to symptom transience/ spontaneous resolution but they did also acknowledge the possibility that the Japanese Ama communities keep such accidents secret.

It is of note that only one study reported on a freediving accident, given that competitive apnoea and freediving is now more common.[27] However, there does seem to be much in the way of anecdotal data involving freedivers, as evidenced by the database created to investigate the risk of DCS in extreme BH dives by Fitz-Clarke, who gathered information from 'reliable sources', including websites maintained by champion divers. These data identified 192 dives to 100 m or deeper with two cases of DCS recorded.[9,10] Perhaps some impetus should be made to follow competitive freedivers and publish findings if more is to be learned on this subject. It would be helpful if the BH apnoea diving community could be made aware that in gathering data, the safety of their sport would likely improve as researchers would be able to optimise techniques/protocols to aid this. For example, several of the studies reported here discuss the surface intervals employed by the afflicted divers, reporting that they were thought to be inadequate. They go on to recommend that the surface interval be increased to improve their safety in the future. Table 1 reports all surface intervals where available and shows great variation, from 'a few seconds', to 20–30s, to 5–6 mins; all of these for dives of not dissimilar depths (18–25 m), although the task undertaken during the dive might be very different. It is reassuring, however, to note that the deepest dive reported (100 m) does cite the longest surface interval at 15 mins. The importance of an appropriate surface interval between BH dives for the safety of divers should not be underestimated and is an increasingly common safety strategy used by BH divers. If appropriate data were gathered on this parameter from apnoea competitions/training sessions, then we would be able to offer the participants meaningful and precise advice.

Of the 14 case studies included here, eight made a diagnosis of cerebral DCS, and four mentioned DCI or AGE. In the remaining cases, one gave a diagnosis of endogenous cerebral ischaemia, caused by existing hypertension;[28] and the other, although not giving a diagnosis of DCS, suggested that nitrogen in the body after repetitive BH could cause endothelial dysfunction and disruption of the blood–brain barrier, with subsequent hyperpermeability in the microvasculature and vasogenic oedema. However, a recent BH study has suggested that disruption to the blood-brain barrier after static apnoea is transient and minor in nature and found that apnoea did not show any indication of neuronal damage.[21,33]

There is consensus in the scientific literature that BH-divers can suffer neurological symptoms after diving but to what extent this attributable to DCS or AGE (thus a decompression illness [DCI]) is not completely understood.[4,20,22,27,34–38] As mentioned previously, theoretical calculations have been made that suggest over the course of a bout of repetitive diving, nitrogen supersaturation and thus the evolution of bubbles in the circulation and tissues is possible, with any resulting illness identifying DCS as the likely culprit.[6,39] However, a single deep dive could result in AGE, possibly from expansion from a collapsed state of lung compression and pulmonary atelectasis, causing overexpansion of regional lung segments during ascent. If one part of the lung does not open up, other parts will suffer from the gas expanding to volumes beyond the anatomical limits in that region. In an experimental simulation of lung volumes below residual volume MRI with hyperpolarised gas has showed regional bronchial collapse in volunteer elite divers.[40]

The risk of AGE from a single deep dive could potentially be even higher if the diver uses glossopharyngeal insufflation to overfill the lungs prior to diving, as this manoeuvre has been shown to cause transient neurological symptoms suggestive of AGE when performed dry and in water.[41]

Conclusions

In summary, this review finds that the literature supports both DCS and AGE as potential causes of DCI in BH divers; both should be considered a risk for this cohort of divers, just as for those breathing compressed gas while underwater. The review has identified some gaps in the literature for future study, especially the optimisation of surface interval periods, but also the lingering question whether single dive DCI is AGE or DCS. We also suggest using DCI as terminology when the underlying pathology is unclear, as DCI should cover DCS, AGE and the older terminology of Taravana syndrome.

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Safety and performance of intravenous pumps and syringe drivers in hyperbaric environments

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Abstract
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Introduction: Critically ill patients require continuation of their care when receiving hyperbaric oxygen treatment. This care may be facilitated via portable electrically powered devices such as intravenous (IV) infusion pumps and syringe drivers, which may create risks in the absence of a comprehensive safety evaluation. We reviewed published safety data for IV infusion pumps and powered syringe drivers in hyperbaric environments and compared the evaluation processes to key requirements documented in safety standards and guidelines.
Methods: A systematic literature review was undertaken to identify English language papers published in the last 15 years, describing the safety evaluations of IV pumps and/or syringe drivers for use in hyperbaric environments. Papers were critically assessed in relation to the requirements of international standards and safety recommendations.
Results: Eight studies of IV infusion devices were identified. There were deficiencies in the published safety evaluations of IV pumps for hyperbaric use. Despite a simple, published process for evaluating new devices, and available guidelines for fire safety, only two devices had comprehensive safety assessments. Most studies focused only on whether the device functioned normally under pressure and did not consider implosion/explosion risk, fire safety, toxicity, oxygen compatibility or risk of pressure damage.
Conclusions: Intravenous infusion (and other electrically powered) devices require comprehensive assessment before use under hyperbaric conditions. This would be enhanced by a publicly accessible database hosting the risk assessments. Facilities should conduct their own assessments specific to their environment and practices.

Introduction

Comprehensive hyperbaric facilities are capable of providing in-chamber intensive care to patients who are critically ill from a wide range of causes.[1] In such facilities, hyperbaric oxygen (HBO) treatment may be delivered to critically ill patients suffering from necrotizing fasciitis, gas gangrene, arterial gas embolism, decompression sickness or carbon monoxide poisoning, among other indications.[2,3] Much of the supportive care provided to such patients is facilitated via portable electrically powered devices such as intravenous (IV) infusion pumps and syringe drivers which are essential for the delivery of certain medications at precise infusion rates.[4] Depending on the complexity of the critical care needed, patients may require infusions via multiple devices to maintain physiological stability.

The Undersea and Hyperbaric Medicine Society (UHMS) has published recommendations for assessing the safety of portable devices before their use in the hyperbaric environment.[5] These require that all portable medical electrically powered equipment taken into hyperbaric chambers: (i) are not at risk of explosion or implosion; (ii) do not pose a fire risk; (iii) contain no toxic material; (iv) are oxygen compatible; (v) will not be damaged by pressure; and (vi) must function normally under pressure.[5–7] Equipment is deemed safe and serviceable only if it conforms to these criteria and can successfully perform its intended function under expected conditions, including the required pressure and oxygen concentration, and does not produce excessive heat or contain ignition sources. Specifically, the National Fire Protection Association (NFPA) 99 Health Care Facilities Code, 2021 Edition, Chapter 14, details requirements for portable patient care devices (Section 14.2.9.3.16) for both battery-operated and cord-connected devices, categories under which IV pumps and powered syringes fall as therapeutic patient-related electrically powered equipment.[8]

In order for critically ill patients to safely receive HBO treatment, it is imperative that IV infusion pumps and syringe drivers must be safe and able to deliver accurate doses of medication and maintain appropriate flow rates in a pressurised environment. Most available infusion pumps

are not approved for hyperbaric applications, although there are exceptions. Most have not undergone independent hyperbaric safety assessment, even if approved by the Food and Drug Administration (FDA) in the USA.[9] Moreover, some pumps may not function at all under hyperbaric conditions or might experience technical problems affecting their accuracy.[10]

According to a preliminary review of the literature, very early papers on this topic focused primarily on pump function and physical integrity or registration rather than safety, for example, assessment of electrical and fire risk received limited attention.[10–12] One study assessed the function of 29 pumps under hyperbaric conditions, but did not propose a process for safety risk assessment prior to chamber entry.[10] Another study noted that only one syringe pump used in European hyperbaric chambers had received *Conformité Européene* (CE) certification indicating that it complied with the safety standards outlined by the European Medical Device Directive (MDD 93/42).[11]

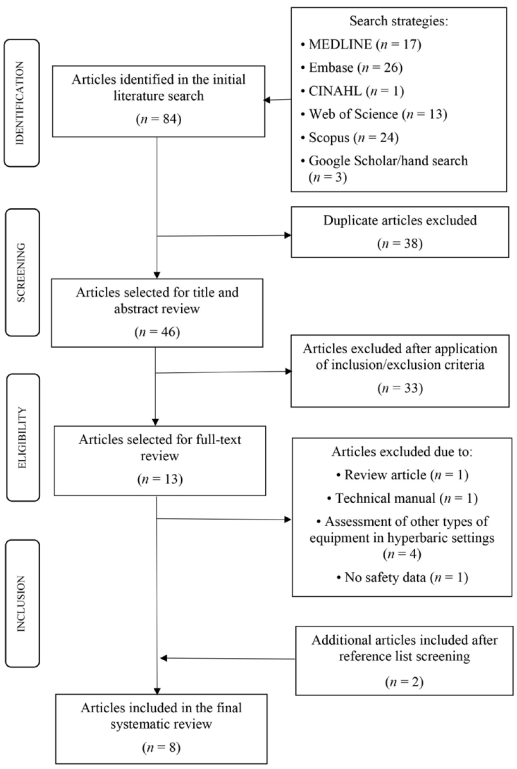
This paper aimed to review published safety data for IV infusion pumps and powered syringe drivers in hyperbaric environments and to compare the safety evaluation processes to key guidelines found in the Australian and New Zealand Standards 4774.2, UHMS processes, NFPA 99 Health Care Facilities Code and NFPA 53 recommended practice for oxygen-enriched environments.[6–8,13,14]

Methods

A systematic literature review was undertaken to identify papers describing the assessment of IV pumps and/or syringe drivers for use in hyperbaric environments, focusing primarily on safety and mitigation of fire risk. The review was conducted in line with the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) guidelines.[15] The inclusion criteria consisted of all full-text articles published in English over a 15-year period between May 2006 and April 2021 and describing the safety assessment of powered IV syringe drivers and infusion pumps in hyperbaric environments. Only experimental studies were considered eligible for inclusion. Technical manuals, manufacturer-funded reports and review articles were excluded from the analysis.

A literature search was conducted of the MEDLINE® (National Library of Medicine, Bethesda, Maryland, USA), Embase® (Elsevier, Amsterdam, the Netherlands), SCOPUS® (Elsevier), Cumulative Index to Nursing and Allied Health Literature (CINAHL; EBSCO Information Services, Ipswich, Massachusetts, USA), Web of Science (Clarivate Analytics, Philadelphia, Pennsylvania, USA) and Google Scholar (Google Inc., Mountain View, California, USA) databases. The following search terms were employed in various combinations: "*hyperbaric oxygen*", "*hyperbaric critical care*", "*intravenous pumps*", "*infusion devices*", "*syringe drivers*", "*safety*", "*risk*", "*risk assessment*" and "*fire risk*". In addition, a hand search was conducted of relevant diving and hyperbaric medicine textbooks as well as the journal websites and workshop/conference proceedings of the South Pacific Underwater Medicine Society (SPUMS), UHMS, European Underwater and Baromedical Society (EUBS) and International Congress on Hyperbaric Medicine (ICHM).

Figure 1
Flow chart summarising the literature search



Following the literature search, the titles, abstracts and reference lists of retrieved articles were reviewed to identify relevant articles and remove any duplicate records. Full-text versions of the remaining articles were critically evaluated by two researchers to determine their eligibility for inclusion in the review. At this stage, the reference lists of the full-text papers were searched to identify additional relevant articles. For each selected article we reviewed the research objective, study design, methodology and results. The focus of the assessment was the safety and serviceability of the device in hyperbaric clinical practice. Finally, technical documents for the NFPA publications 99 and 53 and Australian and New Zealand Standards 4774.2 were reviewed to determine the safety requirements for portable patient care-related electrically powered equipment usage in oxygen-enriched environments and hyperbaric facilities.[8,13,14]

Results

A flow chart summarising the literature search is outlined in Figure 1. The raw data resulting from the search strategies are provided in Appendix 1.

Table 1

Characteristics of studies describing the safety assessment of intravenous pumps and/or syringe drivers for use in hyperbaric environments; *Denotes device without electrical components; FDA – Food and Drug Administration; HBO – hyperbaric oxygen; NiMH – nickel-metal hydride; O_2 – oxygen; PRBCs – packed red blood cells; Ref – reference number

Ref	Device name	Type of device	Methodology	Chamber type and pressure	Type of safety assessment	Summary of outcomes	Remarks
16	Baxter elastomeric LV10 Infusor™	*Elastomeric IV infusion pump	Delivery of various antibiotic solutions measured by weight. Flow rates assessed at different pressures and intervals.	Multiplace 101.3 (sea level), 203, 243 and 284 kPa	Function under pressure	• Flow rate affected by pressure changes. • Could not confirm elastomeric infusion pumps are always safe.	• Choice of antibiotic (ceftazidime vs. flucloxacillin) could affect flow rate.
17	Hospira Plum A+(Hb) infusion pump	IV infusion pump	Flow accuracy evaluated at various rates (1, 100, 250 and 999 ml·h^{-1}), viscosities (normal saline vs. PRBCs at 100 ml·h^{-1}), pressures and volumes. Occlusion alarm settings adjusted to assess battery life.	Mono and multiplace Monoplace: 86.1, 202.7 and 304 kPa Multiplace: 202.7 and 304 kPa	Function under pressure	• Pump functioned within manufacturer limits during multiplace trials. • Delivery variations in monoplace trials. • Battery life insufficient for a clinical HBO session at higher flow rates.	• Pump approved by the FDA but has since been retired. • Uses a self-contained lead acid rechargeable battery. • Tubing pliability may affect delivery, especially at low flow rates.
18	Carefusion Alaris PC infusion pump (model 8015 PC unit and 8100 large volume pump module)	IV infusion pump	Standardised experiment to assess basic suitability, inspect internal components and assess performance. Volume delivery assessed using 0.9% normal saline at 10, 50, 125 and 500 ml·h^{-1}	Multiplace Basic suitability: Up to 304 kPa at 10 kPa·min^{-1} Performance: 283.7 kPa at 30 kPa·min^{-1}, up to 405.3 kPa at 180 kPa·min^{-1}	Implosion Explosion Fire O_2 compatibility Pressure damage Function under pressure	• Pump posed no additional ignition risk. • Battery posed minimal risk of fire or explosion. • Pump performance within specifications for flow rate and occlusion alarms.	• Testing limited to expected conditions. • Locking pins installed due to ignition risk. • Spare units recommended in-chamber in case of device failure.
19	BBraun Perfusor Space syringe	Peristaltic syringe driver	Three different brands of 50-ml syringes delivering 0.9% saline assessed at ambient and increased pressure. Tested for force generation, pump flow accuracy at 1, 5, 10 and 40 ml·h^{-1} and occlusion alarm parameters.	Multiplace Compression to 284 kPa at 30 kPa·min^{-1}, continued for 30 minutes at 284 kPa, decompression at 30 kPa·min^{-1}	Implosion Explosion Fire O_2 compatibility Pressure damage Function under pressure	• Performance depended on syringe type and flow rate. • Syringe deformation during compression. • Two brands showed unacceptable stiction upon compression to 284 kPa.	• Potential to under-deliver during compression and over-deliver during decompression. • Testing using internal NiMH battery pack. • Powered by a stepper motor.

Table 1 continued.

20	Zyno Medical Z-800F, CME BodyGuard 323 ColorVision™ and Baxter Flo-Gard® 6201	Infusion pumps	Pumps connected to chamber pass-through with rigid small-bore tubing. Saline infused at 1–100 ml·h^{-1}.	Monoplace 86.1–304 kPa	Function under pressure	• Pumps functioned within manufacturer limits. • At low flow rates, tubing pliability affected fluid delivery.	• Occlusion pressure adjusted to 304 kPa. • None of the pumps were cleared by the FDA for HBO use at time of testing.
21	Unknown	Infusion syringe	Twenty 60-ml syringes filled with 20 ml of red food dye-coloured water. Volume assessed at various pressures.	Multiplace 243.2 kPa over 2 minutes, continued for 1 minute to 283.7 kPa	Function under pressure	• Over-delivery of fluid during decompression due to expansion of air. • Degree of effect dependent upon compression rate.	• Pressure changes during treatment may affect fluid delivery at low infusion rates.
22	Baxter elastomeric LV10 Infusor™	*Elastomeric IV infusion pump	Groups of 10 pumps assessed using either antibiotics or dopamine over 2-hour intervals (one group under normobaric and the other under hyperbaric conditions). Fluid delivery and flow rates determined by measuring fluid volume and weight.	Multiplace Control group: 101.3 kPa (sea level) Hyperbaric group: Compression over 10 minutes, continued at 243 kPa for 90 minutes, decompression for 20 minutes	Function under pressure	• Hyperbaric group delivered non-statistically larger volumes. • Both groups gradually delivered less fluid over time. • Neither group attained the manufacturer specified flow rate.	• Both groups tested at similar temperatures (22–23°C). • No statistical difference between groups in flow rate; however, a significant difference in flow rate based on fluid type (antibiotics vs. dopamine).
23	On-Q Pain Pump	Elastomeric pain ball infusion pump	0.2% ropivacaine delivered at 14 ml·h^{-1}. Fluid output measured by fluid volume and weight under normobaric and hyperbaric conditions.	Monoplace 101.3, 202.7, 243.2, 283.7 and 304 kPa for 9 minutes, compression/ decompression over 7 minutes	Function under pressure	• Fluid output stayed within acceptable limits (± 10–20%). • No statistical differences between groups in terms of output.	• Both groups showed gradual decrease in fluid output with time. • Potential delivery variations due to fluid viscosity.

Eighty-four papers were identified during the initial literature search, of which 38 were excluded due to duplication. The remaining 46 papers underwent abstract and title screening to determine their eligibility according to the inclusion and exclusion criteria. A total of 33 papers were excluded at this stage, with the remaining 13 articles selected for full-text review. The reference lists of these papers revealed two additional articles. Finally, seven papers were rejected for the reasons described in Figure 1. Thus, a total of eight studies were found that assessed different IV pump and syringes in hyperbaric environments.

All eight papers included in the final analysis were experimental studies assessing infusion equipment in hyperbaric settings.[16–23] An overview of the studies is presented in Table 1. Of the studies, six evaluated infusion pumps,[16–18,20,22,23] and two assessed syringe drivers.[19,21] The majority of the studies were conducted under multiplace hyperbaric conditions.[16,18,19,21,22] Of the remaining studies, two were conducted under monoplace conditions, and one was conducted under both multiplace and monoplace chamber conditions.[17,20,23]

Three studies assessed elastomeric devices, while the remaining five studies evaluated electrically powered infusion devices. The electrically driven devices potentially offer several routes of medication administration, including IV, intra-arterial, subcutaneous or epidural routes. For clarity, one of the studies in Table 1 assessed an unnamed syringe driver device to determine the effect of air spaces on syringe function in a hyperbaric environment.[21]

Two of the eight studies documented a more comprehensive safety evaluation, both from the same centre.[18,19] There is sufficient detail in these published papers to demonstrate that the majority of the UHMS assessment criteria were considered, apart from toxicity.[5,6,18,19] Six of the eight studies focussed primarily on the performance of the equipment in hyperbaric settings, particularly flow rate function and accuracy.[16,17,20–23] These studies did not consider fire safety, oxygen compatibility, ignition sources or toxicity.

Three studies revealed that the tested infusion pumps performed to the manufacturer's specifications, including occlusion alarm testing.[17,18,20] Three studies indicated that flow rates were not significantly impacted by increases in ambient pressure.[16,18,22] However, one study offered evidence to suggest that flow rate might be affected by choice of antibiotic; as such, the authors could not categorically determine that elastomeric infusion pumps are safe in hyperbaric settings.[16] Similarly, one study reported significant differences in flow rate depending on the type of medication being infused.[22]

One study noted that flow rates at low infusion rates (10 $ml \cdot h^{-1}$) fell below performance specifications, although this finding may be related to measurement methods.[18] Another also observed variations in fluid delivery during monoplace trials, particularly during the compression and decompression phases. The authors concluded that tubing compliance may affect fluid volume delivery, especially when infusion rates are low during compression and decompression.[17,20] At higher flow rates (999 $ml \cdot h^{-1}$), the battery life of the Hospira PlumA+ (Hb) pump in multiplace trials appeared to be less than the duration of a standard clinical hyperbaric session.[17] Lewis et al. found that the volumes delivered by the elastomeric pumps under hyperbaric conditions were not significantly different to the normobaric control group, although pumps operated in either condition did not achieve the flow rate claimed by the manufacturer, and delivered declining volumes over time.[22] Tobias et al. similarly noted no significant differences in fluid output between elastomeric pumps operated in hyperbaric and normobaric conditions, with volume delivery remaining within acceptable limits; however, the researchers also observed a gradual decrease in fluid output over time, regardless of group allocation.[23]

Both studies assessing syringe drivers indicated that changes in pressure affected equipment performance.[19,21] In one, the syringe drivers performed to the manufacturer's specifications; however, the researchers warned that performance was contingent on syringe type and flow rates. Issues with syringe deformation during compression resulted in significant stiction in two out of three brands of syringes – an effect which worsened with increasing syringe size. As a result, the authors cautioned that the device may under-deliver during compression and over-deliver during decompression.[19] In the other study, compression of the air spaces within the syringe during normal HBO treatment resulted in statistically significant changes in fluid volume delivery, with the degree of effect dependent on the rate of compression. It was suggested that fluid delivery would decrease or even halt entirely during compression, while extra fluid would be delivered during decompression as the air spaces within the syringe re-expanded.[21]

Discussion

Most patients receiving HBO treatment do not require infusions. However, some emergency cases and critically ill individuals frequently require continuous infusions of various drugs, including vasopressors, sedatives, insulin or antibiotics.[24]

This project sought to systematically review current literature evaluating the hyperbaric safety of IV infusion pumps and syringe drivers and compare this guidance with key technical

Footnote: * Appendix 1 is available on DHM Journal's website: https://www.dhmjournal.com/index.php/journals?id=310

safety reference standards. Seven infusion devices assessed in these experimental studies were evaluated based on a single criterion of the UHMS recommendations, namely whether the device functioned normally under pressure.[5–7] These studies did not consider implosion/explosion risk, fire safety, toxicity, oxygen compatibility or risk of pressure damage, which is of significant concern. Sources of heat and ignition within equipment such as lithium-ion batteries and brushed motors could precipitate fire in the oxygen-enriched hyperbaric environment.

Section 3.9 of the Australian/New Zealand (ANZ) Standards 4774.2, which considers work performed in HBO facilities, states that: "*Portable electronic or electrical systems (e.g., entertainment units) shall have a risk assessment report completed by a competent person for risk of ignition and ability to support combustion prior to being taken into the chamber*".[13] Thus, only two of the identified studies attempted to conduct a risk assessment consistent with ANZ standards and considered all UHMS criteria except for toxicity.[5,6,18,19] Both were from the same centre.

One study conducted three additional phases of testing, including basic suitability testing, internal inspection and surface temperature measurement to determine fire risk, implosion risk, oxygen compatibility and pressure damage. The authors concluded that the infusion pump itself was not at risk of pressure damage or ignition.[18] Component surface temperatures reached a maximum of 74°C, which was below the maximum allowable (85°C) under NFPA99 Code.[8] Additional components were also found to present minimal risk of fire or explosion under conditions of expected use, including other potential sources of ignition such as the stepper motor, encapsulated lithium battery and lubricating grease. The authors identified a possible risk of spark ignition due to the design of the device which allowed electrical connection or disconnection of the modules during operation.[18] Another study documented the safety assessment process which preceded the evaluation of the BBraunPerfusor Space syringe.[19] This did not follow UHMS recommendations, but was locally developed and followed a local matrix.[19,25] There was sufficient description in the text to identify that issues of pressure deformity, fire, internal ignition source and oxygen compatibility had been evaluated. Unfortunately, the locally used matrix was never published other than in an abstract which lacked sufficient detail to be properly evaluated, although it was again referred to in Smale and Tsouras's paper.[18,25]

Overall, the majority of the tested infusion pumps conformed to the manufacturer's specifications, with reported flow rate and output variations falling within clinically acceptable ranges; however, there were several findings of note.

It is possible that different antibiotic solutions may affect flow rates. Four out of five infusions of ceftazidime were above the set range for clinically acceptable infusion (9–12 $ml \cdot h^{-1}$) whereas trials using flucloxacillin demonstrated flow rates within acceptable limits.[16] Similarly, significant flow rate variations could occur depending on choice of infused medication (antibiotics vs. dopamine).[22] In monoplace situations, the pliability of infusion pump tubing external to the chamber may affect fluid volume delivery, especially at low flow rates.[17,20] This occurs because the expansion of the tubing during compression results in a reduction in the amount of fluid delivered. Subsequently, when the internal chamber pressure decreases during decompression, the extra fluid in the expanded tubing is delivered in a single bolus, even if the pump is turned off. This occurred even with rigid, small bore tubing and would likely be amplified with normal medical-grade IV tubing.[17]

Both assessments of syringe infusion devices demonstrated significant variations in fluid delivery, especially at low infusion rates. In particular, the devices had the potential to release too much fluid during the decompression phase and not enough during compression, likely as result of the re-expansion and compression of pockets of air within the syringe.[19,21] In addition, syringe deformation and stiction occurred during pressurisation with both the Terumo and Becton Dickinson syringes.[19] Stiction refers to the occurrence of static friction between the plunger seal and the wall of the syringe which impedes the normal movement of the syringe, resulting in a jerky, start-and-stop type of motion. The subsequent flow irregularities and inadvertent boluses from this unintentional movement can be detrimental to critically ill patients.[26] The researchers were obliged to continue their experiments using the BBraun syringe, with which there was less lateral movement of the plunger due to the stiffer barrel and the increased distance between the plunger O-rings.[19]

Two studies evaluated the Baxter LV10 Infusor™, a large-volume, non-electronic, balloon-driven, elastomeric infusion pump.[16,22] Although not an electrically powered device, the documented assessments were detected by the literature search and included for analysis. We considered that elastomeric infusion devices should be subject to the same safety evaluation processes as any other equipment.

Depending on the type of hyperbaric chamber utilised, supportive devices such as infusion pumps may either be placed within the chamber itself, for instance in the context of multiplace chambers treating multiple people, or externally, in the case of monoplace chambers which only have room for a single patient. In the latter, infusions are provided through ports in the chamber hull.[24] Both chamber types pose challenges when operating infusion devices. Pumps placed outside of monoplace chambers have the advantage of keeping the electrically powered device outside the hyperbaric environment. However, they must work against a considerable pressure gradient to deliver fluids into the pressurised chamber. Many infusion pumps have a much lower default upper occlusion pressure setting

than is feasible in monoplace settings.[20] In contrast, pumps within multiplace chambers have no pressure differential to overcome but pose a greater risk due to their location within the hyperbaric chamber.[24]

Fire is a key risk for devices taken into multiplace hyperbaric chambers. These chambers are pressurised with air, but oxygen levels within the chamber may rise if not carefully monitored and controlled. Materials may ignite and burn more easily; moreover, because the chamber is pressurised and enclosed, rapid extinguishing of the fire or evacuation of the patient may be difficult, if not impossible.[27] Thus, there should be strong risk awareness and zero tolerance for devices that could cause a fire. Potential sources of ignition such as sparks must be eliminated (e.g., brushed electric motors or wire connections) and devices must not heat up when operating. Moreover, it is imperative that the devices should not support combustion; in other words, that they should be composed of oxygen-compatible materials with a low probability of ignition.[28,29]

Using a non-contact infrared thermometer, one study confirmed that the operating surface temperature of the internal components of the Carefusion Alaris PC infusion pump did not exceed 75°C, as per section 14.2.9.3.11 of the NFPA 99 Code.[8,18] However, their risk evaluation identified issues that could conceivably pose a risk of ignition in an oxygen-rich environment. Specifically, the device included electronic components, lubricating grease and an encapsulated lithium battery. The brushless stepper motor was considered low risk. This would not be the case for a brushed motor which can produce sparks. A key area of risk allowed for the electrical connection and disconnection of the large-volume pump module from the PC unit. This created a risk of electrical sparks but was controlled by preventing disconnection using locking pins. The researchers also recommended bringing two spare units into the chamber in case of device failure to avoid having to latch or unlatch the modules during operation or being forced to subject a new device to rapid changes in pressure and temperature due to transfer through the entrance lock. The lubricating grease applied to the door clamping mechanism was determined to be a stable phenylmethyl silicone-based grease which did not pose a risk of spontaneous ignition under expected hyperbaric conditions. The researchers confirmed the PC unit included a non-rechargeable, single-cell lithium battery encapsulated in a 0.4-mm nylon casing.[18]

Lithium-ion batteries have revolutionised portable electronic devices by facilitating reductions in equipment size and weight and allowing greater power output, longer endurance and the ability to supply voltage more effectively than previous battery technologies. Nevertheless, despite their extensive usage in medical devices, lithium-ion batteries are susceptible to thermal runaway and fire, with pressure exposure increasing failure risk.[30] One study has demonstrated damage to pump batteries when exposed to pressure, an event which could lead to an in-chamber fire.[12] Although the NFPA previously prohibited devices using lithium-ion batteries in hyperbaric chambers unless specifically qualified by the manufacturer or by a recognised testing agency, such restrictions have been lifted in recent editions.[8,14] Nevertheless, as noted by Burman, the code intends that power sources remain outside of the chamber.[31] It can be concluded that all providers using battery-operated electrically powered devices in an oxygen-enriched environment should ensure that the batteries are non-damaged, secure, contained in fully enclosed housings and under no circumstances allow charging of any battery type while in-chamber.[4,8,31]

Following a risk assessment, Smale and Tsouras deemed the battery in the Carefusion Alaris PC infusion pump to pose minimal risk of fire or explosion.[18] This determination was based on previous data confirming the oxygen compatibility of the encapsulated nylon at 12,000 kPa to be 200°C in a 99.5% oxygen atmosphere.[32] These conditions fall considerably outside the upper range of temperature and pressure expected in clinical hyperbaric settings.[18] Moreover, non-rechargeable, single-cell, low-voltage lithium batteries are considered preferrable in hyperbaric environments compared to larger, higher voltage or rechargeable batteries.[4] None of the other studies identified in this review assessed the ignition risk posed by the batteries of their tested infusion devices. One study reported that the life of the self-contained, lead-acid rechargeable battery in the Hospira Plum A+(Hb) infusion pump did not match the duration of a typical HBO treatment session at high pump flow rates.[17] Battery endurance is a key consideration for infusion devices proposed for use in multiplace chamber settings.[24] Historically, unsealed lead-acid batteries are discouraged in hyperbaric chambers due to the risk of spillage and hydrogen production during recharging, while nickel-metal hydride batteries (such as in the BBraunPerfusor space syringe driver[19]) are considered to be the most suitable type of rechargeable battery.[33]

Most IV infusion devices are used in 'off-label' fashion in hyperbaric facilities, signifying that their sale for use in hyperbaric chambers is unsupported by the manufacturer. In one study, none of the three pumps assessed had been cleared by the FDA for use in hyperbaric chambers.[20] Moreover, even those few devices which have been granted FDA clearance for hyperbaric use have not had a published risk assessment.[9] These devices appear to have a limited design life and may be discontinued by the manufacturer, as was the Hospira Plum A+ (Hb) infusion pump which was retired in June 2014.[17] Once a hyperbaric compatible device becomes unavailable, the search for, and risk assessment of appropriate replacements must recommence. The safety evaluation of electrically powered devices for hyperbaric use needs to be on-going and continuous across the entire hyperbaric medicine community.

In view of the near universal uptake of medical equipment incorporating lithium-ion batteries, coupled with their serious risk of thermal runaway and fire in hyperbaric environments, the authors strongly recommend that a thorough safety/risk assessment should precede any testing of IV pump integrity and function under pressure, covering – as a minimum – the six UHMS criteria. This recommendation would sensibly be extended to all portable electrically powered devices intended for use within the hyperbaric environment.

This review has demonstrated deficiencies in the published safety evaluations of IV pumps for use in the hyperbaric environment. Despite a simple, published process for evaluating new devices (UHMS), including NFPA codes and recommendations for fire safety, only eight studies of IV infusion devices were identified in this review, and only two had comprehensive safety assessments. It is of concern that most studies focused only on whether the device functioned normally under pressure. Given the widespread use of HBO treatment, it would be useful to establish a central database hosting comprehensive equipment risk assessment to permit quick reference by intending new users. Nonetheless, it is important to note that the burden of proof of safety for using an off-label and non-FDA-cleared device in a hyperbaric chamber, an intrinsically hazardous environment, remains the responsibility of the facility; as such, no study or risk assessment should be considered to bestow unilateral safety approval, either tacit or implied, as there are too many unknown variables at play. However, such a database could be useful for guidance.

LIMITATIONS

The search strategy may have missed key studies that were published outside of the medical literature. In addition, articles published in languages other than English could not be included in the systematic search. As such, it is possible that relevant studies published in other languages were not included in the final analysis.

Conclusions

A systematic review of published safety data describing the assessment of IV pumps and/or syringe drivers for use in hyperbaric environments revealed that many recent studies on this topic have concentrated primarily on the performance and function of devices and have missed many aspects of a comprehensive safety assessment. There is a need for facilities to conduct their own comprehensive safety assessments of IV infusion devices for use under hyperbaric conditions. This should include important criteria such as explosion or implosion risk, ignition and fire risk, toxicity, oxygen compatibility and pressure damage. This process could be enhanced by a publicly accessible database hosting the risk assessments to guide future hyperbaric practitioners in their selection of equipment. It would also be useful if manufacturers would support the development and assessment of infusion devices which conform to technical safety standards for use in hyperbaric chambers, although this may be unrealistic due to the limited market for such applications.

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Short communication

Validation of sham treatment in hyperbaric medicine: a randomised trial

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Abstract
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Introduction: This study aimed to determine the lowest possible atmospheric pressure in the 111–152 kPa (1.1–1.5 atmospheres absolute [atm abs]) range that would require the patients to equalise their ears, allowing an effective sham for a 203 kPa (2.0 atm abs) hyperbaric exposure.
Methods: We performed a randomised controlled study on 60 volunteers divided into 3 groups (compression to 111, 132 and 152 kPa (1.1, 1.3, 1.5 atm abs) to determine the minimum pressure to obtain blinding. Secondly, we applied additional blinding strategies (faster compression with ventilation during the fictitious compression time, heating at compression, cooling at decompression) on 25 new volunteers in order to enhance blinding.
Results: The number of participants who did not believe they had been compressed to 203 kPa was significantly higher in the 111 kPa compressed arm than in the other two arms (11/18 vs 5/19 and 4/18 respectively; $P = 0.049$ and $P = 0.041$, Fisher's exact test). There was no difference between compressions to 132 and 152 kPa. By applying additional blinding strategies, the number of participants who believed they had been compressed to 203 kPa increased to 86.5 %.
Conclusions: A compression to 132 kPa, (1.3 atm abs, 3 metres of seawater equivalent) combined with the additional blinding strategies of forced ventilation, enclosure heating and compression in five minutes, simulates a therapeutic compression table and can be used as a hyperbaric placebo.

Introduction

In hyperbaric medicine, the recommended therapeutic indications are regularly criticised due to a lack of high-level evidence, as well as the limited number of randomised trials. A sham procedure can be defined as one performed on a control group participant to ensure that he or she experiences the same incidental effects of the procedure as do those participants on whom a true procedure is performed. Randomised trials which do not include a sham control group may be at risk of bias due to a placebo effect. The addition of a sham group (i.e., a placebo control group) allowing patient-blinding can improve the quality of evidence.

Conducting a sham control treatment in hyperbaric medicine is particularly challenging as increased atmospheric pressure is often easily perceived by patients who need to equalise their ears. Conversely, an absence of increased pressure may also be easily identified by patients in the sham control group who will not need to equalise their ears. In hyperbaric medicine one challenge in conducting a sham treatment is to determine the lowest atmospheric pressure that still requires patients to equalise their ears, (allowing blinding) whilst minimising the biological effect of that pressure.

A recent systematic review of 42 studies involving placebo groups in hyperbaric medicine identified three types of strategies for conducting a sham: shallow air compressions to 111–152 kPa (1.1–1.5 atmospheres absolute [atm abs]) breathing 21% oxygen; equivalent depth compressions to 203–253 kPa (2.0–2.5 atm abs) breathing a gas mixture adjusted to deliver an inspired PO_2 similar to air at 101.3 kPa (1.0 atm abs); or equivalent depth compressions to 203–253 kPa (2.0–2.5 atm abs) breathing 21% oxygen.[1] A risk/benefit analysis favoured shallow air compressions (1.1–1.5 ATA) breathing 21% oxygen. This would allow acceptable blinding at a very low level of risk whilst minimising the risk of a biological effect from the minimally increased inspired PO_2. However, the optimal pressure for a sham treatment within the 111–152 kPa range remained undetermined.

In order to guide hyperbaric medicine researchers in conducting sham treatments, this study aimed to determine the lowest possible atmospheric pressure in the 111–152 kPa range that would include a requirement for patients to equalise their ears, allowing an adequate sham for treatments conducted at 203–253 kPa.

Methods

ETHICS APPROVAL AND PARTICIPANTS

Application was made to our institutional ethics board '*Commission Cantonale d'Ethique de la Recherche*' (Req-2018-00387) and the study was exempted from comprehensive ethical review. Each volunteer was given a detailed participant information sheet which explained what would take place during the session, and gave written informed consent. Participants were volunteer healthcare professionals from the University Hospital of Geneva, Switzerland recruited through information published on the hospital's intranet site. All were evaluated by a hyperbaric physician to exclude any contraindication.

DESIGN, INTERVENTION AND OUTCOME MEASURES

The study was carried out in two stages.

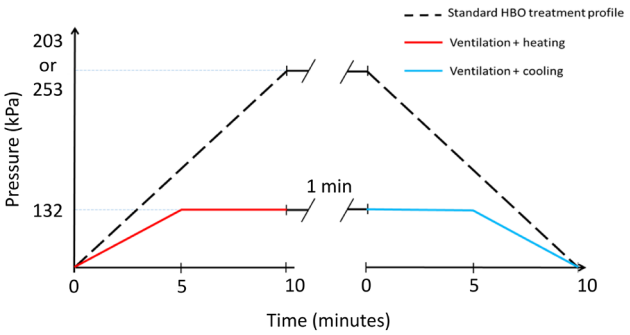
In Stage 1, we conducted a randomised trial, using a 1:1:1 ratio and block size of six. Volunteers were randomised to three pressure profiles: Group A, 111 kPa (1.1 atm abs, 1 metre sea water [msw] equivalent); Group B, 132 kPa (1.3 atm abs, 3 msw); Group C, 152 kPa (1.5 atm abs, 5 msw). Randomisation was performed using the RAND function in Excel. All three groups had identical diving sequences; pressurisation over 10 minutes, duration at pressure for one minute, and decompression over 10 minutes.

For Stage 2, a new cohort of 25 participants was recruited. All were compressed to 132 kPa (1.3 atm abs, 3 msw) and we decreased the compression time from 10 minutes to five minutes to increase the sensation of pressure change to a rate closer to that experienced during a normal treatment and thus improve blinding. Additionally, forced ventilation using air addition accompanied by simultaneous air exhaust (sometimes referred to as 'flushing') more closely replicated the noise of a normal treatment during compression. To mimic the patients' usual experience of heat during compression and cold during decompression, we used the hyperbaric chamber's air conditioning system to heat during the sham compression, and during decompression both forced ventilation and cooling of the chamber was performed (Figure 1).

Each participant was compressed separately in the department's hyperbaric multi-place chamber (HAUX-STARMED 2400/4/SC3) and accompanied (with the exception of volunteers from the hyperbaric department) by a hyperbaric nurse.

Immediately after the decompression, participants were asked to indicate whether or not they believed they had been compressed to an equivalent depth of 10 msw (203 kPa, 2.0 atm abs). This pressure was chosen as the purpose of this study was to validate a sham for a planned double-blinded, randomised controlled trial on the effects of hyperbaric oxygen treatment (HBOT) during sickle cell crisis. In this planned trial, the patients in the treatment arm will be treated using an adapted exposure at 203 kPa (2.0 atm abs) (Clinical Trials: NCT05289700). Our primary outcome was, therefore, to measure participants' blinding perception after decompression, defined as their belief that they had been compressed to 10 msw equivalent during the session.

Figure 1
Table profile with additional blinding strategies; compression over five minutes with forced ventilation ('flushing') for 10 minutes (theoretical duration of compression), heating during compression and cooling during decompression



The participant information sheet contained the following information:

"*You are going to participate in a study aimed at validating a placebo group in hyperbaric conditions.*
You will enter the chamber accompanied by a nurse. You will or will not undergo compression at 10 meters depth following a standard profile including:
– Pressurisation for 10 minutes. During this phase you may feel a sensation of heat and noise (gas compression). You will also need to balance your ears by performing a so-called Valsalva manoeuvre or by swallowing. These manoeuvres are close together at first, then more and more distant.
– A stay at the bottom reduced to a few minutes. Normally this phase lasts 65 minutes.
– A descent lasting 10 minutes. During this phase you may feel a sensation of cold and noise (gas decompression). No balancing manoeuvres are required. These are done on their own."

On completion of the hyperbaric compression and decompression participants were asked to answer four questions with an answer chosen from (yes/no/I can't tell):

1. Do you think you have undergone compression as explained to you (compressed to 10 msw)?
2. Did you need to balance your ears?
3. Did you feel hot on compression?
4. Did you feel cold on decompression?

Table 1
Participant characteristics; F – female; IQR – interquartile range; M – male

Parameter	Group A (111 kPa)	Group B (132 kPa)	Group C (152 kPa)
Sex ratio (M/F)	16/4	12/8	13/7
Age (years) Median [IQR]	32.5 [27.7;43.0]	32.0 [27.5;38.7]	36.0 [30.7;44.2]
Relevant experience: None/Diver/Hyperbaric medicine attendant	9/4/7	10/4/6	9/4/7

Table 2
Response to the question "*have you been compressed to 10 metres*?" with participants stratified according to diving or hyperbaric experience; ? – participants who responded "*I can't tell*"

Relevant experience	Group A (111 kPa)			Group B (132 kPa)			Group C (152 kPa)		
	Yes	No	?	Yes	No	?	Yes	No	?
None	5	4	0	7	3	0	8	0	1
Diver	0	3	1	2	1	1	2	2	0
Hyperbaric attendant	2	4	1	6	1	0	4	2	1
Total	7	11	2	14	5	1	14	4	2

Table 3
Responses from the 25 new volunteers with no diving experience in Stage 2; ? – participants who responded "*I can't tell*"

Question	Yes	No	?
Do you think you have undergone compression as explained to you?	22	1	2
Did you need to balance your ears?	25	0	0
Did you feel hot on compression?	5	20	0
Did you get cold on decompression?	22	3	0

SAMPLE SIZE AND ANALYSIS

For Stage 1 we elected to recruit a convenient sample size of 60 total, i.e., 20 participants per group. Based on our primary outcome, a sample of 60 participants for three groups, and assuming a power of 0.8 and alpha risk of 0.05 (two-tailed), an effect size of 0.4 could be detected.

All participants who responded "*I can't tell*" were excluded as their response were not helpful in answering our research question. Our binary primary outcome was analysed using a Fisher's exact test, comparing B and C groups to A group. A two-tailed P-value of < 0.05 was considered significant for all analyses. Statistical analysis was performed using SPSS 16.0 (SPSS, Chicago, IL).

Results

In Stage 1 sixty volunteers were recruited and included in the analysis, i.e., 20 in each group. Relevant demographics are shown in Table 1. The number of participants responding "*No, I was not compressed to 10 msw*" was significantly higher in the 111 kPa group than in the 132 and 152 kPa groups (11/18 vs 5/19 and 4/18 respectively; $P = 0.049$ and $P = 0.041$). There was no significant difference in responses between participants compressed to 132 kPa (3 msw) or 152 kPa (5 msw) ($P = 1$). We note that the 111 kPa group had a higher proportion of males than in the 132 or 152 kPa groups (4:1 vs 1.5:1 or 2:1). Diving/hyperbaric attendant experience did not appear to dramatically impact perception of pressurisation, particularly in the 132 and 152 kPa exposures although numbers are small (Table 2).

In the second stage, a further twenty-five new volunteers without hyperbaric experience were included. All were compressed to 132 kPa (1.3 atm abs, 3 msw equivalent). The number of participants who believed they had been compressed to 203 kPa (2.0 atm abs, 10 msw) increased to 22/25 (88%) with one who believed they were not, and two who couldn't tell. One hundred percent of volunteers reported equalising their ears (Table 3). One volunteer described ear pain without it being necessary to interrupt the session. An examination post session, showed a grade one barotrauma.

Discussion

In order to initiate randomised, double-blinded, placebo-controlled trials in hyperbaric medicine, we evaluated the shallow compression profile that best simulated a therapeutic table. Although compression to an equivalent pressure such as 203 or 253 kPa (2.0 or 2.5 atm abs) creates strong conditions for blinding, as concluded by others,[1] we believed that a lower pressure compression was simpler and ethically acceptable.[1]

We hypothesised that a lower pressure compression could give the patient the impression that they participated in a true HBOT treatment at 203 or 253 kPa (2.0 or 2.5 atm abs). By testing several pressures we were able to identify the lowest pressure that seemed adequate for effective blinding. Compression to 111 kPa (1.1 atm abs, 1 msw equivalent) in 10 minutes did not appear to create sufficient blinding. There was no difference in blinding success between a compression to 132 vs 152 kPa (1.3 atm abs, 3 msw vs 1.5 atm abs, 5 msw) and blinding seemed acceptable. Although the group with a low degree of blinding had a much higher proportion of males, this difference seems an implausible explanation for the poor blinding success. Blinding was further improved by reducing the compression and decompression time to five minutes while maintaining forced ventilation for 10 minutes and using additional blinding strategies such as the addition of heat to compression and cold to decompression. On the second set of volunteers, blinding was almost total with only one participant claiming not to have been compressed while all claimed to have balanced their ears.

The inspired PO_2 breathing air at a pressure of 132 kPa (1.3 atm abs) is equivalent to breathing 28% oxygen at 101.3 kPa (1.0 atm abs). Even if this small rise in inspired PO_2 does not correspond to a totally inert placebo, it should not have a significant impact on results of most trials of true HBOT.

Conclusion

A compression to 132 kPa (1.3 atm abs, 3 msw equivalent) in conjunction with confounding elements such as forced ventilation, enclosure heating and compression over five minutes, simulates a therapeutic compression table and can be used as a hyperbaric sham. This profile has the advantage of being extremely low risk and with an inspired PO_2 equivalent to 28% oxygen at atmospheric pressure, there can be no oxygen effect that would require hyperbaric exposure to achieve.

References

1 Lansdorp CA, van Hulst RA. Double-blind trials in hyperbaric medicine: A narrative review on past experiences and considerations in designing sham hyperbaric treatment. Clin Trials. 2018;15(5):462–76. doi: 10.1177/1740774518776952. PMID: 29865904. PMCID: PMC6136075.

Acknowledgements

We would like to thank all the staff of the diving and hyperbaric unit and Tamara Mann for her revision.

Conflicts of interest and funding: nil

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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

The beginning of 2023 marks a seismic shift in the landscape of diving and hyperbaric medicine in Australia with the near-simultaneous retirements of Professor Michael Bennett, AM and Clinical Professor David Smart, AM.

Both David ('Smarty') and Mike are Past Presidents of SPUMS and have made significant contributions to the practice of, teaching and research in the field of diving and hyperbaric medicine. Their brief bios are following in the SPUMS notices and news.

Fortunately, despite being retired from all clinical work, Mike and David will continue to be involved with SPUMS for a while longer. On behalf of all SPUMS members, I wish them a long and happy retirement

The new SPUMS website is up and running and is a vast enhancement on the previous platform with improved graphics and layout. On behalf of SPUMS ExCom and our members, I would like to express our thanks for the financial support from the Australasian Diving Safety Foundation to enable SPUMS to update our website and to Nicky Telles, Xavier Vrijdag and the others who made this happen. A major benefit of the new website is the ability for membership auto-renewal. This aids SPUMS in streamlining our processes and the benefit for members is not having to remember/complete the bureaucracy to renew. Payments are made via Stripe, an online payment processing and commerce solution that is used by many of the world's biggest corporations. All payment information is encrypted, and Stripe doesn't share any credentials with our website.

Registration for the SPUMS Annual Scientific Meeting (ASM) is now possible via our website.

The SPUMS 2023 ASM will be held in Cairns and will include diving with the programme and registration is available now on the SPUMS website: https://spums.au/index.php/asm-registration.

Conference theme: *Diver health and ocean health amidst the storm clouds of climate change. A shared vision for underwater medicine and marine science.*

Convenors: David Smart and Cathy Meehan
Date: Sunday 4 June to Friday 9 June 2023
Venue: Crystal Brook Riley Hotel, Cairns, Australia

There will also be a workshop to develop a SPUMS Position Statement on paediatric diving.

If you are yet to register, I strongly encourage you to do so soon. Pre- and post-conference liveaboard trips to the Great Barrier Reef are available for registrants which feature diving with minke whales.

I look forward to seeing you in Cairns in June.

The next introductory course in diving and hyperbaric medicine will again be held in Fremantle from 27 February–10 March 2023 and is fully subscribed; it will already have been run by the time this issue is published. This course is held yearly only and always fills early, so if you want to register for 2024, don't delay. It is planned that the 2024 course will have similar dates to this year's course. Information available from: 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. Please contact John Lippmann at johnl@adsf.org.au for more information.

Dr Neil Banham
SPUMS President

Clinical Professor David Smart AM – Biography

- Emergency medicine specialist, and specialist in diving and hyperbaric medicine, President, South Pacific Underwater Medicine Society (SPUMS)
- Recreational scuba diver since 1983 – currently master diver qualification
- Over 3,000 hours logged underwater
- Recreational diving photographer
- Roles with SPUMS
  - Executive member and Australian Standards Representative: 1999 to present
  - Chairman Australian and New Zealand Hyperbaric Medicine Group: 2001–2013
  - SPUMS Education officer and censor: 2002–2013
  - SPUMS President: 2014–2020
  - SPUMS Immediate Past President 2020 to present
  - Diving and Hyperbaric Medicine Journal Academic Board 2008–2020
  - Diving and Hyperbaric Medicine Journal Governance Committee 2020–present
- Leader in diving and hyperbaric medicine in Australia, the South Pacific, and internationally working in the field since 1985, with a continuous commitment to diving safety and education for over 30 years.

Contributions to diving safety in Tasmania and Australia

- Diving medicine specialist and medical co-director, Department of Diving and Hyperbaric Medicine Royal Hobart Hospital since 1994 – managed between 50 and 80 diving accidents annually in Tasmania, including 25–30 cases of decompression illness annually
- Co-author of Tasmania's Emergency management and retrieval protocol for Diver Emergencies – updated every second year for two decades
- Teaching of Tasmanian recreational diver seminars annually – combined dive clubs weekend Bicheno Tasmania in June, diver safety and awareness evenings run for the general public at Royal Hobart Hospital and marine safety events with Marine and Safety Tasmania.
- Medical Doctorate Thesis published in 2005 – Carbon Monoxide Poisoning – since then have been an active campaigner for preventing carbon monoxide poisoning in the community and also preventing carbon monoxide poisoning in divers
- Production of Diver safety videos with Marine and Safety Tasmania:
  - Recipe for safe diving from Tasmania (2003) https://www.youtube.com/watch?v=1IZiGA4MEdA
  - (Hookah diving safety 2013) https://www.facebook.com/watch/?v=323310065275274 (CO poisoning 2017)
- Supervisor of training, diving and hyperbaric medicine, at Royal Hobart Hospital, 2003 to present
- Leader in the establishment of diving and hyperbaric medicine specialist training with the Australian and New Zealand College of Anaesthetists, Diploma of Advanced Diving and Hyperbaric Medicine
- National Chair of Examiners in diving and hyperbaric medicine, Australian and New Zealand College of Anaesthetists Diploma of Advanced Diving and Hyperbaric Medicine 2017 to present
- Contributor to Australian Resuscitation Council guidelines on first aid management of injured divers
- Member Australian Standards Diving Safety Committees from 1999–2020 for Australian and New Zealand Standards AS/NZS 2299.1, 2299.2, 4005.1, 2815.1, 2815.2, 2815.3, 2815.4, 2815.5 and 2815.6, and 4774.2 (including revisions)

Research papers on diver safety and accident management

- Working with Tasmania's aquaculture industry for over 30 years, leading to very significant reductions in decompression illness and improvements in diver safety
  - SPUMS Journal. 1990;20:159–65.
  - Papers and proceedings of the Royal Society of Tasmania. 1999;133(1):77–83.
  - Diving and Hyperbaric Medicine. 2014;44(3):124–36.
  - Management of diving emergencies. Emergency Medicine. 1997;9:42–4.
- Health risk management in Tasmania's abalone industry. Diving and Hyperbaric Medicine. 2010;40(2):83–7.
- Australian standards for occupational and recreational divers. Change in the wind? Diving and Hyperbaric Medicine. 2010;40(3):160–1.
- Diving related pulmonary oedema – dive accident investigation to assist the forensic pathologist. Diving and Hyperbaric Medicine. 2014;44(2):97–100.
- Safety in hyperbaric environments – lithium batteries. Diving and Hyperbaric Medicine. 2011;41(3):165.
- Epilepsy, scuba diving and risk assessment. Diving and Hyperbaric Medicine. 2013;43(1):37–41.

- Management of inner ear barotrauma in divers and recommendations for returning to diving. Diving and Hyperbaric Medicine. 2014;44(4):208–22.
- Occupational diving training in Australia: Diving and Hyperbaric Medicine. 2017;47(4):214–5.
- A 20-year review of diving deaths in Tasmania. Diving and Hyperbaric Medicine. 2019;49(1):21–9.

Educational activities – training doctors and divers in Diver safety, management of diving accidents and assessment of medical fitness to dive in order to prevent diving accidents.

- ANZHMG Introductory Course in Diving and Hyperbaric Medicine Faculty 1998 to present
- MOUM Course Royal Australian Navy 2004 to present
- Medical Support of Offshore and Saturation Diving 2016
- Teacher medical management of Diving Accidents – diver medical technician courses, ADAS Accredited, Occupational and Offshore Divers the Underwater Centre Tasmania, 1997–2017
- Teacher at Tasmanian Hyperbaric Nursing Courses from 1995-present
- COVID Safety – use of AMRON hoods for oxygen administration at 1 ATA – developed by Professor Smart – Local Royal Hobart Hospital Procedures
- COVID and diving fitness – President's report. Diving and Hyperbaric Medicine. 2020;50(2):188–90

Presentations on diving safety

International Outreach – Teaching management of diving accidents to doctors and nurses in Fiji, 2002 to present (last three years via Emergency Life Support Courses)

Fiji hyperbaric facility **–** Specialist advice regarding safety issues in the previous Fiji hyperbaric facility, and assistance with the specifications/implementation of CWMH Suva hyperbaric facility

Stay Safe when you dive – educational publication – Tasmanian seafood industry

Research and Presentations on Hyperbaric Attendant Safety

2009: Hyperbaric chamber attendant safety 1. Doppler analysis of decompression stress in multiplace chamber attendants. Diving and Hyperbaric Medicine. 2009;39(2):63–70.

2009: Hyperbaric chamber attendant safety 2. 14-year health report of multiplace chamber attendants. Diving and Hyperbaric Medicine. 2009;39(2):71–6.

SPUMS representative Australian Standards Committees for preparation of Australian and New Zealand occupational diving safety and training standards 1999–present

These standards are key documents for the operational safety of divers in Australia and New Zealand.

AS/NZS 2299.1 (1999), (2007), (2015) Occupational diving operations: standard operational practice (including medical guidelines)

AS/NZS 2299.1 (2002) Occupational diving operations: scientific diving

AS/NZS 2815.1 (2008) Training and certification of occupational divers: occupational scuba diver standard

AS/NZS 2815.2 (2013) Training and certification of occupational divers: surface supply diving to 30 m

AS/NZS 2815.5 (2006), (2013) Training and certification of occupational divers: dive supervisor

AS/NZS 2815.6 (2013) Training and certification of occupational divers: restricted occupational scuba diver

AS/NZS 4774.2 (2004), (2019) – Work in compressed air and hyperbaric facilities: hyperbaric oxygen facilities

AS 4005.1, 4005.2, 4005.3, 4005.4, 4005.5 (2000) Training and certification of recreational divers (including medical guidelines)

SPUMS representative: Australian Federal work health and safety legislation

- SPUMS representative: review of diving at work section of the federal legislation (2014–18)

Tasmanian diving industry safety

- Tasmanian abalone industry, diving code of practice review 2018–19
- Worksafe Tasmania – presentations on diving safety to Tasmanian occupational and scientific diving sector
- Advisor to CSIRO diving committee and Australian Antarctic division of diving safety
- Advisor to aquaculture industry on diving health and safety standards

Expedition work

Australian Antarctic division research expedition #68 Casey Base 2014 – medical supervisor of diving operations,

including setting up emergency protocols, recompression chamber facilities, overseeing dive safety and review of operational procedures

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

SPUMS diving medical health risk assessment for diving (1999), (2011), (2020)

Australian Resuscitation Council – Guideline 9.3.5 (2011) resuscitation of divers who have used compressed gas

International collaborations – for medical guidance and prevention of diver injury

- Stellenbosch Collaborative – content provider to the development of a modular course in diving and hyperbaric medicine Stellenbosch University, South Africa (2012). Approved by Undersea and Hyperbaric Medicine Society
- Joint position statement on persistent foramen ovale and diving (2015) with United Kingdom Sports Diving Medical Committee – first author
- Patent foramen ovale and fitness to dive consensus (2015) – with Divers Alert Network and Undersea and Hyperbaric Medical Society – speaker and panel discussion. ISBN 978-1-941027-71-4
- Review of cardiovascular risk assessment for diving (2020). Diving and Hyperbaric Medicine. 2020;50(3) – 3rd author
- European diving technology committee invited contributors to review medical training standards and fitness-to-dive standards

Awards relating to diving safety

2003 National finalist Australian Healthcare Association Baxter Healthcare Innovation Awards. improving health outcomes for Tasmania's aquaculture industry divers

2005 Undersea and Hyperbaric Medicine Society USA Craig Hoffman Award for contribution to diving safety in Tasmania, Australia

2013 Derek Craig Award for research and contribution to professional diving safety

Australian Diver Accreditation Scheme

2015 Undersea and Hyperbaric Medicine Society USA. Award for excellence in commercial diving. For outstanding contributions to the commercial diving industry in the area of increased productivity or performance of the working diver. Specific recognition is given to practical application of biomedical knowledge and science to the solution of problems encountered in diving operations

2019 Appointed Member of the Order of Australia for services to hyperbaric and diving medicine and professional organisations (Governor General's Honours list)

Educational book chapters covering diving accidents

Dysbarism – Textbook of Adult Emergency Medicine

Oxygen Therapy – Textbook of Adult Emergency Medicine

DIVING ENVIRONMENT

Promotor of preservation of the diving environment – attached SPUMS Presidents report

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

- Appointed convenor of SPUMS 51st Annual Scientific Meeting. **Theme:** *Diver health and Ocean health amid the storm clouds of climate change. A shared vision for underwater medicine and marine science*

RELATED INTERESTS – Marine toxinology

- Participant in IMAS marine research on recreational Fisheries and sustainability
- Speaker on marine food toxins and their effects on humans, and impact of ballast water delivering exotic marine species to remote environments
- Support for the diving operations of the free ocean carbon experiment (Antarctic Section) 2014–15
- Scombroid fish poisoning: Med J Aust. 1992;157:748–51
- Faculty member, international clinical toxinology short course – marine venoms and food poisons
- International and Australian animal venoms and poisons
- Publication: Clinical toxinology of shellfish poisoning

Prepared by Neil Banham
President, SPUMS
With the kind assistance of Annette Smart

Professor Michael Heywood Bennett AM, Conjoint, UNSW – Biography

Michael Bennett was born into a distinguished military family and followed the tradition of moving around between several primary and secondary schools in Australia and England. Despite the many schools, Michael got accepted into the medicine program at the University of New South Wales much to his parents' surprise. Michael enjoyed university life and he met friends that remain his best friends to this day. He graduated in 1979 and immediately did his junior medical rotations at the Prince of Wales and Prince Henry group of hospitals.

However, he soon felt the longing for traveling and informed the hospitals he was going to South America for a year. He was told that in that case, he should not expect to be re-employed in Sydney on his return. He was determined and embarked on a year-long exploration of South America which gave him Spanish language skills that he continues to practice today.

Michael planned to become a country GP and thought some anaesthetic training in England would be useful, so he moved to London in 1982. Anaesthesia eventually became his career because his consultants kept encouraging him to take on more jobs in the field. He stayed in England for almost ten years and gained his anaesthetic Fellowship in 1986. During his time in England, he formed many friendships and also met the love of his life, Dr Susan Pugh, while she was performing CPR. Susan completed her hospital training and later became a British GP. After Michael completed his Fellowship, he did a year in retrieval medicine in London and when he was invited to apply for a similar position in Sydney. He convinced Susan to marry him, and they started a new chapter of their life in Australia in 1990.

Since that year Michael has spent all his time working for the Prince of Wales and Prince Henry hospitals group. He initially had to do some anaesthetic re-training, but as soon as he received his Australian qualification, he was handed the keys to the hyperbaric unit by Professor Torda who recognised his interest and talent for this 'obscure' subspecialty. Michael was thriving in his new field and soon took on several leadership roles. After Michael started in his new job the Prince Henry Hospital closed and the Hyperbaric Unit moved to the Prince of Wales Hospital. This took a year and Michael used the time to get a Master's Degree in Clinical Epidemiology which has benefitted him throughout his professional life.

Michael was one of the early adopters of evidence-based medicine and became a strong supporter of the Cochrane database and systematic reviews. He embarked enthusiastically on producing evidence in diving and

hyperbaric medicine and made this the subject of his MD which he was awarded in 2006.

He has since written multiple Cochrane reviews and maintains a database of all RCTs in diving and hyperbaric medicine. This work has given him an important role on the hyperbaric oxygen therapy committee of the Undersea and Hyperbaric Medical Society (UHMS). Michael has made many academic contributions including two books, several book chapters and more than 150 refereed journal articles. However, his proudest publication was the ultimate chapter in Hyperbaric and Diving Medicine published in Harrison's Principles of Internal Medicine in 2015.

Michael Bennett is an inspirational leader to many people, and he has been a supervisor to generations of students, doctors, and Ph.D. candidates. He does not turn anybody down and if anything has a problem saying 'no'. He has been on just about every committee in both anaesthesia and diving and hyperbaric medicine and has received many awards too numerous to list. The culmination was when he was awarded a Member of the Order of Australia (AM) in 2021.

Outside his professional career, Michael has many interests including skiing, scuba diving, travelling, studying Spanish as well as enjoying good food and wine. His 'cellar cull' dinners are legendary, and we hope they will continue in Michael's long and joyful retirement. We wish him well.

Some of the milestones of MB's professional career:

Prince of Wales Hospital Clinical School

- Academic head, Department of Anaesthesia, current
- Conjoint Professor, University of New South Wales, since 2004
- Senior Staff Specialist, Anaesthesia and Diving Hyperbaric Medicine, current
- Chair, management committee, Glenn MacEnally Simulation and Learning Centre

Australia and New Zealand College of Anaesthetists

- Executive member, SIG in diving and hyperbaric medicine, 1999–2014
- Chief examiner, Certificate in diving and hyperbaric medicine
- Chairman, scholar role sub-committee, 2014–2018

South Pacific Underwater Medicine Society

- President, 2007–2014

Undersea and Hyperbaric Medicine Society (USA)

- Vice-president, 2002–2005, and 2008–2012
- Member, education committee, since 2000
- Member, hyperbaric oxygen therapy committee, since 2000

Standards Australia

- Chair, non-diving work in compressed air and hyperbaric treatment facilities, since 1998

Awards and recognition include:

- UHMS Excellence in Hyperbaric Medicine Award, 2018
- Divers Alert Network Asia-Pacific Contribution to Dive Safety Award, 2012
- Foundation Fellowship of the Undersea and Hyperbaric Medical Society, 2012
- Australian Diving and Hyperbaric Research Foundation Grant, ISSNHL, 2010
- UHMS Foundation Grant for RCT in ISSNHL, 2009
- ANZCA Citation for Services to Anaesthesia and Hyperbaric Medicine, 2002
- Albert R Behnke Award for Outstanding Scientific Achievement, UHMS, 2001

Prepared by Dr Jan Lehm

Royal Australian Navy Medical Officers' Underwater Medicine Course

Date: 16–27 October 2023, 18–29 March 2024
Venue: HMAS Penguin, Sydney

Cost: The course cost remains at AUD$1,355.00 (excl GST).

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.

For information and application forms contact:

Rajeev Karekar, for Officer in Charge
Submarine and Underwater Medicine Unit

HMAS Penguin
Middle Head Rd, Mosman
NSW 2088, Australia
Phone: *+61 (0)2-9647-5572*
Fax: *+61 (0)2-9647-511*
Email: *rajeev.karekar@defence.gov.au*

HBOE
HBOEvidence

HBOEvidence

Professor Mike Bennett is retiring and as such is seeking an interested person/group to continue his HBOEvidence site:

The database of randomised controlled trials in diving and hyperbaric medicine: hboevidence wikis.unsw.edu.au

The HBOEvidence site is planned to be integrated into the SPUMS website in the near future.

Those interested in participating in this project can contact
Mike Bennett m.bennett@unsw.edu.au
or
Neil Banham president@spums.org.au

SPUMS ASM Instagram

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The **NEW**
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.

SPUMS Diploma in Diving and Hyperbaric Medicine

Requirements for candidates (May 2014)

In order for the Diploma of Diving and Hyperbaric Medicine to be awarded by the Society, the candidate must comply with the following conditions: They must

1 be medically qualified, and remain a current financial member of the Society at least until they have completed all requirements of the Diploma;
2 supply evidence of satisfactory completion of an examined two-week full-time course in diving and hyperbaric medicine at an approved facility. The list of such approved facilities may be found on the SPUMS website;
3 have completed the equivalent (as determined by the Education Officer) of at least six months' full-time clinical training in an approved Hyperbaric Medicine Unit;
4 submit a written proposal for research in a relevant area of underwater or hyperbaric medicine, in a standard format, for approval before commencing the research project;
5 produce, to the satisfaction of the Academic Board, a written report on the approved research project, in the form of a scientific paper suitable for publication. Accompanying this report should be a request to be considered for the SPUMS Diploma and supporting documentation for 1–4 above.

In the absence of other documentation, it will be assumed that the paper is to be submitted for publication in *Diving and Hyperbaric Medicine*. As such, the structure of the paper needs to broadly comply with the 'Instructions for authors' available on the SPUMS website https://spums.org.au/ or at https://www.dhmjournal.com/.

The paper may be submitted to journals other than *Diving and Hyperbaric Medicine*; however, even if published in another journal, the completed paper must be submitted to the Education Officer (EO) for assessment as a diploma paper. If the paper has been accepted for publication or published in another journal, then evidence of this should be provided.

The diploma paper will be assessed, and changes may be requested, before it is regarded to be of the standard required for award of the Diploma. Once completed to the reviewers' satisfaction, papers not already submitted to, or accepted by, other journals should be forwarded to the Editor of *Diving and Hyperbaric Medicine* for consideration. At this point the Diploma will be awarded, provided all other requirements are satisfied. Diploma projects submitted to *Diving and Hyperbaric Medicine* for consideration of publication will be subject to the Journal's own peer review process.

Additional information – prospective approval of projects is required

The candidate must contact the EO in writing (or email) to advise of their intended candidacy and to discuss the proposed topic of their research. A written research proposal must be submitted before commencement of the research project.

All research reports must clearly test a hypothesis. Original basic and clinical research are acceptable. Case series reports may be acceptable if thoroughly documented, subject to quantitative analysis and if the subject is extensively researched in detail. Reports of a single case are insufficient. Review articles may be acceptable if the world literature is thoroughly analysed and discussed and the subject has not recently been similarly reviewed. Previously published material will not be considered. It is expected that the research project and the written report will be primarily the work of the candidate, and that the candidate is the first author where there are more than one.

It is expected that all research will be conducted in accordance with the joint NHMRC/AVCC statement and guidelines on research practice, available at: https://www.nhmrc.gov.au/about-us/publications/australian-code-responsible-conduct-research-2018, or the equivalent requirement of the country in which the research is conducted. All research involving humans, including case series, or animals must be accompanied by documentary evidence of approval by an appropriate research ethics committee. Human studies must comply with the Declaration of Helsinki (1975, revised 2013). Clinical trials commenced after 2011 must have been registered at a recognised trial registry site such as the Australia and New Zealand Clinical Trials Registry http://www.anzctr.org.au/ and details of the registration provided in the accompanying letter. Studies using animals must comply with National Health and Medical Research Council Guidelines or their equivalent in the country in which the work was conducted.

The SPUMS Diploma will not be awarded until all requirements are completed. The individual components do not necessarily need to be completed in the order outlined above. However, it is mandatory that the research proposal is approved prior to commencing research.

Projects will be deemed to have lapsed if:

- the project is inactive for a period of three years, or
- the candidate fails to renew SPUMS Membership in any year after their Diploma project is registered (but not completed).

For unforeseen delays where the project will exceed three years, candidates must explain to the EO by email why they wish their diploma project to remain active, and a three-year extension may be approved. If there are extenuating circumstances why a candidate is unable to maintain financial membership, then these must be advised by email to the EO for consideration by the SPUMS Executive. If a project has lapsed, and the candidate wishes to continue with their DipDHM, then they must submit a new application as per these guidelines.

The Academic Board reserves the right to modify any of these requirements from time to time. As of October 2020, the SPUMS Academic Board consists of:

Associate Professor David Cooper, Education Officer, Hobart
Professor Simon Mitchell, Auckland

All enquiries and applications should be addressed to:
Associate Professor David Cooper
education@spums.org.au

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

Notices and news

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

Annual Scientific Meeting 2023

After our first post-COVID EUBS Annual Scientific Meeting in Prague in 2022, we are keen to resume our previous routine of having a face-to-face meeting in a different European city every year.

The EUBS 2023 conference is scheduled for 13–16 September 2023, in the beautiful seaside city of Porto (Oporto), Portugal. The local organising committee, chaired by Dr Oscar Camacho of the Matosinhos Hyperbaric Unit and Chairman of the Portuguese Hyperbaric and Diving Medicine College, is preparing an exciting scientific and social programme. At this time we do not plan to stream the conference online, we feel that in our professional community, there is a major benefit from meeting in person, both during lectures, discussions, in and outside the meeting room and during social events.

Registrations and abstract submissions are open now, so please visit the dedicated conference website www.eubs2023.com and book your EUBS annual meeting experience as soon as possible.Your colleagues and friends will be waiting for you.

EUBS Executive Committee

Every year, a new executive committee member needs to be elected, and elections start before our next general assembly (during the EUBS Annual Scientific Meeting).

This year, in line with the EUBS constitution change approved in 2021, we will need a new Member-at-Large, who will be nominated for a period of four years.

Candidates will be presented to the executive committee by 15 June 2023, and the voting will be, as usual, by internet ballot, starting on 30 June 2023. If you want to contribute and help our society, please come forward and send your short CV to our secretary (secretary@eubs.org) before 1 June 2023.

If you do not want to present yourself, why not nominate someone else? Suggestions are welcome at the same email address.

EUBS and ECHM joint position statement on the use of 'mild hyperbaric therapies' in humans

Within the European Union hyperbaric chambers are regarded as 'class IIb medical device' according to the Medical Devices Regulation (MDR) and must meet strict safety standards to prevent harm to patients, caregivers, and third parties.

ECHM and EUBS have published a joint position statement on the use of pressure chambers marketed as 'low-pressure hyperbaric chambers' and claimed to be beneficial for a wide range of effects and wellness purposes.

You can download the document from the ECHM website and from the EUBS website on the EUBS Endorsed Docs and Guidelines page (http://www.eubs.org/?page_id=227). The document is available in several languages already (English, Spanish, Greek, Dutch, French, Czech, Hungarian [Magyar] and German). More languages will be added as translations come in and are approved.

Please distribute this document freely, as widely as possible.

EUBS Affiliate society agreements

For 2023, the agreement has been renewed with the following scientific societies in order to promote membership and contact among the hyperbaric and diving scientists and practitioners in Europe and worldwide. Members of these societies benefit from a 10% reduction on the EUBS membership fees when providing proof of their membership of the 'other' society. Simply indicate the Affiliate Society from the drop-down list on the EUBS membership application or renewal form.

Belgian Society for Diving and Hyperbaric Medicine (http://www.sbmhs-bvoog.be)
Scott Haldane Foundation, The Netherlands (http://www.scotthaldane.org)
Italian Society for Diving and Hyperbaric Medicine (http://www.simsi.it/)
German Society for Diving and Underwater Medicine (http://www.gtuem.org)
French Society for Diving and Hyperbaric Medicine (http://www.medsubhyp.com)

Swiss Society for Underwater and Hyperbaric Medicine (http://www.suhms.org)
Undersea and Hyperbaric Medical Society (http://www.uhms.org)
Spanish Society for Diving and Hyperbaric Medicine (www.asemhs.org)
Austrian Society for Underwater and Hyperbaric Medicine (www.asuhm.at)
Dutch Society for Diving Medicine (www.duikgeneeskunde.nl)

We are pleased to announce that in exchange, EUBS members benefit from a substantial reduction to their UHMS membership – simply mention your EUBS membership when enrolling/renewing your UHMS membership.

EUBS website

Please visit the EUBS Website for the latest news and updates. The 'EUBS History' section (under the Menu item 'The Society') is still missing 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.

By popular demand, EUBS members can also download the complete abstract book of previous EUBS meetings from the Members area.

While on the EUBS website, make sure you take a look at our Corporate Members' webpage (http://www.eubs.org/?page_id=91). On this page, logos and links are placed of those organisations, societies and companies that support EUBS financially. EUBS is grateful for their continuing support and would suggest that if you contact any of them, please do so by clicking on the link at that page, so they'll know that you did through the EUBS website.

OXYNET Database

Since 2004, a public online database of European Hyperbaric Chambers and Centers has been available, started and initially maintained by the OXYNET Working Group of the COST B14 project of the European Commission, later by the European Committee for Hyperbaric Medicine (ECHM). The original website www.oxynet.org will soon no longer be accessible, and the full OXYNET database of hyperbaric centers has been placed on the EUBS website (http://www.eubs.org/?page_id=1366).

If you have updated information or any other request or remark, please send an email to oxynet@eubs.org. If you can collect information for more than one center in your area or country, please do.

The

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.


13-16
SEPTEMBER
DIVENTOS
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EUBS
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PORTO 2023

Courses and meetings

Scott Haldane Foundation

As an institute dedicated to education in diving medicine, the Scott Haldane Foundation (SHF) has organised more than 300 courses all over the world, over the past 30 years. SHF is targeting an international audience with courses worldwide

We are happy that the world has reopened after the COVID-19 pandemic and we can announce courses around the world again. Below is the schedule of upcoming SHF courses in 2023.

The courses Medical Examiner of Divers (parts 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).

2023 (first half)

24–25 March	Medical Examiner of Divers part 1 (level 1), The Netherlands
30 March–1 April	Medical Examiner of Divers part 2 (level 1), The Netherlands
13–20 May	Medical Examiner of Divers part 2 (level 1) Bonaire, Dutch Caribbean
9–10 June	In-depth course Nightmares for the Diving Doc (level 2d), The Netherlands
On request	Internship HBOt (level 2d certification) NL/Belgium

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

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

EUBS and SPUMS members are able to access the German Society's large database of publications in diving and hyperbaric medicine. EUBS members have had this access for many years. SPUMS members should log into the SPUMS website, click on 'Resources' then on 'GTÜM database' in the pull-down menu. In the new window, click on the link provided and enter the user name and password listed on the page that appears in order to access the database.

The Science of Diving

Support EUBS by buying the PHYPODE book '*The science of diving*'. Written for anyone with an interest in the latest research in diving physiology and pathology. The royalties from this book are being donated to the EUBS.

Available from:
Morebooks
https://www.morebooks.de/store/gb/book/the-science-of-diving/isbn/978-3-659-66233-1

Historical Diving Society Australia - Pacific

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

DHM Journal Facebook

Find us at:
https://www.facebook.com/divingandhyperbaricmedicine

Diving and Hyperbaric Medicine: Instructions for authors (full version)
(updated February 2023)

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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European Editor: euroeditor@dhmjournal.com
Editorial Manager: editorialassist@dhmjournal.com
Journal information: info@dhmjournal.com

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

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

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 will be considered. 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),

The main text of the manuscript should be divided into **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.

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 above.

Title page: Irrespective of article type, it must have a title page that lists the title of the paper, all authors' names in full and their affiliations and provide full contact details for the first (and corresponding, if different) author(s).

ORCiD requirements: ORCiD An ORCiD is now required for all corresponding authors when submitting to *Diving and Hyperbaric Medicine*. The ORCiD must be entered into Manuscript Manager when submitting (the site will prompt you to create one if you do not have one). Please add your ORCiD to the title page of your manuscript.

What is an ORCiD? ORCID provides a persistent digital identifier (an ORCID iD) that you own and control, and that distinguishes you from every other researcher. You can connect your iD with your professional information – affiliations, grants, publications, peer review, and more. You can use your iD to share your information with other systems, ensuring you get recognition for all your contributions, saving you time and hassle, and reducing the risk of errors. For more information see https://orcid.org/.

Keywords: The title page must also list a maximum of seven keywords best describing the paper. These should be chosen from the list on the journal website DHM Keywords 2023 or on the Manuscript Manager website. New keywords, complementary with the US National Library of Medicine NML MeSH, https://www.nlm.nih.gov/mesh/meshhome.html/ may be used but are at the discretion of the Editor. Do not use keyword terms that already appear in the title of your article.

Text format: The preferred format is Microsoft Office Word or rich text format (RTF), with 1.5 line spacing, using both upper and lower case throughout. The preferred font is Times New Roman, font size 11 or 12. Please avoid using auto-formatting tools such as automatic spaces before and after paragraphs. Lines must be numbered continuously throughout the manuscript to facilitate the review process.

Section Headings should conform to the current format in DHM
This is:
Section heading (for Introduction, Methods, etc.)
SUBSECTION HEADING 1
Subsection heading 2

Numbering: All pages must be numbered, but no other text should appear in the header and footer space of the document. Do not use underlining. No running title is required.

English spelling will be in accordance with the Concise Oxford Dictionary, 11th edition revised (or later). Oxford: Oxford University Press; 2006.

Measurements will be in SI units (mmHg are acceptable for blood pressure measurements) and normal ranges should be included where appropriate. Authors are referred to the online BIPM brochure, International Bureau of Weights and Measures (2006), The International System of Units (SI), 8th ed, available as a pdf at https://www.bipm.org/en/publications/si-brochure/. Atmospheric and gas partial pressures and blood gas values should be presented in kPa (atmospheres absolute [abbreviated as atm abs]/bar/mmHg may be provided in parenthesis on the first occasion). The ambient pressure should always be given in absolute not gauge values unless there is a particular reason to use gauge pressure and the distinction is made clear. Water depths should be presented in metres of sea (or fresh) water (msw or mfw). Cylinder pressures may be presented as 'bar'.

Abbreviations may be used once they have been shown in parenthesis after the complete expression. For example, decompression illness (DCI) can thereafter be referred to as DCI. This applies separately to the abstract and main text. Use generally accepted abbreviations that readers are likely to be familiar with rather than neologisms of your own invention. The overuse of abbreviations is strongly discouraged.

References: References should be numbered consecutively in the order in which they are first mentioned in the text, tables or figures where they should appear as superscript numbers, either following the statement referenced,1 or at the end of the sentence, after the full stop.[1,2] Do not use references in the Abstract. References appearing in tables or figures or their legends should continue the sequence of reference numbering in the main text of the article in accordance with the position of first citing the table/figure in the text. Use MEDLINE abbreviations for journal names. Journals not indexed in MEDLINE should have the journal name written in full.

The Journal reference style is based exactly on that of the International Committee of Medical Journal Editors (ICMJE) Recommendations for the Conduct, Reporting, Editing and Publication of Scholarly Work in Medical Journals:Sample References (updated April 2018) https://www.nlm.nih.gov/bsd/uniform_requirements.html. Examples of the formats for different types of references (journal articles, books, monographs, electronic material, etc.) are given in detail on this website. Authors MUST consult this in preparing their reference list.

An example of a journal reference in the ICMJE format is:

Wilson CM, Sayer MDJ. Transportation of divers with decompression illness on the west coast of Scotland. Diving Hyperb Med. 2011;41(2):64–69.

If a journal uses continuous pagination throughout a volume (as many do) then the month and issue number should be omitted and the pagination reduced. Therefore, the shortened ICMJE version used in DHM is:

Wilson CM, Sayer MDJ. Transportation of divers with decompression illness on the west coast of Scotland. Diving Hyperb Med. 2011;41:64–9.

If an article has a unique identifier for the citation (e.g., doi number, PubMed PMID, PubMed Central PMCID) then this must be included at the end of the reference. The format and order for this is:
doi: number. PMID: number. PMCID: number. For example:

Doolette DJ, Mitchell SJ. In-water recompression. Diving Hyperb Med. 2018;48:84–95. doi: 10.28920/dhm48.2.84-95. PMID: 29888380. PMCID: PMC6156824.

An example book reference is:

Kindwall EP, Whelan HT, editors. Hyperbaric medicine practice, 3rd ed. Flagstaff (AZ): Best Publishing Company; 2008.

Examples of many other types of references are to be found on the National Library of Medicine site (see link previous page).

When citing workshop/conference proceedings or technical reports, authors are requested to investigate their availability online and provide an online source for the reference if available. The date that the reference was cited (year/month/day) from the source should be noted. For example:

Mitchell SJ, Doolette DJ, Wacholz CJ, Vann R, editors. Management of mild or marginal decompression illness in remote locations workshop proceedings. Durham (NC): Divers Alert Network; 2005. [cited 2022 May 5]. Available from: https://world.dan.org/wp-content/uploads/2021/06/remotewrkshpfinal05-1.pdf.

Additional notes regarding referencing in DHM are:

If using EndNote to prepare the references in the document see EndNote website for advice. Once accepted, the final version of the submitted text should have all EndNote field codes removed.

Verifying the accuracy of references against the original documents is the responsibility of authors.

Personal communications should appear as such in the text and not be included in the reference list (e.g., Smith AN, personal communication, year).

Abstracts from meeting proceedings should not be used as references unless absolutely essential, as these are generally not peer-reviewed material.

Please avoid using auto-formatting functions like numbering, indentations, and spaces before and after paragraphs in compiling your reference list.

Tables must not be embedded in the main manuscript document. They are to be uploaded as separate Word documents (one document per table) in Manuscript Manager (use the 'other' category when asked to select a description of the document being uploaded). Name the document with the first author's name and table number as appropriate. Tables need to be labelled at the top of the page with first author name and the Table number.

Tables should be presented using MS Word table format with frames shown, auto-formatted to fit content. Please avoid complicated, large tables whenever possible. Very large tables (full page or more) may not be incorporated into the final article but, rather, displayed in the journal website as additional material at the Editor's discretion.

The title of the table and caption are not to be included in the table. These appear in the 'legends and captions' section at the end of the manuscript document. Legends should generally contain fewer than 40 words and should be thorough enough to be understood independently of the main text.

The table must be mentioned within the text of the article, e.g., "Differences in rates of decompression illness were not significant (Table 1)", etc. The approximate positions of tables and figures should also be identified in the manuscript text.

Figures (including photos, graphs, diagrams, illustrations and radiographs) must not be embedded in the main manuscript document. They are to be uploaded as separate electronic files in high resolution preferably TIF but DHM accepts JPEG format in Manuscript Manager. Name the document with the first author's name and figure number as appropriate. Figures should be uploaded to Manuscript Manager in their numbered order, which results in them being compiled in the review document in correct order.

The title of the figure and caption are not to be included in the figure. These appear in the 'legends and captions' section at the end of the manuscript document. Legends should generally contain fewer than 40 words and should be thorough enough to be understood independently of the main text. Magnification should be indicated in the captions for photomicrographs, and consideration given to the positioning of labels on diagnostic material as this can greatly influence the size of reproduction that can be achieved in the published article.

Graphs may be submitted either in colour or grey-scale, with no unnecessary shading, grid lines or box lines. Please choose the simplest graphical format that displays the data effectively. 3-D graphs are discouraged unless they are necessary to display 3-D data. Both markers and lines should be unique to facilitate easy discrimination of the data being presented. Special attention should be given to ensuring that font sizes within a diagram are sufficiently large to be legible should the diagram be sized for single-column presentation. The preferred font in diagrams and graphs is Times New Roman. Graph symbol keys should appear within the white space of the figure (not outside the axes) if possible or be included in the legend. Please ensure that axes are labelled using sentence case and the same data formatting conventions presented below.

Any graphs or histograms created in Excel should be sent within their original Excel file, including the data table(s) from which they were produced. This allows the journal office to edit figures for maximum legibility when printed. Upload the spreadsheet to Manuscript Manager with the other manuscript documents and select the designator 'other' and the option 'hide from reviewers' so that the spreadsheet is not incorporated in the review document.

Any photograph or radiograph of a patient must be de-identified. Patient details must be removed, and photographs made unrecognisable. Colour photos are acceptable.

If any figures, images or tables are to be reproduced from previous publications, it is the responsibility of the author(s) to obtain the necessary permissions. This permission should be acknowledged in the figure caption using the format "*Reproduced with permission of* ….." or, if necessary, another format specified by the copyright holder granting permission.

Miscellaneous data formatting conventions: Please follow the following recommendations when presenting data in text, figures or graphs.

Standard deviations and standard errors should be expressed as mean (SD), not mean ± SD.

Composite units of measurement should be expressed as (for example) g·L-1 or mL·kg-1·min1, not g/L or mL/kg/min Please use a space between symbols like <, >, ≤, ≥. Thus (for example) > 25, not >25.

Please use decimal points and not commas in decimals. For example: 2.5, not 2,5.

Numbers greater than 999 should contain commas. For example: 1,000 or 25,300,000.

Please leave a space between a number and unit of measurement. For example: 25 msw.

Please italicise *n* when used to indicate number and *P* when used to indicate *P*-values.

Please leave spaces in expressions like $n = 25$ or $P < 0.05$ (not n=25 or P<0.05).

For number ranges please use an en-dash without spaces. For example: 17–420. This also applies to page ranges when citing references.

Percent signs should immediately follow a number without a space. For example: 51% not 51 %.

Other manuscript requirements and guidelines

DHM follows as much as possible the Recommendations for the conduct, reporting, editing and publication of scholarly work in medical journals. International Committee of Medical Journal Editors; December 2015.

Available from: http://www.icmje.org/recommendations/. Authors are strongly encouraged to read this and other documents on the ICMJE website in preparing their submission. Authors should also consult guidelines for specific types of study (e.g., the CONSORT guidelines for the reporting of randomised controlled trials); see http://www.equator-network.org/.

Trial design, analysis and presentation. Before preparing their manuscript, authors must read the summary advice on the journal website on the reporting of trial design, sample size calculation, statistical methods and results. http://www.dhmjournal.com/images/Docs/Trial-design-analysis-and-presentation.pdf.

Consent and ethical approval. Studies on human subjects must comply with the Helsinki Declaration of 1975, revised October 2013 (see https://www.dhmjournal.com/index.php/author-instructions for a copy).

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. It is insufficient to refer to previous publications for details of animal welfare and procedural care. The Physiological Society provides detailed advice regarding animal experimentation and its reporting in research publications and this link is provided with their kind permission: https://physoc.onlinelibrary.wiley.com/doi/full/10.1113/jphysiol.2010.192278.

A statement affirming Ethics Committee (Institutional Review Board) approval (and the approval number) should be included in the text at the beginning of the methods section. A copy of that approval should be uploaded with the submission. Similarly, a statement affirming the securing of written informed consent from subjects should be included in the methods where this was part of the methodology.

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/ or EudraCT in Europe https://eudract.ema.europa.eu/. Details of the registration must be provided in the accompanying MSF, and should also be mentioned in the methods section.

For individual case reports, evidence of informed patient consent to anonymous publication of their clinical details

and/or images, etc. must be provided. Case series, where only limited anonymous summary data are reported do not require patient consent, but must have been assessed by an ethics committee and, if indicated, have ethics approval. Consult your local ethics committee if you are unsure.

Authorship: Authors must have contributed significantly to the study (see guideline to authorship at: https://www.dhmjournal.com/images/Docs/Guideline_to_authorship_in_DHM_journal-2015.pdf.

Inclusion of more than six authors in any one manuscript requires strong justification. Other contributors may be listed in the Acknowledgements section.

Mandatory submission form (MSF): A fully completed MSF must be signed by the first author and the corresponding author (if different) and must be uploaded with other manuscript documents in Manuscript Manager with all submissions, irrespective of type. Authors should be listed with the principal author first. Authors should be listed on the MSF in the order intended for the published paper. The form requires the full postal address, phone number and e-mail address supplied for the first author; if the corresponding author is not the first author, then full contact details for both are required. The MSF is available for download on the DHM website.
https://www.dhmjournal.com/index.php/author-instructions

Conflict of interest form: All conflicts of interest by any author must be reported in summary in the Mandatory Submission Form. If your paper is accepted and any conflicts have been listed here, then more detailed information will be required using the ICMJE form available on the ICMJE website at: http://www.icmje.org/coi_disclosure.pdf. A form for each author for whom a potential conflict was listed must be submitted.

All potential conflicts of interest, financial or otherwise (e.g., consultancies, equity interests, patent-licensing arrangements, lack of access to data, or lack of control of the decision to publish) by any author must be declared. DHM reserves the right to seek further clarification as necessary. All conflicts or a declaration of no conflicts will appear at the end of the published article. Failure to report potential conflicts of interest prior to peer review may result in publication delays or rejection of the manuscript.

Authors should consult the WAME website http://www.wame.org/about/conflict-of-interest-in-peer-reviewed-medical if they need further clarification.

Peer review and publication process. All submitted manuscripts will be subject to open peer review usually by a member of the Editorial Board and/or external reviewers. Reviewer comments will be provided to authors with any recommendations for improvement before acceptance for publication, or if the article is rejected. DHM believes that a transparent review process is indicated in such a small specialty; reviewers are often able to identify the origin of manuscripts and, in the interests of fairness, the authors are, therefore, generally provided the names of their reviewers. The review process typically takes about eight weeks but can be longer. If additional reviews are needed, this will prolong the process. Papers are generally scheduled for publication in order of final acceptance. The Editor retains the right to delay or expedite publication in the interests of the Journal.

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6 Excel spreadsheet with data and graphs if graphs have been generated in Excel.
7 Submission letter; authors can use this to communicate any particular considerations or issues they wish the editor to be aware of in relation to their manuscript. The letter should state that the paper is being submitted exclusively to DHM.

Documents on DHM website

The following files are available on the DHM website to assist authors in preparing their submission:

Instructions for Authors 2023 (this document)
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Trial design analysis and presentation
English as a second language
Guideline to authorship in DHM 2015
Helsinki Declaration revised 2013
Is ethics approval needed?

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Scholarships for Diving Medical Training for Doctors

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