Diving and Hyperbaric Medicine

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EUBS

Professor Michael Heywood Bennett AM, 1956–2023

Australian diving fatalities

Hyperbaric chamber ventilation

Hookah evaluation after a fatal accident

The EMMA capnometer in hyperbaric chambers

HBOT effect on osteoporosis in vivo

Fitness after mild COVID-19

A new hyperbaric ventilator

Hyperbaric medicine teaching at Canadian medical schools

Rebreather Forum Four consensus statements

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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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The Editor's offering

The premature death of Professor Michael Bennett is a seismic event in the field of hyperbaric medicine. There is an obituary for Mike in this issue of the journal, but I'm invoking editor's privilege to provide a focused personal perspective on his contribution to our field.

Mike was a consummate scientist; careful, methodical, and self-critical. We have been enlightened by his substantial body of original work, but his essential legacy to the field is the legitimacy he earned us in the wider medical community through his advocacy for evidence-based practice. Hyperbaric medicine historically has been (and there's the ongoing potential to be) discredited by the activities of practitioners advocating hyperbaric oxygen treatment (HBOT) for unproven, disproved or implausible indications. Mike knew this and to our great benefit fought back against it. Among his many publications he published more than 50 review articles or systematic reviews containing scrupulously objective appraisal of evidence in support (or not) of various proposed indications for HBOT. He also created and maintained a freely available website hosting reviews of relevant randomised studies. In doing so he, more than anyone else in the field, has contributed to a perception among our colleagues in the wider medical community that we are a legitimate discipline and not alternative medicine practitioners. There is no better evidence for this than his rare invitation for a non-American to contribute a chapter on "*Hyperbaric and Diving Medicine*" to the iconic medical textbook Harrison's Principles of Internal Medicine. Prior to this our field had little or no presence in the most influential general medical texts. That chapter, first published in 2011 (18th edition), has persisted through four editions and in the most recent (21st edition) was included in the print version of this standard work for the first time.[1] Mike's approach to evidence-based medicine was pragmatic; indeed, he sensibly believed that the quality of evidence in support of using HBOT to treat a particular problem should match the prevalence of that problem. It follows that he didn't insist that every potential indication had to be supported by randomised trials. One can only hope that leaders in our field recognise the priceless nature of the recognition Mike has earned us, and don't condone or reward the activities of anyone more concerned with profit than efficacy.

I will personally remember Mike as the wittiest, funniest, most starbright, academically generous colleague and friend I have ever known or had the pleasure to work with. He was a very close friend and I feel a deep sense of loss on his passing, both personally and professionally; indeed, as for many of Mike's friends, this sense is still evolving and we still struggle to fully comprehend that it has happened. Our grief can only pale in comparison to that of Mike's soulmate Sue and his wider family. On behalf of the journal and its parent societies I offer them our most sincere condolences.

In this issue there are articles on a wide variety of topics.

John Lippmann and colleagues publish another paper in the thematic area of diving accident analysis in comparing recent Australian diving fatalities with those in an earlier era. The rise in the importance of age, obesity and cardiac disease as contributing factors is an interesting finding. Lyubisa Matity and colleagues provide a technical but fascinating insight into the field of hyperbaric chamber ventilation, and reveal that ventilation may not conform to the predictions of well-stirred models. Darren Meehan and colleagues provide a template for evaluation of 'hookah' surface supply breathing apparatus following a fatality. Alicia Tucker and David Smart present a detailed evaluation of the EMMA capnometer as a potential solution to the long-standing challenge of measuring end tidal CO_2 in ventilated patients in a hyperbaric chamber. Xiaoling Peng and colleagues showed that HBOT on its own or in combination with exercise ameliorated bone microarchitecture deterioration in a rat model of osteoporosis. Given that exercise on its own seemed as effective, there must be some question about the implications for HBOT as a therapeutic intervention. Nevertheless, one of the reviewers, a 'non-hyperbaric' world authority on osteoporosis and bone metabolism, pointed out the highly interesting mechanistic data included in this paper. Jan Peter Schaap and colleagues publish a highly topical paper suggesting that in terms of fitness, there is little or no difference between hyperbaric military personnel who had suffered mild COVID-19 and those who had not. There are also articles on a new hyperbaric compatible ventilator, the lack of presence of hyperbaric medicine in Canadian medical school curricula, the Rebreather Forum Four consensus statements, and three fascinating case reports (another left ventricular assist device in the hyperbaric chamber, glans penis ischaemia treated with HBOT and hemiplegia as a manifestation of carbon monoxide poisoning.

Congratulations to SPUMS and the UHMS for running their excellent annual meetings in the last month. I am greatly looking forward to meeting colleagues at the EUBS meeting in Porto in September.

Reference

1 Bennett MH, Mitchell SJ. Hyperbaric and diving medicine. In: Jameson J, Fauci AS, Kasper DL, Hauser SL, Longo DL, Loscalzo J, editors. Harrison's principles of internal medicine. 21st ed. New York: McGraw-Hill; 2022. p. 3623–30.

Professor Simon Mitchell
Editor

Cover photo:

Professor Michael Heywood Bennett AM, 1956–2023.

Original articles

Compressed gas diving fatalities in Australian waters 2014 to 2018

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Keywords
Carbon monoxide; Cardiovascular; Diving deaths; Fitness to dive; Obesity; Scuba

Abstract
(Lippmann J, Lawrence C, Fock A. Compressed gas diving fatalities in Australian waters, 2014 to 2018. Diving and Hyperbaric Medicine. 2023 June 30;53(2):76–84. doi: 10.28920/dhm53.2.76-84. PMID: 37365124.)
Introduction: This study aimed to investigate compressed gas diving deaths in Australia from 2014–2018 and make comparison to those from 2001–2013 to identify ongoing problems and assess countermeasures.
Methods: Media reports and the National Coronial Information System were searched to identify scuba diving deaths for 2014–2018, inclusive. Data were extracted from the witness and police reports, medical histories, and autopsies. An Excel® database was created and a chain of events analysis conducted. Comparisons were made with the earlier report.
Results: Forty-two fatalities were identified, 38 using scuba and four using surface-supplied breathing apparatus involving 30 males and 12 females. The mean age of victims was 49.7 years, six years higher than the previous cohort. Fifty-four percent were obese. Six victims were unqualified, three were under instruction and at least 28 were experienced divers, significantly more than in the previous cohort. Health-related predisposing factors, predominantly obesity and cardiac-related, were identified as likely contributory to 26 incidents, and planning shortcomings to at least 22 deaths. One-third of the disabling conditions were primary drowning and one-quarter were cardiac. Three divers died subsequent to carbon monoxide poisoning and three likely from immersion pulmonary oedema.
Conclusions: Advancing age, obesity and the associated cardiac disease have become increasingly prevalent in diving fatalities and the need for appropriate assessment of fitness to dive is evident.

Introduction

Scuba diving attracts a wide cross-section of the community, from pre-teen children to the elderly, with the Professional Association of Diving Instructors (PADI) reportedly issuing one million certifications per year.[1] It is difficult to determine the number of active Australian divers, and estimates have ranged from around 85,000 to 400,000.[2,3] These estimates were based on surveys, and the wide variability results largely from the different timings, sampling procedures and sample sizes (16,000 respondents vs 1,600 respectively) and subsequent extrapolation, highlighting the difficulty of obtaining reliable activity estimates for a broad population. On the other hand, the reporting of the number of diving fatalities in Australia is relatively accurate because of routine police and coronial investigation and documentation. From 1972 to 2018 inclusive, there was an average of 8.7 scuba deaths and 1.6 surface-supplied breathing apparatus (SSBA) deaths per year.[4] A previous analysis of 126 scuba deaths from 2001–2013 highlighted the increasing age of victims, the prevalence of pre-existing medical conditions, inexperience, a poor buddy system, and failure to ditch weights in an emergency as key issues.[5] This current report investigates scuba and SSBA fatalities over the subsequent five-year period, compares these to the previous period, to determine the ongoing problems and assess countermeasures.

Methods

This represents a complete, or near-complete, case series of scuba and SSBA deaths in Australia from 1 January 2014 to 31 December 2018. For inclusion, the diver must have been reported to have been wearing a scuba set or using SSBA.

ETHICS APPROVAL

Ethics approvals for the collection and reporting of these data were received from the Victorian Department of Justice Human Research Ethics Committee to access the National Coronial Information System (NCIS; CF/21/18434).[6]

SEARCH

A comprehensive keyword search was made of the NCIS for scuba diving-related deaths throughout Australia for the period 1 January 2014 to 31 December 2018. Keywords included scuba, compressed air, and compressed gas and div*. Data obtained from the NCIS was matched with that listed on the Australasian Diving Safety Foundaton (ADSF) fatality database[4] obtained via media or word of mouth.

REVIEW PROCEDURE AND OUTCOME MEASURES

The investigator reviewed all datasets, a range of outcome measures were extracted for each case and entered into a specially created anonymised and protected Microsoft Excel® spreadsheet. Where available, these data included demographics, health factors, training and experience, the origin of victims, dive location and conditions, buddy circumstances and oversight, dive purpose and depth, equipment used and resuscitation factors.

ANALYSIS

A chain of events analysis (CEA) was performed for each case using existing templates with minor modifications.[7]

Descriptive analyses based on means and standard deviations or medians and ranges, and *t*-tests, $\chi 2$ and Mann-Whitney U tests for comparisons of age or body mass index (BMI), as appropriate, were conducted using SPSS Version 28.0.1.1 (IBM Armonk, NY; 2021). The level of statistical significance assumed was $P = 0.05$.

Results

There were 42 recorded compressed gas diving-related fatalities throughout Australia from 2014 to 2018. These comprised 38 scuba divers, two of whom were using closed circuit rebreathers (CCRs) and one individual who was doing scuba training but was not wearing a scuba unit but is included for completeness. Four divers were using SSBA (hookah). Brief summaries of the cases can be found at Appendix 1.

DEMOGRAPHICS

There were 30 male and 12 female victims. The mean (SD) age was 49.7 (11.5) years with males on average two years older than females (50.2 vs 48.5 years) ($P = 0.92$).

The mean (SD) BMI (available for 35 divers) was 29.1 (4.4) $kg{\cdot}m^{-2}$ and slightly higher for females (30.1 vs 28.7 $kg{\cdot}m^{-2}$). Nine of the victims (seven men and two women) were classified as overweight (BMI 25–29.9 $kg{\cdot}m^{-2}$), and 19 (12 men and seven women) were obese (BMI ≥ 30 $kg{\cdot}m^{-2}$).

CERTIFICATIONS AND EXPERIENCE

The level of certification was unreported in eight cases. Six of the victims were unqualified, three of these were under instruction; two undergoing open water training and one was on an introductory scuba dive. Thirteen victims held ‘open water’ (OW) certifications, five had ‘advanced open water’ (AOW) certifications, four were instructors, one a divemaster, and one a qualified commercial diver. Three divers had no experience, seven were novices (≤ 30 dives), 16 were experienced (30–199 dives), and 12 were very experienced (≥ 200 dives). Experience was unreported for four of the victims.

ORIGIN, LOCATION, SETTING AND ACTIVITY

Thirty-one of the victims were diving locally, eight were overseas tourists (seven of whom were diving in Queensland), and three were interstate tourists. Queensland and Victoria had the highest number of deaths with 11 each, followed by New South Wales and Western Australia, each with eight deaths. There were two deaths in South Australia and two in Tasmania. Twenty-seven of the deaths were in a private setting and the remaining 15 occurred in a commercial setting, including Queensland (7), New South Wales (4), Victoria (3) and Western Australia (1).

Twenty of the victims were underwater sightseeing, one in a wreck and another in a cave. Fourteen were harvesting seafood (mainly crayfish) and six deaths were associated with training or an introductory scuba experience. One death appeared to have been suicide.

The training-related deaths included a double fatality (instructor and student) who both drowned during an open water training dive, a student who suffered a heart attack after the ‘swim test’ in a pool, an OW student who likely suffered immersion pulmonary oedema (IPO) and a student who drowned while undergoing commercial dive training. One victim died after separating from their instructor in poor visibility on an introductory scuba dive.

BUDDY AND SUPERVISION CIRCUMSTANCES

Eleven victims had set out diving solo, 17 with a buddy, and 14 in a group. Fifteen divers had separated from their buddy or group before the incident, at least four of these intentionally to ascend or hunt crayfish and one to collect more scallops. Another five separated during the incident. Overall, only 11 victims were with their buddy or buddies at the time of their demise.

Twenty-two divers were under some supervision, including 12 of the 14 who were diving in a commercial setting. Six of the 12 solo divers had some supervision, from either a boat or the shore.

DEPTH AND BREATHING GAS SUPPLY

Of the 35 incidents where the dive depths were reported, more than one-third were to depths of ≤ 10 metres of seawater (msw), and two-thirds were ≤ 20 msw, with the deepest reported depth being 39 msw. At least 14 of the deaths occurred either at or very near the surface.

All the victims were breathing air, except one CCR diver who was set up for breathing oxygen but had not opened the valve. In the 32 incidents for which the remaining gas supply was reported and relevant, 28 divers had sufficient supply to reach the surface and four had exhausted their breathing gas.

EQUIPMENT TESTING AND BREATHING GAS ANALYSIS

Thirty-three reports indicated that the diver's equipment had been tested and no significant faults were found in 26 of these, other than the cylinder valves which had not been opened in two cases. Faults were reported in seven cases and included buoyancy compensator device (BCD) malfunctions, leaking regulators, torn mouthpieces, high-demand valve breathing resistance and a severed SSBA hose.

The results of a cylinder breathing gas analysis were reported in 20 cases, 15 of which met the required air purity standards.[8] However, elevated water vapour was found in three cylinders and potentially lethal levels of carbon monoxide (CO) and carbon dioxide in another.

POSITIVE BUOYANCY ATTEMPTS

Of the 34 divers who were known to be wearing a BCD, four were found with an inflated BCD, 24 divers' BCDs were not inflated, and in the other cases, the state of BCD inflation was not reported. At least 28 divers were found still wearing their weights and seven had ditched their weights. In the remainder, the weight circumstances were not reported. Only two victims were reported to have both inflated their BCDs and ditched their weights.

RESCUE AND RESUSCITATION

A rescue attempt (i.e., the victim was accessed and landed relatively quickly, with an arguable possibility of survival) was made with 27 (two-thirds) of the victims and all but one of the bodies of the remaining divers were later recovered after extended submersion periods of up to 24 hours. Water, regurgitated stomach contents and/or froth which required management was present in the airways of more than half of the rescued scuba divers. Basic life support (BLS) was attempted in 28 cases. It was not performed in other cases due to the delays in body recoveries and the absence or condition of the bodies. Not all victims who were 'rescued' received BLS, and BLS was performed on some victims whose bodies were recovered after an extended period of submersion.

An automatic external defibrillator (AED) was available on site and used by staff or bystanders (sometimes medical) in seven cases, five of which were in Queensland where they are mandated in a commercial setting. Shocks (1–4) were delivered in three cases.

CHAIN OF EVENTS ANALYSIS

Predisposing factors

Seventy-nine predisposing factors were identified in 40 of the incidents, the main ones being health-related which were identified in 26 incidents, often with multiple factors present. The most common health factors were obesity (19), ischaemic heart disease (IHD) (9), left ventricular hypertrophy (LVH) (8) and cardiomegaly (7). Alcohol intoxication directly contributed to one death and recreational drugs were possible contributors to another three. Mental health conditions were implicated in three deaths.

Planning shortcomings likely contributed to 22 fatalities, including eight where it should have been apparent that the conditions were unsuitable. Six divers set out solo, another two with intentionally loose buddy systems, and, with four divers, the incident occurred after they had intentionally separated during the dive. Other planning shortcomings included the decision to dive near fishing activities, poor suitability and positioning of SSBA equipment, and the decision to enable an untrained friend with substantial health conditions to try scuba.

Lack of training, poor skills and/or inexperience likely contributed to at least 11 deaths, three of which occurred under the supervision of an instructor, and another under a friend with no instructional certification. One of the victims was self-taught and the remainder were certified but had insufficient overall, or recent experience for the dive undertaken. At least seven of these deaths resulted in primary drowning and one in cerebral arterial gas embolism (CAGE), both of which are often associated with the inexperienced.

Equipment-related issues contributed to 10 of these deaths, the most common being leaking cylinder valves (3), overweighting (2), faulty demand valves (2), and poor SSBA air intake setups (2). Another case involved contaminated cylinder air from a poorly maintained and positioned compressor.

Inadequate supervision was identified as a factor in nine incidents. These included five instances of poor surface oversight with a variety of outcomes which included a boat reversing onto a diver, and failure to notice a hookah compressor malfunction, among others. Failures with in-water supervision by instructors were associated with three deaths.

Organisational shortcomings predisposed to at least two fatalities. One involved inadequate maintenance and

Figure 1
Likely disabling agents associated with 35 of 42 scuba fatalities; no disabling agents could be identified in seven incidents

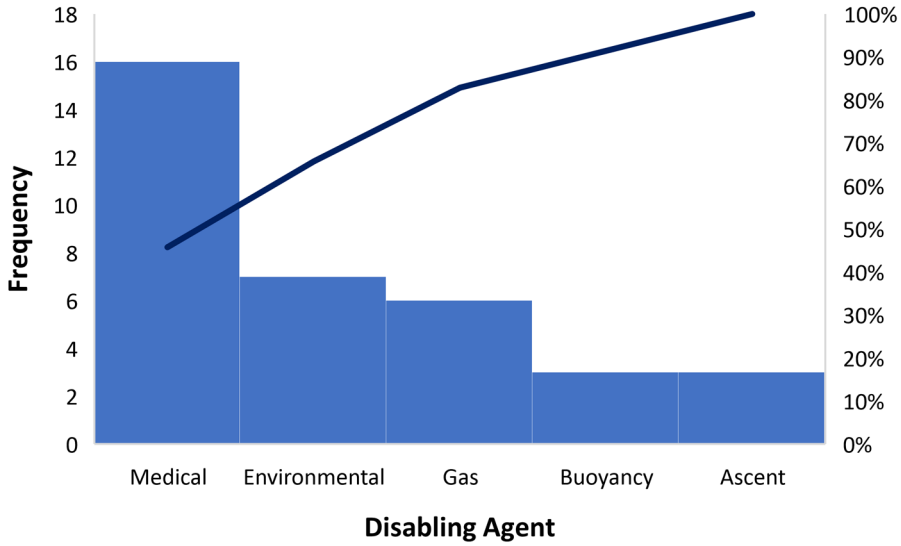
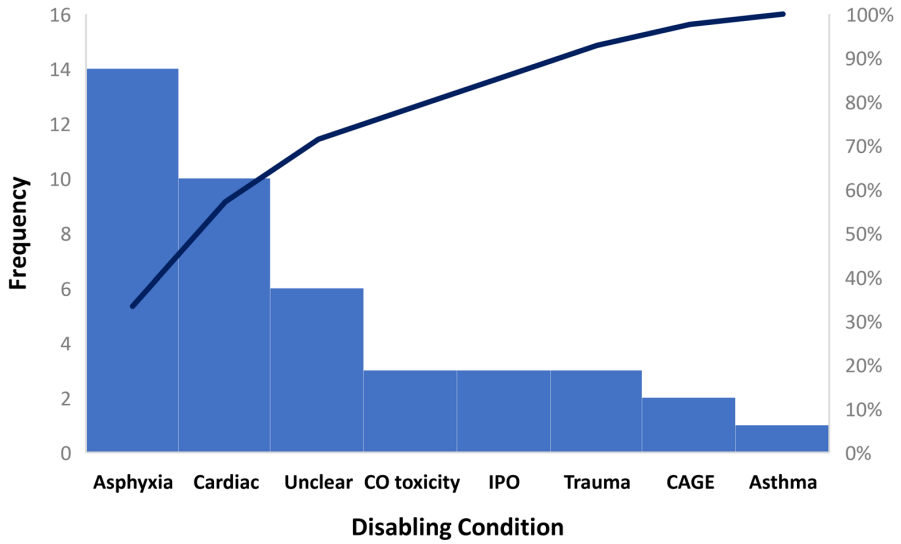


Figure 2
Disabling conditions in 42 scuba diving fatalities; CO – carbon monoxide; CAGE – cerebral arterial gas embolism; IPO – immersion pulmonary oedema



operational procedures for an air compressor, while the other involved poor dive site/conditions/ratios and selection procedures of a dive operator.

Triggers

Forty-seven likely or possible triggers were identified in 32 of the incidents. There was insufficient information to try to identify possible triggers in ten cases. The main triggers (20) were environment-related and included adverse sea conditions such as swell, chop, current and poor visibility. Eleven of the environmental triggers were believed to have been associated with the direct effects of immersion, which can impact cardiac function and lead to dysrhythmias in susceptible persons, especially when combined with other stressors such as exertion and anxiety. Exertion was thought to have been a likely trigger in at least eight cases, six of these also likely exacerbated by immersion. One case involved substantial pre-dive exertion. Five of the cases linked to exertion were in obese individuals.

Gas supply triggers were identified as likely or possible for nine incidents. These included four in which the diver had exhausted their breathing gas and two where the divers had

Table 1
Comparison of some fatality victim characteristics between the 2001–13 and 2014–18 periods

Characteristic	2001–13	2014–18	*P*
Mean age (years)	44	50	0.02
Male victims	99/126 (79%)	30/42 (71%)	NS
Overweight or obese	83/108 (77%)	28/35 (80%)	NS
Obese	40/108 (37%)	19/35 (54%)	NS
Experienced or very experienced	58/110 (53%)	28/38 (74%)	0.024
Solo	13/125 (10%)	11/42 (27%)	< 0.001
Commercial setting	58/126 (46%)	15/42 (36%)	NS
Tourist victims	35/126 (28%)	11/42 (26%)	NS
Cardiac disabling condition	32/126 (25%)	10/42 (24%)	NS

entered the water with the cylinder valve closed (one of these being diver error and the other intentional). Another two incidents were triggered by contaminated breathing gas. The final incident resulted from the loss of air supply after the displacement of a full-face mask. Two of the three trauma-related deaths resulted from adverse contact with sharks and the other with a boat.

Disabling agents

The main disabling agents appeared to have been medical factors, predominantly cardiac-related (Figure 1). However, IPO was the likely disabling agent in two to three cases, and asthma, aortic dissection, and seizure in one each.

Disabling conditions

The predominant disabling conditions identified were asphyxia (primary drowning), cardiac causes, carbon monoxide toxicity and immersion pulmonary oedema. In six cases, no clear disabling condition could be identified with reasonable confidence, and in two cases due to all or most of the victim's body not being recovered (Figure 2). Inexperience, poor planning, and lack of fitness/obesity were identified as predisposing factors in at least 13 of the 16 asphyxia incidents. Pre-existing medical conditions were associated with all 10 of the identified cardiac deaths with at least seven of the victims under some medical oversight. In at least two cases, the divers were not under medical care.

A comparison of some key characteristics of the fatality victims in the periods 2001–13 and 2014–18 was compiled and is shown in Table 1.

Discussion

The victims of these compressed gas diving incidents were predominantly older males who were experienced divers and many of whom were obese. There was a high prevalence of pre-existing medical conditions, mainly cardiac. Almost two-thirds of the victims were alone at the time of their incident, and most were found still wearing their weights and with uninflated BCDs. Supervision shortcomings were associated with almost one-quarter of the deaths. Two of the SSBA divers and one scuba diver died subsequent to CO poisoning.

DEMOGRAPHICS AND MEDICAL HISTORY

The victims in this series were on average almost six years older than those during 2001 to 2013, and, although there was a lower proportion of males, the difference was not statistically significant. Of interest, more than three-quarters of the divers in both series were overweight or obese with 54% of the 35 victims for whom data were available in this series being obese. Current Australian data indicate that 67% of Australian adults are reportedly overweight or obese, with the proportion who are obese being 31%. For the ages 45–54 years, which more closely reflects the scuba victims, the proportion who are obese is 37.4%.[9] This is substantially lower than the diving victims in both this and the earlier series, highlighting concerns about obesity and fitness to dive raised previously.[5,10] The prevalence of obesity increases with age which is consistent with the higher ages of victims in this series.

The Undersea and Hyperbaric Medical Society (UHMS) recommends that "*Asymptomatic candidates over 45 years of age with risk factors for coronary artery disease should undergo evaluation by a physician*."[11] However, more cautious advice from the South Pacific Underwater Medicine Society (SPUMS) is "*from the age of 45 years, all candidates should have regular assessments at no longer than five yearly intervals, with emphasis on evaluation of cardiovascular fitness and pulmonary reserves*."[12] The latter is reflected in the Australian Water Safety Strategy.[13]

In support of the recommendations for close diving medical oversight at 45 years or more, combined Australian data for 2001 to 2018 reveals that 92 (56%) of the 164 scuba victims were 45 years or older, with 84% of this subgroup likely to have suffered a cardiac-related disabling condition. Over the

extended period, at least 25% (possibly up to 30%) of all the scuba victims likely suffered a cardiac-related disabling condition.

Unfortunately, there is currently no mechanism for reviewing the fitness of certified divers. Active education by dive agencies and clubs to encourage those over 45 to seek regular review should be encouraged. Our data would suggest that any new certification on a diver over 45 years of age should have a comprehensive fitness-to-dive (FTD) assessment rather than just a questionnaire. This especially applies to those divers taking up technical diving courses that require significant physical exertion.

Seven of the victims were reported to have had medical assessments within the six months prior to their deaths, although there might have been others who were not reported. It appears that medical conditions directly contributed to the deaths of at least four of these individuals. Despite assessment guidelines, it is inevitable that some individuals with significant contraindications will be assessed as fit to dive even if a medical is conducted.[14] This can result from the candidate withholding important information (e.g., SC10 in this series – Appendix 1), a lack of diving medical knowledge by a doctor untrained in diving medicine, and, sometimes, as a result of an unsuitable candidate 'slipping through the cracks' even with a doctor with relevant training (e.g., SC25 in this series – Appendix 1). Fitness to dive assessments have some inherent limitations and the standard tests included in them will not always reveal underlying problems. Examining doctors may face the difficulty of selecting which candidates to investigate further and the most appropriate tests to conduct. A 2019 SPUMS workshop on FTD resulted in the creation of a flowchart for the screening of divers aged 45 years or more which is incorporated as an appendix in the 2020 SPUMS medical assessment form.[12,15] This tool, aimed at identifying those with a higher risk of a cardiac event, may variously include a resting 12 lead ECG, use of a cardiovascular risk calculation tool, cardiological referral, coronary artery calcium score, and exercise stress test as determined by the cardiologist in consultation with diving doctor if the cardiologist is unfamiliar with dive medicine. It is hoped that such guidelines if widely and conscientiously implemented, will reduce the number of deaths in older, often obese divers. However, regardless of age, if there is any doubt concerning a candidate's exercise capacity, these authors suggest that some form of exercise assessment should be considered along with the use of the SPUMS cardiovascular risk flowchart.

EXPERIENCE

Victims in this series were generally more experienced than those in the earlier group, likely associated with their higher age. Almost all those with cardiac-related disabling conditions were classified as experienced, again highlighting the need for older divers to monitor their diving fitness. However, interestingly, most of those classified as primary drownings were also experienced, highlighting the need for experienced divers not to become complacent about what may be challenging sea conditions or when using unfamiliar equipment. As in earlier reports, lack of recent experience was also a factor in several deaths, reinforcing the potential benefit of a cautious return to diving.

TOURISTS AND SETTING

There was a similar proportion of tourist victims over both periods and, although a lower proportion of deaths occurred in a commercial setting in this series, the difference was not significant. Over both periods, most of the tourist deaths occurred in Queensland which is unsurprising given the higher level of diving visitation to that State. As reported elsewhere, many of the scuba victims in Queensland are older divers, often with predisposing medical conditions.[16]

BUDDY AND RESCUE CIRCUMSTANCES

One of the fundamental edicts of diving is to dive with a buddy, the obvious benefit being the potential for more timely assistance and rescue in the event of a problem. Solo diving has been a significant and constant feature and of concern in many fatality reports from around the world.[5,10,17–19] The relatively high number of solo divers in this series, many of whom were highly experienced, indicates a continued level of complacency. This is especially risky in the aging diver who is at a higher risk of suffering an unexpected medical episode. Additionally, the high proportion of divers who were separated from their buddies at the time of the incident once again reinforces the importance of, and the need for, improved buddy monitoring.

The low proportion of victims who had ditched weights or inflated their BCDs again suggests the lack of automaticity and the need for greater emphasis and continued reinforcement of this important self-rescue procedure. Reaching the surface generally reduces delays in rescue and the implementation of BLS. Ready access to an AED remains uncommon in the diving scenario, other than in a commercial setting in Queensland where they are mandated. With the greater prevalence of cardiac-related problems in divers, there is a need to increase the availability of AEDs at dive sites, as well as refine supervision, rescue and first aid efficiency to minimise delays to defibrillation and enhance the likelihood of successful resuscitation.[20]

SUPERVISION

Shortcomings in supervision, whether with trainees, novices or experienced divers contributed to a number of deaths, with

Footnote: * Appendix 1 is available on DHM Journal's website: https://www.dhmjournal.com/index.php/journals?id=313

supervision failures occurring on the surface or underwater. For example, a diver on an introductory scuba experience drowned after becoming separated from the instructor in poor visibility. A student and their instructor both drowned during an open water training dive after swimming into very rough conditions. Such incidents can be mitigated through increased training and awareness of potential problems, improved planning, higher supervision-to-participant ratios, and closer monitoring.

CARBON MONOXIDE (CO) POISONING

Although relatively common in SSBA divers,[21] CO poisoning is rarely identified in scuba divers, albeit likely underreported.[22,23] Two of the SSBA divers in this series succumbed to CO poisoning, apparently from compressor exhaust contaminating their air. One was the result of poor compressor positioning and stability, and the other from poor design with the air intake too close to the exhaust outlet, as well as poor positioning. In both cases, better supervision might have prevented the death.

The CO-related death of the scuba diver appears to be the only documented case in Australia that has been positively confirmed by both breathing gas and postmortem toxicological analysis. It resulted primarily from a combination of poor compressor placement (allowing overheating), poor maintenance, and inadequate awareness and oversight. Scuba compressors need to be installed and maintained by professionals with relevant expertise, with regular and appropriate oil and filter changes, and the air quality monitored regularly. The use of fitted and portable CO detectors would reduce the likelihood of contamination.

IMMERSION PULMONARY OEDEMA

There were three divers in this series who were likely victims of IPO. This assessment was based on careful consideration of the victims' diving and medical histories, witness accounts, and autopsies. A history of dyspnoea with immersion can indicate a susceptible individual, as can a medical history of hypertension or chronic cardiac pathology (e.g., mitral or aortic valve disease, IHD, myocardial fibrosis, ventricular hypertrophy).[24] Witness reports of dyspnoea, coughing (especially with expectoration of blood-stained sputum), and cyanosis can be indicative. Although these are commonly associated with drowning, in cases where there was little likelihood that the victim inhaled water, IPO may be favoured over drowning, although cardiac dysfunction also needs to be considered. The increased respiratory effort associated with faulty regulators, certain rebreathers and, possibly, some snorkels has been implicated and is a consideration.

Although prompt investigations such as blood gases, chest X-ray or CT, blood tests and echocardiography can assist with the diagnosis of IPO in survivors and may help explain the aetiology, this is far more difficult postmortem. Autopsy findings of pulmonary oedema in the airways can readily be attributed to drowning or cardiac disease. The presence of sand or other sediment in the airways, lungs or other gas spaces may sometimes help distinguish drowning from IPO. Elevated postmortem vitreous sodium chloride levels have been suggested as a diagnostic test for saltwater drowning but have limitations and should be used with caution.[25]

Based on the above criteria, Australian scuba fatality data suggest that 3.6% (6/164) of scuba fatalities from 2001–18 resulted from IPO.[14] Similarly, New Zealand data, based on the same criteria, suggest a rate of 2.1% (1/48) for 2007–16.[10] Divers Alert Network (DAN) data indicate that 2.2% of 8,348 diving-related emergency and enquiry calls to its hotline from 2014 to 2018 related to IPO.[26] However, given the diagnostic difficulties, it is believed that IPO may be underreported with many cases attributed to drowning or cardiac dysrhythmia.

SHARK ATTACKS

Both victims of fatal shark attacks in this series were diving near where others were fishing, and one was carrying a bag of scallops. Seafood collection, as well as diving near fishing activities, have been identified as major risk factors for shark attacks on divers and snorkellers.[27] Although rarely documented by investigating personnel, the use of burley to attract fish has become more prevalent and would likely increase the risk to nearby divers.

LIMITATIONS

Even using multiple sources, it is possible that some fatalities were not recorded due to limitations in recording and NCIS searches. As with any uncontrolled case series, the collection and analysis of the fatality data are subject to inevitable limitations and uncertainties associated with the investigations. Witness reports varied in their likely reliability. Police reports varied in their content, often related to the expertise of the investigators. Given that many incidents were unwitnessed, some of the assertions in the reports are speculative. Many data items were not available which rendered the study data incomplete, thus limiting the conclusions that can be drawn. The CEA attempts to identify the predominant features of each case, but there always remains an element of uncertainty.

Conclusions

Advancing age, obesity and the associated cardiac disease have become increasingly prevalent in diving fatalities, and the need for appropriate assessment of fitness to dive is evident. The high proportion of victims who were alone at the time of their incident indicates that the message of close buddy monitoring is still not getting through sufficiently. Similarly, the number of victims found still wearing their

weights and with uninflated BCDs reveals the persistence of this problem and the ongoing need to better inform divers about the importance of positive buoyancy in an emergency.

Breathing gas suppliers, whether professional, club-based, or private need to ensure that the gas supplied is free from contaminants. Users of SSBA need appropriate training to ensure they are aware of the potential hazards and the necessary measures to minimise these. Deaths from CO poisoning are usually preventable by appropriate education, equipment maintenance and/or supervision. Divers should avoid diving where there is fishing activity.

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Effectiveness of hyperbaric chamber ventilation

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Abstract
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Introduction: Hyperbaric chamber ventilation (HCV) refers to the intentional introduction of fresh gas, whether air, oxygen, or heliox, into a pressurised hyperbaric chamber in order to remove stale or otherwise compromised gas. The minimum required continuous HCV rate is usually determined by mathematical models derived from the contaminant mass balance within a well-stirred compartment. Non-uniform contaminant distribution patterns inside a hyperbaric chamber could emerge and invalidate the predictions of well-stirred models.
Methods: Contaminant distribution was investigated inside a clinical hyperbaric chamber with the aim of comparing well-stirred model predictions with the actual contaminant concentration measurements.
Results: Local ventilation effectiveness inside a clinical hyperbaric chamber may be compromised, leading to higher contaminant concentration values compared to the predictions of a mathematical model with a well-stirred assumption.
Conclusions: A well-stirred assumption in mathematical models is a useful simplification that allows reasonably accurate estimates of HCV requirements. However, local ventilation effectiveness values in a particular hyperbaric chamber might vary, with the potential for hazardous contaminant accumulation in under-ventilated zones.

Introduction

From the moment a clinical hyperbaric chamber is pressurised, the occupants (patients and medical attendants) are physically trapped in a sealed confined space which requires accurate atmosphere control measures to sustain physiological parameters compatible with survival. Hyperbaric chamber ventilation (HCV) refers to the intentional introduction of fresh gas, whether air, oxygen, or heliox, into a pressurised hyperbaric chamber in order to remove stale or otherwise compromised gas. This is different from atmosphere control in closed-loop systems used in some chambers and in saturation for deep-diving operations where refreshing of the internal atmosphere is mainly achieved by targeted removal of waste gases (including carbon dioxide and other products), and metabolised oxygen replacement. Since most clinical hyperbaric chambers use just breathing air as the pressurisation gas, the scope of this study was limited to ventilation of hyperbaric chambers using air.

The primary aim of HCV is maintenance of safe oxygen levels inside the chamber, and removal of hazardous contaminants, mainly carbon dioxide. Excess oxygen inside a hyperbaric chamber increases the fire risk and is thus considered a contaminant. Additionally, HCV can be applied to remove odours and control physical parameters of chamber atmosphere such as temperature and humidity.

HCV is usually applied during the constant pressure phase of a hyperbaric treatment session, but it can also be utilised during compression or decompression. It can also be applied continuously or intermittently. There is a broad diversity of recommendations when it comes to HCV requirements.[1]

The current European Standard EN 14931: 2006 primarily addresses HCV requirements in terms of controlling the chamber levels of oxygen, carbon dioxide, impurities in the form of organic compounds, as well as humidity.[2] The specified HCV rate of 30 actual litres per minute (ALPM) per person is only stated for a specified duration and as a means of quantifying the minimum required air supply capacity.

In the United States, the NFPA 99: 2021 code requires a minimum HCV rate of 3 cubic feet (84.9 L) per minute (CFM) per occupant not breathing on a built-in breathing system (BIBS), but without specifying pressure and temperature conditions.[3] This requirement can be traced back to the 1968 edition (referred to as NFPA 56D-T at that stage), but it was replaced by 3 actual cubic feet per minute (ACFM) (84.9 ALPM) in NFPA 99: 1993 (NFPA 99) and remained so through to the 2005 edition. There were no

new editions until 2015, when the 3 CFM requirement was reintroduced. There appeared to be no rationale for this change. The NFPA 99 hyperbaric chapter committee has acknowledged that this flow rate needs further investigation as it does not account for the effect of pressure (personal communication with Francois Burman on 26 November 2022). The determination of an acceptable HCV is based on mitigation of not only excess concentration of contaminants in the chamber atmosphere, but their partial pressure as well.

A more nuanced approach to HCV rate requirement is found in USN Diving Manual Rev 7 of 2016, namely 2 ACFM (56.6 ALPM) per occupant at rest and 4 ACFM (113 ALPM) per active occupant.[4] The rationale provided is based on ensuring a carbon dioxide partial pressure of less than 1.5 % surface equivalent value (SEV), and oxygen concentration of less than 25%. The manual emphasises that chamber inlet and exhaust terminal separation is required for optimal chamber ventilation, implying that recommended HCV rates might be inadequate if there is 'short-circuiting' of ventilation gas between terminals.

The minimum required continuous HCV rate has been determined by mathematical models with the aim of controlling the levels of metabolically produced carbon dioxide. Such models are typically derived from the contaminant mass balance within a well-stirred single compartment.[5,6] However, significantly non-uniform contaminant distribution patterns inside a hyperbaric chamber could emerge and invalidate the predictions under this well-stirred assumption. This could pose a hazard, especially in terms of uncontrolled oxygen accumulation (pooling) and the associated risk of fire inside a hyperbaric chamber.

We devised a simple method to investigate the contaminant distribution (specifically oxygen concentration) inside a clinical hyperbaric chamber with the aim of comparing well-stirred model predictions with the actual contaminant concentration measurements. We expressed our findings in terms of relative contaminant removal effectiveness. Mathematical derivation of a well-stirred model of HCV is included. Our findings suggest that local ventilation effectiveness inside a clinical hyperbaric chamber may be compromised leading to higher contaminant concentration values compared to the predictions of a mathematical model with a well-stirred assumption.

Methods

BASIC DERIVATIONS

A glossary containing definitions for mathematical symbols and abbreviations used generally and in the following equations appears at the end of this article (page 91).

A continuity equation can be formulated based on contaminant molar balance during HCV.[5]

$$\dot{n}_c = \dot{n}_{c,gen} - \dot{n}_{c,cons} + \dot{n}_{c,in} - \dot{n}_{c,out} \quad (1)$$

Under standard temperature and pressure conditions (STP), and assuming ideal gas behaviour, the molar amount of contaminant equals the contaminant volume divided by the standard molar volume (the volume occupied by one mole of gas under STP conditions).

$$n = \frac{V_{c,STP}}{V_{mol,STP}} \quad (2)$$

allowing the continuity Eq (1) to be reformulated in terms of contaminant volumes.

$$\dot{V}_{c,STP} = \dot{V}_{c,gen,STP} - \dot{V}_{c,cons,STP} + \dot{V}_{c,in,STP} - \dot{V}_{c,out,STP} \quad (3)$$

Replacing $\dot{V}_{c,in,STP}$ and $\dot{V}_{c,out,STP}$ with their equivalent expressions in terms of contaminant concentrations (z) and ventilation flows yields.

$$\dot{V}_{c,STP} = \dot{V}_{c,gen,STP} - \dot{V}_{c,cons,STP} + z_{in}\dot{V}_{vent,in,STP} - z_{out}\dot{V}_{vent,out,STP} \quad (4)$$

Given that the total volume of the gas in the chamber is constant during constant-pressure HCV, all the volume changes cancel out, i.e.,

$$\dot{V}_{c,gen,STP} - \dot{V}_{c,cons,STP} + \dot{V}_{vent,in,STP} - \dot{V}_{vent,out,STP} = 0 \quad (5)$$

The left-hand side of Eq (4) can thus be expressed in terms of the contaminant concentration change. Also, under perfect mixing assumption, the contaminant concentrations in the chamber and in the exhausted gas are equal. Eq (4) can then be reformulated in the following way:

$$V_{T,STP}\frac{dz}{dt} = G + z_{in}\dot{V}_{vent,in,STP} - z(\dot{V}_{vent,in,STP} + G) \quad (6)$$

where $V_{T,STP}$ is the total gas volume in the chamber, defined as:

$$V_{T,STP} = V_{ch}\frac{P_{ch}T_s}{P_sT_{ch}}$$

and G is the net contaminant volume gain, i.e.,

$$G = \dot{V}_{c,gen,STP} - \dot{V}_{c,cons,STP}$$

with

$$\dot{V}_{vent,in,STP} + \mathrm{G} = \dot{V}_{vent,out,STP}$$

as required by the volume balance of Eq (5).

Solving Eq (6), we obtain the formula for the contaminant concentration in the chamber as a function of time.

$$z(t) = \frac{G + z_{in}\dot{V}_{vent,in,STP}}{\dot{V}_{vent,in,STP} + G} + \left(\mathrm{z}(0) - \left(\frac{G + z_{in}\dot{V}_{vent,in,STP}}{\dot{V}_{vent,in,STP} + G}\right)\right)e^{-\alpha t} \tag{7}$$

with

$$\alpha = \frac{\dot{V}_{vent,in,STP} + G}{V_{T,STP}}$$

In the limit when t $\rightarrow\infty$, we observe the following steady state solution:

$$z_{ss} = \lim_{t\to\infty}\left(\frac{G + z_{in}\dot{V}_{vent,in,STP}}{\dot{V}_{vent,in,STP} + G} + \left(\mathrm{z}(0) - \left(\frac{G + z_{in}\dot{V}_{vent,in,STP}}{\dot{V}_{vent,in,STP} + G}\right)\right)e^{-\alpha t}\right) = \frac{G + z_{in}\dot{V}_{vent,in,STP}}{\dot{V}_{vent,in,STP} + G} \tag{8}$$

The minimum required HCV inflow rate to maintain a set maximum contaminant concentration indefinitely is thus

$$\dot{V}_{vent,in,min,STP} = \frac{G(1 - z_{max})}{z_{max} - z_{in}} \tag{9a}$$

with corresponding HCV outflow

$$\dot{V}_{vent,out,min,STP} = \frac{G(1 - z_{max})}{z_{max} - z_{in}} + G \tag{9b}$$

The transient state Eq (7) and the steady state Eq (8) can be modified based on Dalton's Law that underscores the relationship between the contaminant concentration and the contaminant partial pressure, namely

$$P_c = zP_{ch} \tag{10}$$

The transient state Eq (7) can then be modified to yield the formula for the partial pressure of the contaminant as a function of time

$$P_c(t) = \left(\frac{G + z_{in}\dot{V}_{vent,in,STP}}{\dot{V}_{vent,in,STP} + G} + \left(\mathrm{z}(0) - \left(\frac{G + z_{in}\dot{V}_{vent,in,STP}}{\dot{V}_{vent,in,STP} + G}\right)\right)e^{-\alpha t}\right)P_{ch} \tag{11a}$$

Distributing P_{ch} yields

$$P_c(t) = \frac{G + z_{in}\dot{V}_{vent,in,STP}}{\dot{V}_{vent,in,STP} + G}P_{ch} + \left(P_c(0) - \left(\frac{G + z_{in}\dot{V}_{vent,in,STP}}{\dot{V}_{vent,in,STP} + G}\right)P_{ch}\right)e^{-\alpha t} \tag{11b}$$

with α remaining the same as in Eq (7).

The steady state Eq (8) becomes modified to govern the minimum HCV rate required to control the partial pressure of the contaminant

$$\dot{V}_{vent,in,min,STP} = \frac{G(P_{ch} - P_{c,\max})}{P_{c,max} - P_{c,in}} \tag{12}$$

In the absence of contaminant generation or consumption inside the chamber

$$\dot{V}_{vent,in,STP} = \dot{V}_{vent,out,STP} = \dot{V}_{vent,STP} \tag{13}$$

and Eq (6) reduces to

$$V_{T,STP}\frac{dz}{dt} = \dot{V}_{vent,STP}(z_{in} - \mathrm{z}) \tag{14}$$

Solving Eq (14) we obtain the formula for the contaminant concentration as a function of time in the absence of contaminant generation or consumption:

$$z(t) = z_{in} + (z(0) - z_{in})e^{-kt} \tag{15}$$

where

$$k = \frac{\dot{V}_{vent,STP}}{V_{T,STP}}$$

Solving Eq (15) for $\dot{V}_{vent,STP} \cdot t_{vent}$, the formula for the required volume of ventilation gas $V_{vent,STP}$ is obtained as

$$\dot{V}_{vent,STP} \cdot t_{vent} = V_{vent,STP} = -ln\left(\frac{z_f - z_{in}}{z_0 - z_{in}}\right)V_{T,STP} \tag{16}$$

with $z_0 \equiv z(0)$ and z_f being the final contaminant concentration after $t = t_{vent}$ has elapsed.

EXPERIMENT

Experimental measurements were obtained between 18 July and 19 August 2022, inside a hyperbaric chamber (HAUX-STARCOM 1500/6 MP, Haux Life Support GmbH, Karlsbad, Germany) installed at the Hyperbaric and Tissue Viability Unit located at Gozo General Hospital in Malta.

Oxygen was used as a tracer gas, and oxygen concentrations were measured at 15 sampling points distributed inside the hyperbaric chamber (Figure 1). For each sampling point, three separate measurements were obtained at three different chamber pressures with 135 measurements carried out in total. Measurements were obtained under steady state oxygen concentration created by continuous HCV and oxygen injection. Oxygen levels inside the chamber were measured using a portable HAUX–OXYSEARCH analyser.

The analyser was spanned using normal atmospheric air on the surface. Oxygen partial pressure values were recorded in mbar and converted to volume concentrations via Dalton's Law.

Oxygen injection was carried out by the HAUX-FLOW-CONTROL-UNIT at a set actual volume rate. The source was located between sampling points M1 and M4 (Figure 1). The chamber inlet was located in the proximity of point B2, while the chamber outlet was nearest to M3. Due to the fire hazard, an injection rate of 5 ALPM was chosen in conjunction with the set HCV rate of 1100 ALPM to keep oxygen concentration levels in the chamber to below 23.5%[3] at all measurement locations upon reaching the oxygen contamination steady state with a set HCV.

To investigate the effect of depth on HCV effectiveness, measurements were obtained at three different hyperbaric chamber (absolute) pressures: 160 kPa, 220 kPa, and 280 kPa. The oxygen concentration values obtained were used to

Figure 1
Arrangement of sampling points across the hyperbaric chamber interior

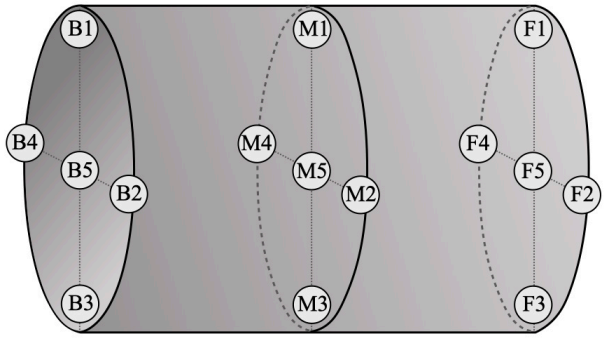
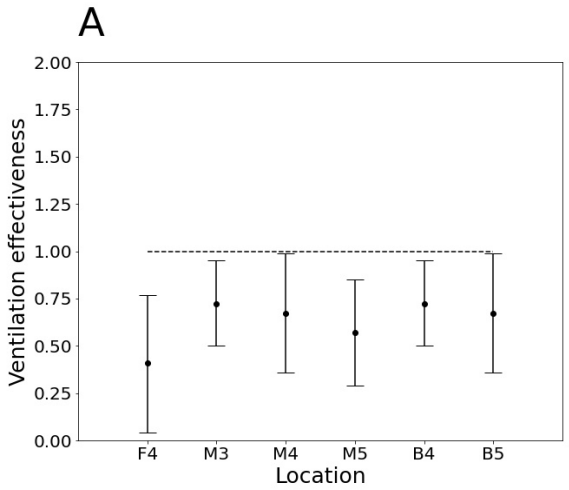
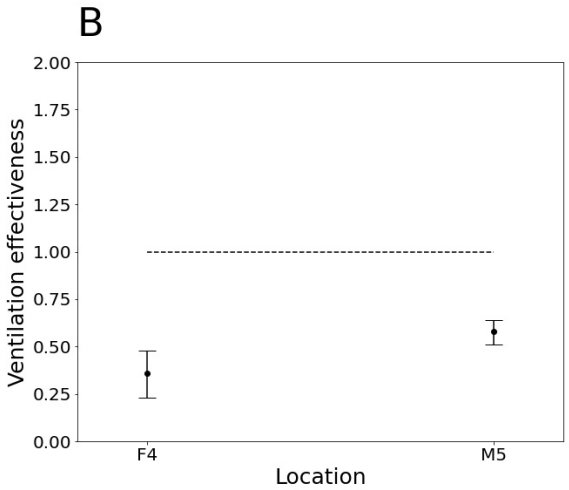


Figure 2
Ventilation effectiveness values (mean ± 95% confidence interval) deviating from well-stirred model predictions at three different hyperbaric chamber pressures, A – 160 kPa, B – 220 kPa and C – 280 kPa





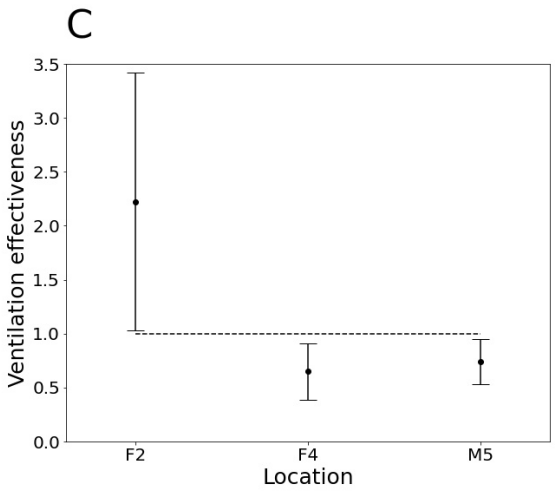


Table 1

Measured mean steady state oxygen concentrations at all sampling points with corresponding ventilation effectiveness values. Statistically significant deviations from well-stirred model marked in bold, also shown in Figure 2; CI – confidence interval

Point	160 kPa				220 kPa				280 kPa			
	$\hat{z}_{ss}$	$\hat{\varepsilon}$	95% CI Lo	95% CI Hi	$\hat{z}_{ss}$	$\hat{\varepsilon}$	95% CI Lo	95% CI Hi	$\hat{z}_{ss}$	$\hat{\varepsilon}$	95% CI Lo	95% CI Hi
F1	0.2115	3.18	-2.29	8.65	0.2133	1.43	-1.14	4.00	0.2113	2.89	0.98	4.81
F2	0.2125	1.49	0.54	2.45	0.2130	1.49	-0.94	3.92	0.2117	**2.22**	1.03	3.42
F3	0.2131	1.18	0.58	1.77	0.2133	1.25	-0.32	2.82	0.2119	1.98	0.65	3.31
F4	0.2196	**0.41**	0.04	0.77	0.2202	**0.36**	0.23	0.48	0.2156	**0.65**	0.39	0.91
F5	0.2146	0.83	0.15	1.51	0.2153	0.72	0.14	1.29	0.2130	1.22	0.76	1.67
M1	0.2146	0.84	0.13	1.56	0.2144	0.89	-0.01	1.79	0.2129	1.39	0.00	2.77
M2	0.2150	0.75	0.27	1.23	0.2150	0.75	0.24	1.26	0.2137	0.97	0.84	1.10
M3	0.2150	**0.72**	0.50	0.95	0.2148	0.77	0.28	1.25	0.2137	0.98	0.69	1.27
M4	0.2154	**0.67**	0.36	0.99	0.2153	0.70	0.30	1.09	0.2140	0.91	0.45	1.36
M5	0.2165	**0.57**	0.29	0.85	0.2162	**0.58**	0.51	0.64	0.2149	**0.74**	0.53	0.95
B1	0.2144	0.83	0.53	1.13	0.2135	1.06	0.49	1.62	0.2126	1.46	0.27	2.64
B2	0.2131	1.18	0.58	1.77	0.2117	3.67	-5.35	12.69	0.2113	3.17	-0.83	7.17
B3	0.2150	0.75	0.27	1.23	0.2139	0.93	0.49	1.36	0.2130	1.26	0.37	2.15
B4	0.2150	**0.72**	0.50	0.95	0.2141	0.90	0.39	1.42	0.2131	1.18	0.63	1.73
B5	0.2154	**0.67**	0.36	0.99	0.2150	0.73	0.40	1.07	0.2140	0.91	0.45	1.36

calculate local HCV effectiveness relative to well-stirred model predictions, at each location, using the following expression:

$$\epsilon_{local} = \frac{z_{ss} - z_{in}}{z_{local} - z_{in}}$$

where z_{ss} represents the steady-state oxygen concentration predicted under well-stirred assumption and computed via Eq (8).

Statistical analysis was performed on results and findings expressed as mean ventilation effectiveness $\hat{\varepsilon}$ ± 95% confidence interval for each of the 15 location points at given pressures.

Results

Statistically significant instances of underventilation ($\hat{\varepsilon} < 1$) were found at all three test pressures (Table 1). At the chamber pressure of 160 kPa, 6 out of 15 points were determined to be underventilated, whereas at 220 kPa and 280 kPa, instances of underventilation were found at 2 out of 15 sampling points. Points F4 and M5 were underventilated at all three chamber pressures (Figure 2). There was a single instance of statistically significant overventilation ($\hat{\varepsilon} > 1$) at 280 kPa (point F2).

Discussion

The primary measure of HCV is its rate, expressed as standard volume of gas (determined at atmospheric pressure at sea level) exchanged per unit of time. Higher HCV rates facilitate contaminant removal from the chamber atmosphere. However, in reality the same HCV rate can have vastly different effects in the presence of different mixing behaviour of the inflowing air. Ventilation effectiveness is a concept used to address such differences in enclosed space ventilation performance.[7,8] Factors that could affect ventilation effectiveness of a hyperbaric chamber include chamber geometry, configuration of inlet and exhaust terminals, spatial obstacles, contaminant distribution, the degree of turbulence, and temperature distribution.

Our results indicate that the local ventilation performance of a clinical hyperbaric chamber could be significantly worse than what a well-stirred model would predict. Several sampling points inside the chamber were underventilated ($\hat{\varepsilon} < 1$) relative to the well-stirred scenario, and the difference found was statistically significant. This was not surprising in the light of our actual experience with HCV. Chamber operators and attendants have long known that stirring the chamber atmosphere manually during HCV tends to facilitate contaminant removal. One study reported that it can take 2.5 times longer to effectively ventilate the chamber if the chamber atmosphere is not manually stirred during ventilation.[9]

It is physically possible for a particular region inside a chamber to have a 'supra-ideal' ventilation performance ($\hat{\varepsilon} > 1$) resulting in more effective contaminant removal than what could be achieved by perfect mixing according to a well-stirred model. Indeed, measured HCV performance for the point F2 at 280 kPa exceeded the predictions of the well-stirred model, possibly due to its proximity to the chamber inlet terminal. The same effect occurs by design during local exhaust ventilation (LEV) whereby the contaminant is removed from the atmosphere before it can spread, and it is thus a preferred method of ventilation of hazardous contaminants in industry.[10] The LEV concept could be utilised in clinical hyperbaric practice. For example, by positioning a patient with poorly fitted and leaking oxygen mask closer to the chamber's exhaust terminal, removal of excess oxygen from the chamber atmosphere would most likely be facilitated during HCV.

One might expect that chamber pressure would affect HCV performance due to its effect on gas density, and we observed several instances of increased pressure exhibiting a ventilation effectiveness-enhancing effect, but the overall effect was not strong. However, our data set was obtained by performing measurements on only three separate occasions and on a relatively narrow pressure domain, implying that a study with more statistical power investigating a broader pressure domain might be able to better examine the phenomenon.

Conclusions

A well-stirred assumption in mathematical models is a useful simplification that allows reasonably accurate estimates of HCV requirements. However, local ventilation effectiveness values in a particular hyperbaric chamber might vary, with the potential for hazardous contaminant accumulation in underventilated zones. When the contaminant in question is oxygen, accumulation increases the risk of a catastrophic chamber fire. It is important to bear in mind that in a chamber environment, HCV is never a well-stirred process throughout the chamber and that considerable deviations in local contaminant concentration might exist relative to the values displayed by the chamber's gas analyser. Efforts should be made to minimise the presence of factors that tend to compromise ventilation effectiveness, such as excessive space partitions or proximity of ventilation terminals. Using more than one oxygen sampling point as well as utilising local exhaust ventilation near the oxygen sources could provide additional safety barriers to hazardous oxygen accumulation. Hyperbaric chamber ventilation effectiveness should be addressed during the design phase prior to chamber manufacture. Thereafter, it should be assessed prior to final certification of the chamber.

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Glossary

Abbreviation or term	Meaning
ACFM	actual cubic feet per minute; flow measure unit at ambient conditions
ALPM	actual litres per minute; flow measure unit at ambient conditions
BIBS	built-in breathing system
CFM	(standard) cubic feet per minute
$\hat{\varepsilon}$	ventilation effectiveness
G	net contaminant volume gain
HBOT	hyperbaric oxygen therapy
HCV	hyperbaric chamber ventilation
LEV	local exhaust ventilation
$\dot{n}_{c}$	the rate at which contaminant moles are accumulating in the chamber
$\dot{n}_{c,cons}$	the rate at which the contaminant moles are consumed in the chamber
$\dot{n}_{c,gen}$	the rate at which contaminant moles are generated in the chamber
$\dot{n}_{c,in}$	the rate at which contaminant moles are entering the chamber via HCV
$\dot{n}_{c,out}$	the rate at which contaminant moles are exiting the chamber via HCV
P_{c}	contaminant partial pressure in the chamber atmosphere
P_{ch}	chamber pressure (absolute)
$P_{c,in}$	contaminant partial pressure in the ventilation gas
$P_{c,max}$	contaminant partial pressure threshold limit value in the chamber atmosphere
P_{s}	standard pressure; 100 kPa (1 bar)
R	chamber pressurisation rate
SEV	surface equivalent value
STP	standard temperature and pressure; 273.15 K (0°C), 100 kPa (1 bar)
T_{ch}	chamber temperature (absolute)
T_{s}	standard temperature; 273.15 K (0°C)
V_{ch}	floodable chamber volume
$V_{T,STP}$	total gas volume in the chamber
$\dot{V}_{vent,in,STP}$	ventilation inflow
$\dot{V}_{vent,out,STP}$	ventilation outflow
z	contaminant volume concentration
z_{in}	contaminant volume concentration in the incoming ventilation gas in the chamber
z_{out}	contaminant volume concentration in the exhausted gas
z_{ss}	contaminant volume concentration at the steady state

Determining best practice for technical assessment of hookah surface supply diving equipment during diving fatality investigation

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Keywords
Diving deaths; Investigations; Surface supply breathing apparatus; SSBA

Abstract
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Introduction: This study aimed to develop a standard process and checklist for technical investigation of hookah diving equipment and apply it to Tasmanian hookah fatality investigations from the last 25 years.
Methods: A literature search was undertaken to identify technical reports and equipment investigations associated with diving accidents. The information was assimilated to create a process and checklist for specifically assessing the hookah apparatus. The checklist was then applied in a gap analysis of Tasmanian hookah diving fatality technical reports from 1995 to 2019.
Results: As no papers specifically describing technical evaluation of hookah equipment were identified, references evaluating scuba equipment were used to create a hookah technical assessment process incorporating unique features of the hookah. Features included: owner responsibility for air quality; maintenance, function; exhaust proximity to air intake; reservoir volume; output non-return valves; line pressure; sufficiency of supply; entanglement; hose severance risk; gas supply failure and hosing attachment to the diver. Seven hookah diving deaths occurred in Tasmania (1995–2019) of which three had documented technical assessment. Gap analysis identified inconsistent structure between reports with variability in the case descriptors. Missing technical data included: overview of the hookah systems; accessories; weights; how the apparatus was worn by the diver; compressor suitability; assessment of hookah function; breathing gas output and exhaust position relative to air intake.
Conclusions: The study demonstrated a need to standardise technical reporting of hookah equipment after diving accidents. The checklist generated may serve as a resource for future hookah assessments and inform strategies for preventing future hookah accidents.

Introduction

From 2001–2013, Australia had 126 scuba fatalities.[1] Although these events are uncommon, they cause significant distress for diver's families, fellow divers, and rescuers. Diving fatalities may also receive media attention which may impact on industries such as diving tourism, and emergency service resource deployment. All fatal accidents require investigation and there could be legal consequences if, for example, there were diving procedural errors, failures in supervision, or issues with the diving equipment.

Diving fatality investigations are intended to determine the factors that contributed to the death so that preventative strategies can be employed to mitigate future adverse events. It is important to conduct such investigations with diligence, utilising methods and procedures that are standardised and consistent with best practice, so that key information is not overlooked. Although somewhat dated, national guidelines have been produced by the Royal College of Pathologists Australasia (RCPA) detailing the process for undertaking autopsy in diving fatalities.[2]

The RCPA autopsy guide briefly refers to specific risks of scuba, hookah and rebreather "*apparatus*", but not the technical evaluation of such equipment. There is limited information regarding the assessment process for a deceased diver's equipment as part of the broader investigation.[2]

The proceedings of a 2010 Divers Alert Network (DAN) workshop on recreational diving fatalities[3] include a paper titled "*Equipment testing*".[4] The authors included very detailed resources in appendices titled: "*Open circuit scuba equipment evaluation forms*", and "*Rebreather evaluation protocol*" which provide checklists for equipment investigation as part of scuba and rebreather deaths.[5]

Equipment inspection checklists were also provided in the proceedings.[6] The workshop focused on scuba and rebreather equipment and there was no specific reference to recreational surface supply breathing apparatus (SSBA). One paper in the DAN workshop proceedings reported that 62 of 351 Australian fatality victims (1 in every 6 deaths) between 1972 and 2005 had been using SSBA.[7] Despite this high prevalence, the diving deaths workshop did not produce any guidance for evaluating the surface supply apparatus and specifically did not mention the hookah apparatus.[3]

Hookah is a very specific type of SSBA. It comprises a petrol, electric or diesel motor as part of a compressor that drives the air supply at low pressure to the diver along a long, flexible, non-kink air hose. The diver receives their air supply to a second stage demand regulator and scuba style mouthpiece, or to a full-face mask and oronasal demand regulator. The whole unit has multiple component parts that are specific to hookah, and it provides compressed air delivery to divers underwater and/or in confined spaces. There are very specific risks related to hookah apparatus that are either not commonly associated with or applicable to scuba or rebreather equipment.[8] These can include the reliance on a motor to deliver the immediate breathing gas supply, the potential for on-scene contamination of the gas supply while diving, a limited air reserve capacity and a low fixed line pressure in the supply line. In addition, there can be limitations on air flow determined by the connections to the compressor, the length, configuration and construction of the air supply line and the depth of the diver. The long supply hose may become kinked, entangled in the marine environment, severed by a boat propeller, or disconnected. Continuity and/or sufficiency of breathing gas can be reduced with increasing depth and/or multiple divers breathing from the same unit. Finally, the air supply compressor is relatively remote from the diver, being in a boat, on land or as part of a flotation device.

Tasmania has a large population of hookah divers, although the exact number is unknown. The hookah apparatus is utilised by the wild seafood industry (e.g., abalone and scallop divers), commercial divers, scientific divers, and recreational divers. In one series, 43% of cases of decompression illness treated at the Tasmanian state hyperbaric facility used hookah apparatus.[9] A recent Tasmanian case series demonstrated that the hookah apparatus was used by 29% (5/17) of divers who died – all being recreational divers.[10] General Australian data reveals that 18% (15/84) of all SSBA deaths from 1965–2019 inclusive occurred in Tasmania, at least 37% of these were diving recreationally.[11]

Occupational divers must adhere to Workplace Health and Safety (WH&S) regulations, and, in the case of commercial abalone divers in Tasmania, are guided by a specific Code of Practice.[12] There is, however, no legislation in Tasmania governing recreational hookah diving which is largely unregulated. In addition, there are no unified national data for Australia covering the number of occupational divers, the apparatus used, hours in the water, or diving accidents. Occupational divers are required to adhere to WH&S Regulations and Australian/New Zealand Standards, but auditing of compliance is infrequent. There is no central reporting requirement for diver numbers or number of dives. This has prevented useful comparisons between recreational and occupational diving accident rates. One study reported 84 SSBA deaths in Australia from 1965 to 2019, (31 recreational divers, 43 occupational divers and 10 unknown).[11] In the period 2000–2019, 14/23 SSBA deaths were recreational divers, flagging a possible trend of increased risk in the recreational population.[13]

Recreational hookah usage is not subject to WH&S legislation and recreational hookah divers are not required to demonstrate any form of training to operate or purchase their equipment, which can be easily bought from websites, or second-hand. There are no mandated maintenance schedules for recreational divers, the responsibility for safety and hookah equipment maintenance resting with the operator. From the authors' observations, hookah divers in Tasmania generally dive alone or in buddy pairs (sometimes using two hoses from the compressor, or a single hose with a 'Y' connector to two second stage regulators. With recreational hookah divers, it appears that tenders in the boat are considered optional. Many have constructed their own home-made apparatus, including one individual who was observed to be using a 40-litre beer keg as the air reservoir. Diving tends to take place along remote shores away from population centres, and mostly is for the purpose of catching seafood.

Faulty equipment has been implicated as a significant contributor to hookah diving fatalities.[10,11] Very little guidance is available covering the technical evaluation of the hookah apparatus during diving fatality investigations, or how to assess hookah apparatus functionality before it is used. A standard procedure for technical evaluation of hookah equipment could add value by guiding future best practice in this area.

The aims of this study were to: develop a standard process for technical assessment of hookah diving equipment as part of a fatal hookah diving accident investigation, based on best available evidence; produce a checklist for recording the technical evaluation of the hookah apparatus that covers all essential information to assist investigators; and use the checklist to undertake a gap analysis of documented technical reports of hookah diving deaths at Royal Hobart Hospital over the past 25 years, identifying ways to improve on previous investigations.

Methods

This study was accepted and registered as a quality improvement project with the Tasmanian Health Service (South) Quality Committee safety reporting and learning system (SRLS) – Project #44-2020. Because all information

accessed in relation to previous accidents was available in the public domain as deidentified reports, ethics approval was not required.

A literature search was undertaken to identify guidance for investigating equipment in diving accidents of all types, so that relevant parts could be applied to the hookah apparatus. Details of the search are available from the authors.

The study methodology focused solely on literature applicable or specific to the technical evaluation of the hookah apparatus. Aspects of diving accident investigation that were not equipment related (e.g., environment, weather, thermal protection etc) were excluded.

The MEDLINE, EMBASE, PUBMED and EMCARE databases were searched for relevant documents published from January 1995 to December 2019. The literature search used the following terms (with synonyms and closely related words): *fatal, death, deceased, diving, diving deaths, equipment, surface supply / surface supplied, hookah apparatus, compressor, equipment failure, device failure, surface supply, investigation, audit, examination*.

The search was not limited by study design but was restricted to English language. Further studies were identified by examining the reference lists of articles and papers published in *Diving and Hyperbaric Medicine*, formerly *the South Pacific Underwater Medicine Society* (SPUMS) *Journal*, and *Undersea and Hyperbaric Medicine*. Also included were workshops proceedings from DAN, SPUMS, European Underwater and Baromedical Society (EUBS) and the Undersea and Hyperbaric Medical Society (UHMS). In addition, a hand search was conducted of texts in diving medicine specifically for chapters on diving deaths, and occupational codes of practice in Australia. Finally, a search was undertaken of references identified in papers located by the initial search providing access to other relevant articles. Manufacturer data sheets from commercial hookah producers were accessed (where available) to identify key functions that were relevant to technical assessment of the apparatus.

Information from the literature search was assimilated to produce a checklist for systematic assessment of hookah diving equipment. This included both the descriptive and functional assessment of the apparatus including hookah air output and quality. Key differences between scuba and hookah were identified and solutions applied to adapt the technical investigation to surface supply hookah apparatus.

Technical equipment reports provided to investigations of Tasmanian diving deaths 1995–2019 were accessed where a hookah apparatus had been used by the deceased diver. These assessments had been performed by technical staff in the Department of Diving and Hyperbaric Medicine at Royal Hobart Hospital and were available in a de-identified format. The technical reports were compared to the checklist as derived above to identify gaps in previous technical assessments for the purposes of quality improvement, and to identify possible gaps in the checklist.

Results

LITERATURE SEARCH

The search identified no papers that exclusively concentrated on hookah equipment investigation in the event of a diving death. Although some publications identified fatalities involving the use of hookah and surface supply equipment there was very little discussion, if any, on the investigation of actual equipment and this was the key reason for rejection of most papers.

We were unable to identify papers that specifically described hookah technical evaluation, nor a system of evaluating surface supply equipment in the event of a diving fatality. The processes for scuba fatality investigations as described in the DAN recreational diving fatalities workshop document were the only detailed description of equipment technical investigation identified by the literature search.[5,6] Although based on scuba, these evaluation forms provided very helpful practical guidance that was adapted to hookah diving equipment investigation. A specific checklist was developed by the authors to permit comprehensive and detailed analysis of hookah equipment.

The hand search also identified an instruction manual from one manufacturer of a hookah apparatus. This provided additional detail on the functional specifications of the equipment.[14] The Tasmanian Abalone Divers Code of Practice provided additional specifications which are requirements for hookah apparatus that were not covered by other references.[12] Both of the above were incorporated into the checklist as they covered areas that had not been identified by other references. A textbook from 2003 did not add additional information to the DAN dive fatality workshop.[3,15]

DEVELOPING A CHECKLIST FOR HOOKAH TECHNICAL ASSESSMENT

The checklist published by Barsky for scuba equipment in the DAN recreational diving fatalities workshop proceedings complimented the paper by Bozanic and Carver and provided a structure for visual inspection that could be applied to the hookah apparatus.[5,6]

The major headings for Bozanic's[5] article were:

(1) Epidemiological and site data
(2) Overview of the complete equipment system
(3) Dive cylinder including pressure
(4) Cylinder valve
(5) Buoyancy compensator
    Manufacturer data including specifications
    Condition report – all components

Configuration
Function testing including inflator function
(6) Weights – configuration, total amount, release capability
(7) Regulator
Manufacturer data and model number
Description and configuration
Service dates
First stage
Manufacturer data and model number
Serial number
Condition
Filters and covers
Second stage primary regulator
Manufacturer data and model number
Serial number
Condition
Filters and covers
Second stage secondary regulator
Manufacturer data and model number
Serial number
Condition
Filters and covers
Inflator hose
Condition
Connection to BCD and/or Dry suit
(8) Maintenance Records

Items 2–8 above were able to be adapted for hookah equipment technical assessment, further populated from published checklists in the workshop proceedings appendices. When creating a checklist for evaluating hookah equipment, scuba and hookah equipment were specifically compared. Parallels for the equipment when comparing scuba air versus hookah air are summarised in Table 1.

The key differences summarised in Table 1 were incorporated into the broad structure of the documents provided by Bozanic and Carver. The subheadings were expanded to permit detailed assessment of the equipment both structurally and when evaluating its function; populated from the key references, and the manufacturers' data sheet. The recommended checklist for technical assessment of the hookah apparatus following an accident appears in Appendix 1.

GAP ANALYSIS OF HOOKAH EQUIPMENT INVESTIGATIONS FOR TASMANIAN DIVING DEATHS

A recent paper reported five deaths in Tasmania between 1995 and 2015 where hookah equipment was utilised, and all these deaths involved equipment failure or inappropriate set-up.[10] An additional two divers died in Tasmania between 2015 and 2019 while using hookah apparatus, making seven cases in total in 25 years.[13]

Two of the earlier deaths were from the same incident and the report consisted only of a written assessment of air quality and a series of equipment photos so these could not be evaluated in detail. However, carbon monoxide (CO) poisoning was implicated, and it was apparent how this occurred, because the air intake was next to the exhaust. One death was due to a shark attack, and the equipment was not subject to a technical assessment. Another death (prior to 2001) had no technical report prepared. This left three deaths with technical assessment reports of the hookah equipment to evaluate for which gap analyses were undertaken using the checklist.

Table 2 summarises the key gaps identified in three available hookah technical reports. All three assessments did not report epidemiological or site descriptions and none reported weather or sea conditions. Details of these first two descriptors were available in the police reports. Exhaust intake position was not described in any report, although this could be deduced from pictures of the apparatus. No report included an assessment of hookah function. Of note was a progressive improvement in the level of detail of assessment between 2008 and 2019, particularly relating to the compressor, the air intake and regulator. The weight configuration/attachment description was available in only one of the three reports.

Discussion

The hookah apparatus is used by divers throughout Australia, recreationally and professionally, but has received very little attention in the medical literature.[11] Our literature search could identify only four papers of relevance to the study. Of 84 Australian surface supply breathing apparatus (SSBA) deaths in a 54-year period, 15 occurred in Tasmania.[11] There were seven Tasmanian hookah diving deaths from 1995–2019, all being recreational divers and which comprised 35% of all Tasmanian diving deaths.[10] In addition, from 2000–2019, 61% (14/23) of all Australian SSBA deaths were in recreational divers.[11] Tasmania's population makes up 2.1% of Australia's population, demonstrating recreational hookah deaths are over-represented in the island state.

Among other causes, previous studies have documented hookah equipment problems as important predisposing factors in causation of diving fatalities.[10,11] Given that the hookah equipment is supplying life-supporting air to the diver, the frequency of problems is of concern. It is also concerning that our literature review identified very little guidance on the technical evaluation of hookah equipment. This may be because other jurisdictions use hookah apparatus less frequently or have more effective safety systems. It is possible that some available dive fatality literature has under-reported hookah equipment problems because the assessments have not been undertaken using a systematic approach.

Footnote: Appendix 1 is available on the DHM website: https://www.dhmjournal.com/index.php/journals?id=314

Table 1
Comparison of technical features of scuba equipment compared to hookah; BCD – buoyancy control device

Equipment item/feature	Scuba	Hookah	Comments regarding differences
Origin of air	Air at or around dive shop or other high-pressure compressor	Air pumped from the local environment by the motor driven hookah pump. Often on anchored boat with potential to move with wind/current	Hookah air intake must be high and pointing into the wind, away from pump exhaust. Relative positions of intake and exhaust need to be monitored with boat movement/wind change
Air quality testing	At approved filling station; mandated by AS/NZS 2299.1	Responsibility of owner. Not mandated	Air quality of hookah may vary during the dive due to set up and environmental conditions
Air supply	Dive cylinder at up to 300 MPa air	Hookah pump at 800 kPa air	Scuba has first stage reducing valve to produce line pressure of 1,000–1,200 kPa. Not present in hookah so pressure may vary with compressor function
Air filtration	Post-compressor macro + charcoal filtration	Particle filter at intake, further filtration macro and charcoal at air delivery	Hookah owner/user responsible
Reserve air supply	Not routinely carried – reliance on buddy system and octopus regulators	Not routinely carried – reliance on small amounts of gas in hookah reserve cylinder	Hookah divers may carry a secondary scuba cylinder or have reserve air cylinder linked to hookah reserve. Australian Standard 2299.1(2015) mandates secondary air supply
Contents gauge	Measures pressure in the scuba cylinder	Not required – relies on continuous pump operation	Scuba diver can anticipate falling air supply as contents of cylinder are used
Air hose	Usually ≤ 1 m long	Could be 50–100 m long	Hookah hose risks kinking, being severed or disconnected
Secondary air hose	Standard via a second low pressure hose – for emergency use	May be added attached to Hookah pump directly or via 'T' or 'Y' connector to supply second diver (not emergency)	Reduced flow if additional hoses added to Hookah from a single source, or via constrictive connectors. No backup for second diver if pump fails
Line pressure	Typically 1,000 kPa to 1,200 kPa	Typically 800 kPa	
Diver regulator	Second stage tuned to the scuba line pressure	Second stage tuned to the hookah line pressure	Regulators can provide 200+ litres per minute, dependent on the line pressure and reserve volume
Regulator attachment	Secured to cylinder via first stage and BCD	Secured by looping under or inside harness or weight belt	If the hookah hose is not effectively secured, there can be considerable drag and possibility of mouthpiece loss
Attachment of gear	Via BCD	Weight vest, harness or belt	System must be configured to easily establish independent buoyancy and/or dump weights
Attachment of weights	Integrated within the BCD, or weight belt	Weight belt or weight vest	Weight vests not always designed for quick release
Adjustable buoyancy	Standard kit via BCD	BCD not routinely used. Buoyancy changes if weights ditched or lift bag used for catch	See above two points
Maintenance records	Cylinder test annually. Other maintenance not mandated	Not mandated – responsibility of owner	Requirement for cylinder test at testing station provides reminder to service other equipment

Table 2
Summary of gaps in three technical assessments of hookah diving fatalities

Descriptor	Investigation 1 gaps	Investigation 2 gaps	Investigation 3 gaps
Case identifiers. Date and location	Not reported but available in police report. Date included	Not reported but available in police report. Date included	Not reported but available in police report. Date included
Weather and sea conditions	Not reported	Not reported	Not reported
General overview of equipment system	Not described as a system except for report of fair to good working order	Not described	Described in detail
Compressor assessment including air output configuration	Not reported	Description limited to air filter, compressor motor, relief valve, absent non-return valves	Incomplete – limited detail. Described condition. Missed maintenance, pressure relief, air hose connections
Air intake, location, filter and air reserve	Not reported. Air intake reported as "*adequate vehicle type*" filter. No air reserve	Intake, location and air reserve not described. Filter described as "*inadequate water filter*". No redundancy	Described in detail. Intake, location and condition, air reserve volume described
Air supply hose and regulator	Described in detail. 'Y' connector. Regulator - aged but good condition but not assessed if hookah specific	Described in detail, including hookah specific kinks	Described in detail, and also described not hookah specific. Covered supply to two divers in detail
Exhaust and position relative to air intake	Not described	Described minimal spacing between exhaust and air inlet	Not described
Accessories – weight harness/belt. Buoyancy	Described. Weight belt attaching air supply to diver. Excess weight. Difficult to jettison	Not described	Not described
Analysis of air quality	Assessed for CO_2 and oil but no other assessment. Stated: "*depleted compressor reservoir volume*"	Full assessment performed	Full assessment performed
Assessment of hookah function	Not done	Not done	Not done
Additional comments	Described regulator hose cut during retrieval of deceased diver from entanglement	Hose reels for regulators not available	Length of hose 153 m

Table 1 demonstrates that it is possible to compare characteristics of scuba and hookah equipment. For both apparatuses, the endpoint is delivery of air to the diver. The scuba diver usually obtains their cylinder gas from a supplier and, in Australia, the supplier must comply with Australian standards and work health and safety legislation, providing a layer of safety. However, the hookah diver is essentially responsible for their own gas supply which is pumped from the ambient environment where they are diving by the hookah pump.

The hookah apparatus has multiple components that could affect air purity and supply to the diver. The air intake must be high and pointing into a non-contaminated source and must be filtered to prevent foreign material entering the system. The pump exhaust must be distant and downwind

from the air intake to prevent contamination with CO and other exhaust contaminants. The motor and compressor must be well maintained with sufficient fuel, because their continuous and correct operation is essential to keep the diver alive. The motor and compressor must be able to match the diver(s) requirements for air when exercising at the depth of operation. The system must have an adequate reservoir to minimise flow and pressure fluctuations, and all air hoses must be non-kink, of sufficient diameter, properly configured, free of holes, and connections must be always secure. Non-return 'check' valves are needed to prevent negative pressure injury if the diver's air supply is cut off. The hookah hose is long (up to 50 m), placing it at risk of entanglement or severance by propellers. The demand valve should be suitably attached to the diver to reduce displacement risk and needs to be tuned to the hookah output 'line pressure'.

Compared to the direct supply from a scuba cylinder via a first stage regulator and demand valve, the hookah supply process is complex. Therefore, it is not surprising that hookah accidents and fatalities occur when a component of the air supply system fails. Fatalities directly due to contamination of the breathing gas are rare in scuba divers,[16,17] although it is likely that some cases were missed due to lack of testing. Gas contamination has been positively identified as the primary contributor in only one of 444 (0.2%) Australian scuba deaths since 1965,[13] compared to 15/84 (18%) in SSBA diving deaths.[11]

Overall, there is a much higher prevalence of equipment problems in SSBA divers than scuba divers. For example, in a series of 126 Australia scuba deaths, 19% of predisposing factors and 12% of triggers were equipment-related, the breathing gas supply being affected in 15 (11%) of these.[18] This compares to equipment-related factors being identified as 48% of predisposing factors and 24% of triggers in Australian SSBA deaths, directly affecting the air supply in 43/84 (51%) of these fatal incidents.[11]

In the Australian SSBA deaths, the main source of problems were compressors which were often poorly maintained, poorly positioned, or inappropriately configured. Hence there is a definite need for a technical equipment evaluation process in the event of an accident. We adapted previously published processes from scuba accident investigation to produce a checklist to guide hookah equipment technical assessment. In the process of checklist production, we had to limit the amount of information collected, so that the process was confined to unique aspects of hookah diving.

The final checklist template (Appendix 1) was used to generate a gap analysis for three hookah fatality equipment reports from a single centre. All three reports identified causes of problems with the hookah diving systems and, also, highlighted flaws in the construction and maintenance. Within the study, a progressive improvement in detail of reporting occurred over time. This may be due to the experience gained with each successive investigation.

Although limited to three equipment evaluations from a single centre, the process identified multiple deficiencies in the reports. Epidemiological, site data, location descriptions, weather and sea conditions were reliant on police reports, which can often be variable and miss relevant issues. These reports had no standard structure and depended on the local investigating policeman's experience and familiarity with diving, which is often non-existent. In addition, there was a lack of information about the overall hookah system in two reports with deficiencies in description of air intake/exhaust positions (CO risk) in all three reports, and with only one report describing the diver's air hose/weight configuration. This is important for two reasons. First, many hookah divers use their weight belt to attach their air hose to their body. Second, nearly three quarters of all SSBA deaths in a prior series were still wearing weights when rescued or recovered.[11] These two issues may be related – a ditched weight belt may mean an unsecured air hose which may well lead to the loss of air supply and make the diver more reluctant to ditch their weights. In addition, two thirds of that study cohort were not wearing a buoyancy device.[11] Weighting/hose security and lack of buoyancy are factors that can be remedied to prevent diving accidents. A structured checklist could assist with identifying trends in accident data, and we believe this should be useful as a reference for future hookah accident investigations.

As far as we are aware this is the first time a process for hookah technical assessment has been documented. The checklist may require further refinement however, our current version at least ensures that the evaluation process is consistent. This initial work may serve as a basis for more thorough data collection and a deeper understanding of hookah accident causation, and future prevention.

LIMITATIONS

The checklist was created from limited resources and a small sample of hookah equipment investigations and, as a result, some pertinent inclusions may have been missed.

Conclusions

The hookah apparatus is significantly different from scuba equipment and carries higher risk for the diver. It appears to be over-represented in diving fatalities in Tasmania, with equipment failure and air contamination occurring more commonly than with scuba. There is currently no regulation or oversight of the use of the recreational hookah apparatus. To improve accident investigation, a formalised process for technical evaluation of hookah equipment (post-accident), which includes a data collection checklist template specific to hookah was created. The checklist may serve as a resource for future hookah equipment assessments and may inform strategies for preventing future hookah accidents.

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Technical validation of the EMMA capnometer under hyperbaric conditions

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Carbon dioxide; Capnography; Hyperbaric oxygen treatment; Intensive care medicine; Patient monitoring

Abstract
(Tucker A, Smart D. Technical validation of the EMMA capnometer under hyperbaric conditions. Diving and Hyperbaric Medicine. 2023 June 30;53(2):100–110. doi: 10.28920/dhm53.2.100-110. PMID: 37365127.)
Introduction: End-tidal carbon dioxide ($ETCO_2$) monitoring is essential for monitoring intubated critical care patients, yet its use in hyperbaric environments can be problematic. We postulated that the EMMA mainstream capnometer may function accurately under hyperbaric conditions.
Methods: Stage 1. The EMMA mainstream capnometer was tested at 101 kPa against a reference side-stream capnometer, Philips IntelliVue M3015B microstream, using 10 customised reference gases of various carbon dioxide (CO_2) concentrations (2.47%–8.09%, or 18.5–60.7 mmHg at 101 kPa) in either air or oxygen. Stage 2. The functionality and accuracy of the EMMA capnometer was tested under hyperbaric conditions, 121–281 kPa, using the same test gases.
Results: At 101 kPa, the EMMA capnometer measured CO_2 at levels lower than expected (mean of differences = -2.5 mmHg (95% CI -2.1 to -2.9, $P < 0.001$)). The Philips capnometer measured CO_2 more closely to expected CO_2 (mean of differences = -1.1 mmHg (95% CI -0.69 to -1.4, $P < 0.001$). Both devices demonstrated a significant linear relationship with expected CO_2. The EMMA capnometer functioned up to the maximum test pressure (281 kPa). The device over-read CO_2 measurements at pressures > 141 kPa. Although variance increased at pressures in the therapeutic range for hyperbaric treatments, a significant linear relationship between expected and EMMA measured CO_2 was demonstrated. The EMMA capnometer tolerated pressures to 281 kPa, but its display was limited to CO_2 < 99 mmHg.
Conclusions: This study validated EMMA capnometer function to 281 kPa in the hyperbaric environment. The device over-read CO_2 measurements at pressures >141 kPa, however there was a linear relationship between expected and measured CO_2. The EMMA capnometer may be clinically useful for monitoring expired CO_2 in patients undergoing hyperbaric oxygen treatment.

Introduction

Hyperbaric oxygen treatment (HBOT) has approved indications in Australia.[1,2] Some patients requiring hyperbaric oxygen are critically ill. They require the same standard of care and monitoring when pressurised as occurs at 101.3 kPa (1 atmosphere absolute [atm abs]).[3–5] For ventilated patients, pulse oximetry and end-tidal carbon dioxide ($ETCO_2$) are essential for monitoring patients.[6] End-tidal CO_2 monitoring provides real-time evidence of ventilatory compromise such as hyper- or hypoventilation, and displacement or obstruction of the tracheal tube.[7,8] Central nervous system (CNS) oxygen toxicity during HBOT may be worsened by hypercapnia.[9] Preventing hypercapnia is an important reason for monitoring $ETCO_2$ in ventilated patients receiving HBOT.

Hyperbaric medical devices must meet strict safety guidelines.[10] Hyperbaric facilities with critical care capability must test equipment to ensure devices are safe and can function in hyperbaric conditions.[11] Some devices may not physically withstand pressure, requiring modification. Modifications carry a risk of voiding the device warranty. Electrical equipment also produces heat which increases fire risk during HBOT.[12] This risk can even occur for battery-operated (or backed up) equipment, especially if powered by lithium batteries, or if sparks are generated by brushed motors.[13]

Options to minimise risk from electrical devices include not using the equipment or situating the devices outside the chamber and connecting the equipment to sensors (including sampling lines) or effectors inside the chamber via 'penetrators' which traverse the chamber hull.[4,14] In-chamber devices may also be placed in nitrogen-flushed housings to reduce fire risk. In such cases, this limits accessibility to the equipment which then must be operated remotely.

Mainstream $ETCO_2$ determination, in hyperbaric conditions, is subject to errors due to the 'pressure broadening effect'

produced by the increased density of gas. As a result, falsely high values of the patient's $ETCO_2$ are usually reported, which need to be corrected using mathematical equations for each device.[15,16] One study demonstrated a good correlation between $ETCO_2$, using a mainstream capnometer, and P_aCO_2, taken during hyperbaric conditions at 284 kPa.[17] In hyperbaric facilities, devices to monitor $ETCO_2$ may increase fire risk because they produce heat via infrared transmitters, particularly with traditional mainstream devices, exacerbated by 100% oxygen under pressure.

To monitor patients' ventilation, hyperbaric physicians have also used arterial blood gas (ABG) P_aCO_2 analysis, transcutaneous carbon dioxide tension ($P_{TC}CO_2$), or externally connected side-stream $ETCO_2$ samplers. In some facilities, in-chamber $ETCO_2$ monitoring is not even used due to the lack of a suitable and easily applicable device (Personal correspondence with Austrailian clinical leads 2019).

Side-stream capnometers aspirate gas from the breathing circuit. Even at sea level pressures, the measurements may be affected by water removal, different conditions at the sampling site and sample cell (temperature and humidity), mixing of the sample gas when drawn through the cell and variable pressure drop across the tubing. Some of these effects can be compensated, but not all.[18] Side-stream capnometers have been used successfully during hyperbaric treatments, with the mains-powered analyser remaining outside of the chamber, and sampling gas lines exiting through a penetrator. Practical limitations to these devices include: the requirement for additional penetrators and potential delays in displaying and calculating corrected $ETCO_2$ which delays clinical interventions. Mass spectrometry has been successfully used with side-stream analysis of decompressed gas samples, but this is clinically impractical.[19]

The EMMA mainstream capnometer (Masimo, Daneryd, Sweden) is a small, in-line device that contains a CO_2 sensor and display in the same unit. It can be rapidly deployed and displays an averaged $ETCO_2$ and respiratory rate.[20]

Given its compact, small size and low voltage (two AAA alkaline batteries), our study planned to technically evaluate the EMMA capnometer under hyperbaric conditions. We assessed whether it would accurately monitor $ETCO_2$ under a range of hyperbaric pressures (121–283 kPa) using test gases of known CO_2 concentrations.

Methods

Low-risk ethics approval was sought from the Human Research Ethics Committee (HREC) Network (Tasmania) but waived following direct communication from the Chair, due to being an equipment validation study without test subjects (personal communication from Chair of HREC, March 2019).

In stage 1, we tested the reliability of the EMMA mainstream capnometer at 101 kPa against a reference microstream (side-stream) capnometer, Philips IntelliVue M3015B using a range of CO_2-containing test gases 2.47%–8.09% (18.5–60.7 mmHg at 101.3 kPa [1 atm abs]).[21]

In stage 2, the functionality and accuracy of the EMMA capnometer was tested under hyperbaric conditions, 121–281 kPa, using the same calibrated test gases as Stage 1.

A third stage was intended to compare the EMMA capnometer against the same reference microstream capnometer in Stage 1 (Philips IntelliVue M3015B), however, the latter failed to function beyond 110 kPa, the cause of which could not be identified or remediated. This stage was abandoned.

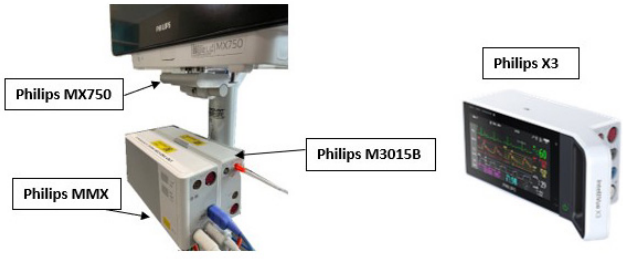
EQUIPMENT

Capnometers

The EMMA mainstream capnometer is a small, alkaline battery-powered, in-line device that contains an infrared CO_2 sensor and display in the same unit (Figure 1). It displays the average maximum measured CO_2 of the last 4 breaths when $ETCO_2$ changes by < 25%, or the last breath when $ETCO_2$

Figure 1
EMMA capnometer

Figure 2
Philips IntelliVue MX750, Philips Microstream capnometer (M3015B), Philips IntelliVue MMX and X3 multi-measurement monitoring modules



changes by > 25%.[20] It also displays a respiratory rate. The EMMA capnometer displays an average of maximum expired CO_2, which in a strict sense, is not the $ETCO_2$. In addition, measurements in this study were conducted on test gases, so we have adopted a convention of describing data values in this study as concentration or partial pressure of CO_2, not $ETCO_2$.

The Philips IntelliVue MX750 (Figure 2) is an integrated critical care monitor. The M3015B (Figure 2) extension module can be attached to either the IntelliVue X3 (Figure 2) or MMX (Figure 2) multi-measurement monitoring modules that integrate with the MX750 monitor. Gas is sampled from a sidestream (microstream M3015B device) which is placed in line with the airway tubing. The infrared CO_2 sensor is located inside the extension module instead of utilising an external sensor.

The MX750 itself was assessed as not suitable for pressurisation, owing to heat production and battery type (lithium ion). Like the MX750, the IntelliVue X3 has an LED touch screen which could be affected with changes in ambient pressure. The screenless MMX monitoring pod and microstream device also contained electronics and motorised pumps. It was assessed by local biomedical technicians as suitable for hyperbaric conditions provided continuous purging with nitrogen occurred.[12] The MX750 power source and output leads traversed the chamber wall via a penetrator. A hyperbaric compatible slave screen inside a nitrogen purged housing allowed viewing of observations within the chamber. Prior to use, both devices were calibrated according to the manufacturer's instructions.[20,21]

Test gases

Ten customised reference gases of various concentrations of CO_2 in either air or oxygen were used. 'Air' and 'oxygen' test gases were chosen to represent a physiological range of expired gas from ventilated patients completing HBOT. This attempted to ensure that the EMMA was validated for the entirety of HBOT, including air breaks. For test cylinders containing 'air', the nitrogen percentage was kept constant, and the oxygen percentage was reduced in substitution of the additional CO_2 (Table 1). 'Oxygen' test cylinders contained a specific CO_2 percentage with the balance of the volumetric composition to 100% made up with oxygen. These were supplied with a certified standard analysis accuracy of ± 2% of the test gas concentration (BOC, Hobart, Australia). Final test gas mixes are summarised in Table 1.

Conversion calculations

Test gases were presented as a percentage of CO_2. Because the capnometers display measured CO_2 in mmHg, all test gas percentages were used to calculate an expected CO_2 (mmHg) measurement for each gas (Table 1). Conversion of test gas percent to mmHg was derived as follows:

Expected PCO_2 (mmHg) = (test gas CO_2%/100) x 760 mmHg
For example, PCO_2 for test gas A1 = (2.58/100) x 760 = 19.6 mmHg

The study was conducted in the Royal Hobart Hospital Department of Diving and Hyperbaric Medicine multi-place hyperbaric chamber (Fink Engineering Triple Lock Chamber S/N:229AH93, 10/2017 - Warana, Queensland, Australia).

Table 1
Test gas mixtures, including calculated expected CO_2 at 101 kPa

Test gas	CO_2 (%)	Expected CO_2 (mmHg @ 101 kPa)	O_2 (%)	N_2 (%)
'Oxygen' test gases				
A1	2.58	19.6	97.4	0
B1	4.06	30.9	95.9	0
C1	5.04	38.3	95.0	0
D1	6.56	49.9	93.4	0
E1	8.09	61.5	91.9	0
'Air' test gases				
A2	2.47	18.8	18.3	79.2
B2	4.05	30.8	17.1	78.8
C2	4.92	37.4	15.7	79.4
D2	6.45	49.0	14.6	78.9
E2	8.00	60.8	12.9	79.1

Chamber pressure was measured in kPa using a Trafag digital transducer gauge (accuracy ± 0.25%, model 8253.77.2417, Trafag, Bubikon, Switzerland). The department is located on the third floor, approximately 10 m above sea level. Local barometric pressure can range from 99 to 104 kPa. During the study period, the pressure ranged between 100 and 101 kPa. For calculations a pressure of 101 kPa (760 mmHg) was assumed. It was accepted that a 0.9% error would occur in the expected CO_2 calculation for test gas mixes. EMMA and Philips capnometer measurements were in whole numbers of mmHg, hence, for a CO_2 measurement in the physiological range, this could lead to greater errors, up to 2.5% (1 mmHg/40 mmHg).

The expected CO_2 for each test gas was calculated at each hyperbaric test pressure (Table 2).
For example, PCO_2 for test gas A1 @ 121 kPa = (2.58/100) x (121/101) x 760 = 23.5 mmHg

Gas delivery and 'airway' apparatus

An oxygen clean flow meter was attached to the test gas cylinder. The test gas was delivered through a test circuit (Figure 3) using standard tubing and connectors. One-way valves were incorporated to prevent backflow.

Both capnometers were placed in series in the circuit. Initial testing demonstrated no difference in output readings if the devices were placed singularly or in a series configuration with either device in the primary position. The exhaust gas was released at least 1.5 m away from the test devices. During chamber testing, flushing occurred at regular intervals to keep the chamber atmosphere at acceptable standards in accordance with AS/NZS4774.2(2019).[22] Chamber temperature and humidity were also kept within the operating ranges of each test device.

Initial testing was performed at 101 kPa to determine the best flow rate and time to reach steady readings for each test device. The Philips device would not show an $ETCO_2$ reading until respiratory effort had been initiated (by detecting changes in flow). To remedy this, a disposable, paediatric resuscitator bag (Laerdal Medical Corporation, Norway), with a ventilation bag volume of 500ml, simulated a respiratory rate of 12·min^{-1}. The paediatric resuscitator bag was used to create variability in the gas flow to simulate ventilation to trigger the Philips capnometer and to allow flushing of the circuit. It achieved both the latter and former with as low volumes as possible so as not to exhaust the test gas supply.

The ventilation bag was compressed by hand to achieve 50–100% of volume delivery. Measured CO_2 readings at 101 kPa were consistent, using this technique. A final flow rate of 3 L·min^{-1} was chosen which produced steady readings for both devices in < 30 seconds.

Repetitions

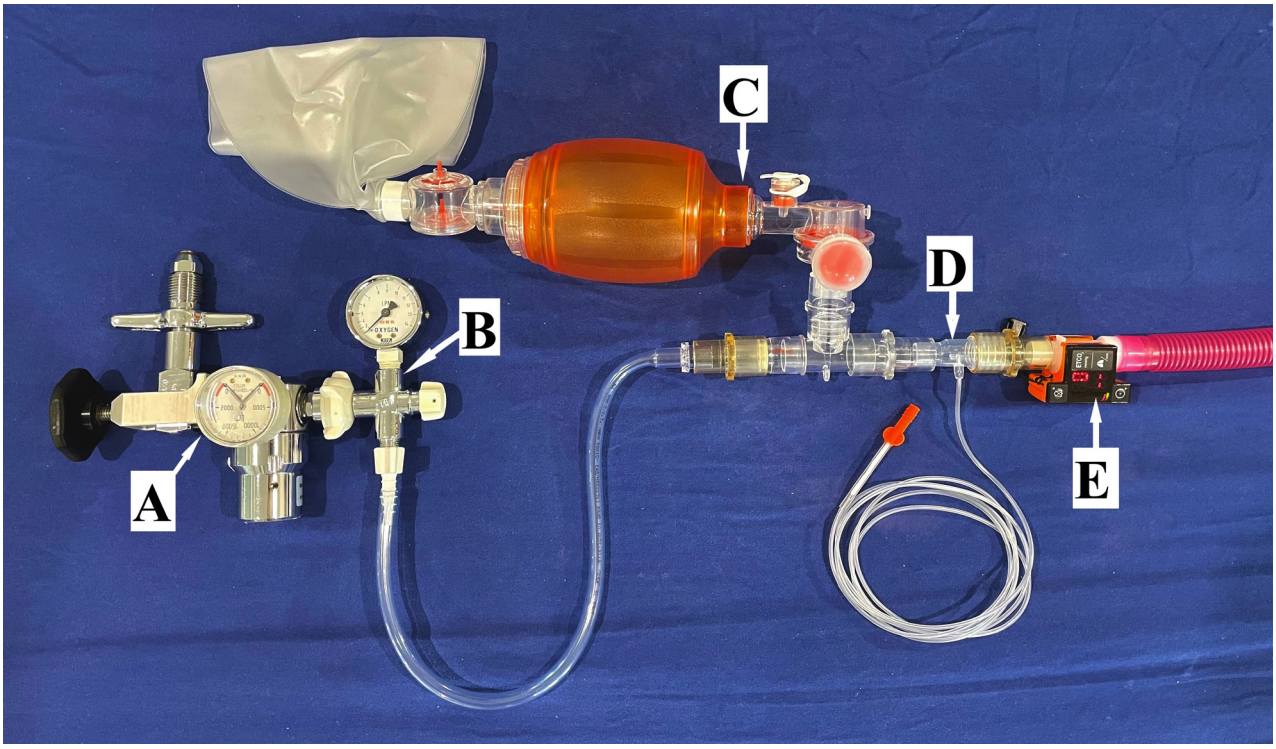
During stage 1, EMMA and Philips capnometer readings were compared at 101 kPa. Ten readings were completed for each test gas at 101 kPa.

In stage 2, measurements of test gases in an ascending pressure profile (20 kPa increments 121–281 kPa) were conducted using the EMMA capnometer. Chamber pressure profiles were within DCIEM dive table no-decompression limits; supervised by trained hyperbaric technicians and

Table 2
Calculated expected CO_2 at various test pressures

Test gas	CO_2 (%)	Expected CO_2 (mmHg)								
		101 kPa	121 kPa	141 kPa	161 kPa	181 kPa	201 kPa	221 kPa	241 kPa	281 kPa
'Oxygen' test gases										
A1	2.58	19.6	23.5	27.4	31.3	35.1	39.0	42.9	46.8	54.9
B1	4.06	30.9	37.0	43.1	49.2	55.3	61.4	67.5	73.6	86.5
C1	5.04	38.3	45.9	53.5	61.1	68.6	76.2	83.8	91.4	107.3
D1	6.56	49.4	59.2	69.0	78.7	88.5	99.2	109.1	119.0	138.7
E1	8.09	61.5	73.7	85.8	98.0	110.2	122.4	134.5	146.7	171.1
'Air' test gases										
A2	2.47	18.8	22.5	26.2	29.9	33.6	37.4	41.1	44.8	52.6
B2	4.05	30.8	36.9	43.0	49.1	55.2	61.3	67.4	73.5	86.2
C2	4.92	37.4	44.8	52.2	59.6	67.0	74.4	81.8	89.2	104.8
D2	6.45	49.0	58.7	68.4	78.1	87.8	97.6	107.3	117.0	136.4
E2	8.00	60.8	72.8	84.9	96.9	109.0	121.1	133.0	145.1	169.2

Figure 3
Test circuit connected to the EMMA and Philips capnometers; A – regulator; B – oxygen clean flow meter; C – paediatric Laerdal bag; D – Philips capnometer; E – EMMA capnometer



clinicians. Ten measurements of CO_2 were made for each test gas at each test pressure.

STATISTICS

Expected CO_2 was paired with the EMMA capnometer and Philips capnometer measured CO_2 at 101 kPa for correlation. Sampling under hyperbaric pressures compared expected CO_2 to the measured CO_2 samples from the EMMA capnometer at each pressure. Data were entered into Excel Spreadsheets (Microsoft® Corporation, Redmond Washington USA) and analysed using GraphPad Prism version 9.1.0.03 for Windows (GraphPad Software, San Diego, California, USA, 2021). Simple descriptive statistics were used to report reproducibility data.

Basic analyses included means, means of differences, standard deviations, and linear regression. Measurement data were subjected to simple linear regression and correlation analysis comparing EMMA Capnometer CO_2 measurements with expected CO_2 for test gases.

Bland-Altman plots were generated to assess agreement between the EMMA measured CO_2 and the expected CO_2 across the range of CO_2 concentrations and hyperbaric pressures. Graphs were produced comparing the EMMA CO_2 measurements at various chamber pressures, compared to expected CO_2 values from the test gas to determine if a predictable relationship would allow a correction equation to be calculated. Statistical significance was accepted when $P < 0.05$.

Results

Data were collected from June 2020 to July 2021.

STAGE 1: DEVICE AGREEMENT AT 101.3 kPa

Measurements across the test gas concentrations using the Philips capnometer at 101 kPa were highly reproducible with standard deviations ranging from 0.0 to 0.7 mmHg (0–1% of the means). Measurements using the EMMA capnometer at 101.3 kPa were also highly reproducible with standard deviations ranging from 0.4 to 0.7 mmHg (0–2% of the means).

Figure 4 shows a graph of the expected CO_2 versus measured EMMA and Philips CO_2 readings at 101 kPa. The grey line indicates the line of exact agreement between the devices and expected CO_2. The EMMA capnometer consistently measured CO_2 at lower levels than expected (mean of differences -2.5 mmHg (95% CI -2.1 to -2.9, $P < 0.001$). The Philips capnometer measured CO_2 more closely to the expected CO_2 (mean of differences -1.1 mmHg (95% CI -0.69 to -1.4, $P < 0.001$). There was a narrow variance in measured CO_2 for both devices. These results were consistent with the manufacturers' stated sensitivities for both devices.[20,21]

Figure 4
Expected CO_2 vs measured CO_2 with EMMA and Philips capnometers at 101 kPa

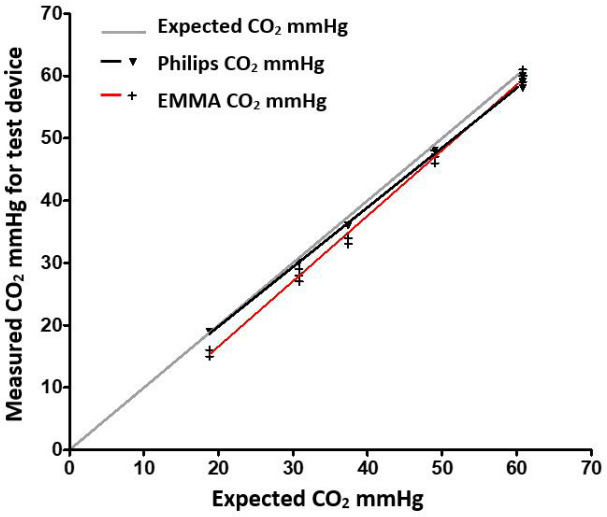


Figure 5
Measured EMMA CO_2 compared to expected CO_2 for oxygen/CO_2 test gases in hyperbaric conditions

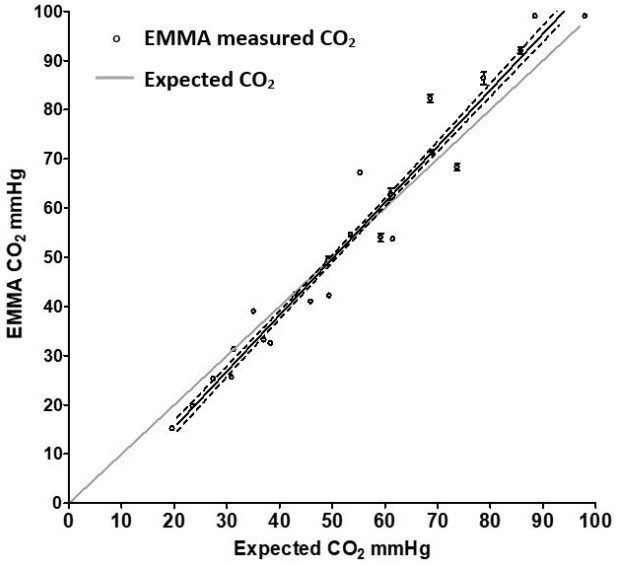


Figure 6
Measured EMMA CO_2 compared to expected CO_2 for air/CO_2 test gases in hyperbaric conditions

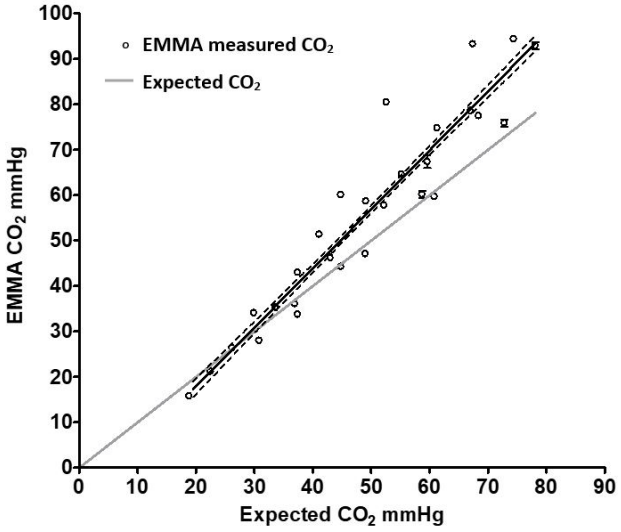
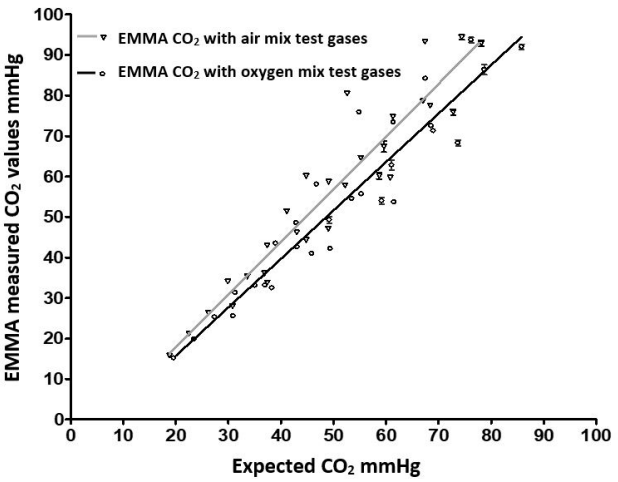


Figure 7
Linear regression graph showing EMMA CO_2 measurements comparing readings during exposure to oxygen and air test gases in hyperbaric conditions



At 101 kPa, both devices demonstrated a linear correlation with expected CO_2 and regression equations could be calculated for each device.

EMMA: Expected CO_2 = EMMA measured CO_2/1.05 + 4.4 (R^2 = 1.0, P < 0.0001).
Philips: Expected CO_2 = Philips measured CO_2/0.96 – 0.7 (R^2 = 1.0, P < 0.0001).

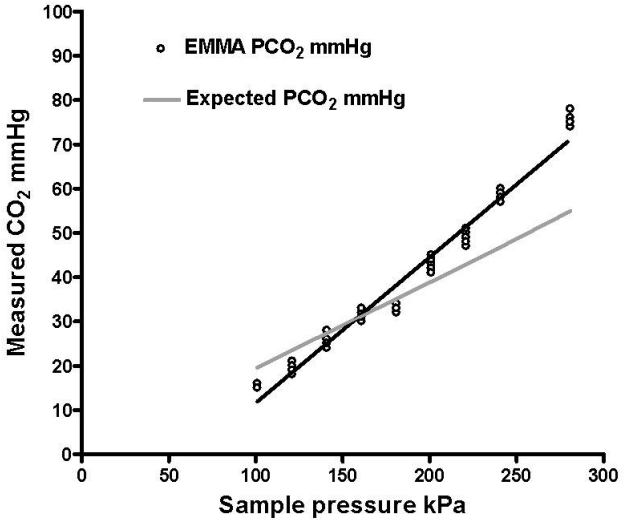
Between devices, the EMMA capnometer under-read compared to the Philips capnometer by a mean of -1.4 mmHg (95% CI -1.8 to -1.0, P < 0.001).

STAGE 2: EMMA CAPNOMETER CO_2 MEASUREMENT UNDER HYPERBARIC CONDITIONS

Figures 5 and 6 show measured EMMA CO_2 versus expected CO_2 for the oxygen/CO_2 and air/CO_2 test gases respectively.

EMMA Device for oxygen/CO_2 Test Gases (Figure 5):
Expected CO_2 = EMMA Measured CO_2/1.2 + 6.9 (R^2 = 0.89, P < 0.0001).

Figure 8
Measured EMMA CO_2 versus expected CO_2 for test gas 2.58% CO_2 in 97.42% oxygen at nine different pressures



EMMA Device for air/CO_2 Test Gases (Figure 6):
Expected CO_2 = EMMA Measured CO_2/1.25 + 6.2 ($R^2 = 0.90$, $P < 0.0001$).

The EMMA device demonstrated a statistically significant linear relationship between measured CO_2 and expected CO_2 for both the oxygen/CO_2 test gas mixes and the air/CO_2 test gas mixes in the hyperbaric environment. The linear regression line gradients were significantly different for oxygen versus air CO_2 measurements ($P = 0.004$) (Figure 7).

The EMMA device consistently under-read the test gas CO_2 pressure for the lower values of expected CO_2. Conversely, as the expected CO_2 increased, the EMMA device consistently over-read the CO_2 values (Figures 5 and 6). This effect was observed for all gas mixes, as pressure increased. An example is shown in Figure 8.

There was a statistically significant greater variance in EMMA CO_2 measurement at higher pressures (> 141 kPa) (Tables 3 and 4).

Bland Altman plots were generated for all samples to assess agreement between the EMMA CO_2 measurements with the expected CO_2 values (Figures 9 and 10). Figure 9 shows the bias of the EMMA capnometer Ratio = 1.01 (SD 0.15) (red line) using 'oxygen' test gases. The 95% limits of agreement for the data = 0.72 to 1.3, represented by the grey lines. Figure 10 shows the bias of the EMMA capnometer Ratio = 1.11 (SD 0.16) (red line), using 'air' test gases. The 95% limits of agreement for the data = 0.80 to 1.42, represented by the grey lines. Both demonstrated a more positive trend of differences with increasing CO_2. This was greater for the 'air' gas samples, for which the bias was also higher. However, the differences were

Table 3
Mean of differences and variance between the EMMA CO_2 readings versus expected for test gases containing air mix; *P*-value for all differences < 0.0001; CI – confidence interval

Pressure (kPa)	Mean of differences (mmHg)	95% CI (mmHg)
101	-2.5	-2.8 to -2.2 (0.6)
121	0.4	-0.26 to 1.1 (1.36)
141	5.2	3.9 to 6.5 (2.6)
161	9.1	7.6 to 11.0 (3.4)
181	17.0	15.0 to 19.0 (4)
201	13.0	10.7 to 15.3 (4.6)
221	18.1	14.3 to 21.9 (7.6)
241	15.3	14.5 to 16.1 (1.6)
281	27.9	27.0 to 28.8 (1.8)

Table 4
Mean of differences and variance between the EMMA CO_2 readings versus expected for test gases containing oxygen mix; *P*-value for differences < 0.0001 except 141 kPa ($P < 0.003$) and 161 kPa ($P < 0.001$); CI – confidence interval

Pressure (kPa)	Mean of differences (mmHg)	95% CI (mmHg)
101	-6.1	-6.5 to -5.7 (0.8)
121	-4.6	-5.1 to -4.1 (1.00)
141	1.4	0.5 to 2.3 (1.8)
161	2.3	0.98 to 3.6 (2.62)
181	9.9	8.3 to 11.0 (2.7)
201	11.33	9.2 to 13.5 (4.3)
221	11.2	8.5 to 13.9 (5.4)
241	11.3	10.6 to 12.0 (1.4)
281	21.0	19.9 to 22.1 (2.2)

Figure 9
Bland Altman plot of EMMA CO_2 vs expected CO_2 ratio using oxygen test gases

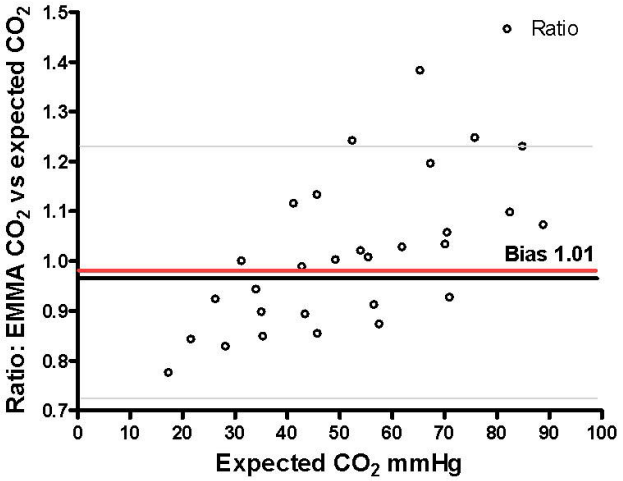
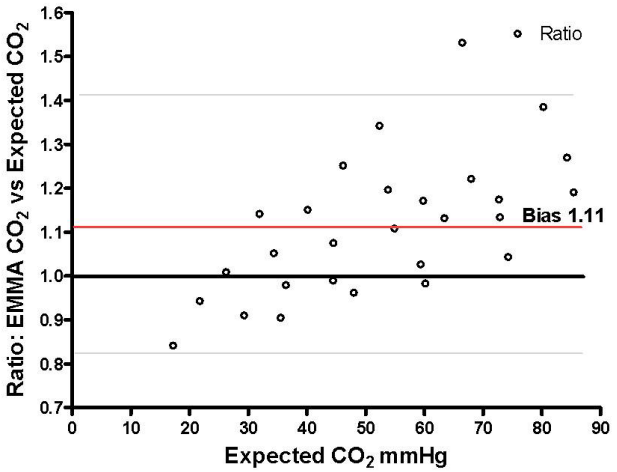


Figure 10
Bland Altman plot of EMMA CO_2 vs expected CO_2 ratio using air test gases



more constant for values within the physiological range of CO_2 (30–60 mmHg) for both types of test gases.

Of note, data collection was limited by the EMMA device having a maximum displayed value of 99 mmHg CO_2. The maximum reading of 99 mmHg was surpassed for a number of the test gases with increased chamber pressures. As a result, measurable data could only be obtained to 141 kPa or 161 kPa for some test gases. Only test gases A1 and A2 were able to be tested to our maximum test pressure of 281 kPa.

Discussion

Our study was designed to technically validate the function of the EMMA capnometer under hyperbaric conditions.

BETWEEN DEVICE AGREEMENT AT 101.3 kPa

At 101 kPa, both the EMMA and Philips capnometers had a linear relationship with the expected CO_2. The EMMA capnometer consistently under-read, by a mean of 2.5 mmHg, compared to the expected CO_2. Similarly, the Philips IntelliVue microstream capnometer also under-read by a mean of 1.1 mmHg.

The two test devices were limited to whole number displays of CO_2, which could produce an error of ± 0.5 mmHg (1.25% at physiological $ETCO_2$ of 40 mmHg). The consistent CO_2 measurements demonstrated that both devices are sufficiently accurate for their intended clinical purpose of monitoring expired CO_2 at 101 kPa. Correction equations for expired CO_2 could be used, but the significant linear correlation means both devices reflect accurate trends in expired CO_2 without the need to apply correction calculations during clinical practice. These results were consistent with the manufacturers' stated sensitivities for both devices.[20,21]

The EMMA capnometer under-read compared to the Philips capnometer by a mean of -1.4 mmHg (95% CI -1.8 to -1.0, $P < 0.001$). Two previous studies have evaluated the EMMA capnometer against integrated $ETCO_2$ monitors under normobaric conditions. In patients undergoing a planned general anaesthetic, one study demonstrated a consistent between-device bias of -2.2 mmHg (limits of agreement -6.0 to +1.6) compared with a reference side-stream capnometer.[23] Another group compared nine EMMA capnometers with an integrated anaesthetic machine side-stream capnograph, using one participant. This study showed good agreement between the devices (median bias -0.3 mmHg), despite study limitations.[24] Both of the above studies assumed that the anaesthesia $ETCO_2$ equipment was the gold standard. Our study showed similar results using test gases. The EMMA capnometer would be an acceptable alternative to the Philips capnometer to monitor trends in CO_2 under normobaric conditions, especially in circumstances where mains power was not available.

VALIDATION OF THE EMMA CAPNOMETER UNDER HYPERBARIC CONDITIONS

Our literature search identified no published studies evaluating the functionality of the EMMA capnometer under increased ambient pressures. Given its size, portability and battery power, the device has potential for monitoring critical care patients in hyperbaric facilities.

Our study demonstrated a linear increase of the EMMA CO_2 measurements with increasing pressures. However, the EMMA capnometer measurements were lower than expected at low pressures and conversely higher than

expected at higher pressures (> 141 kPa). The slope of the EMMA CO_2 line was greater than the expected CO_2 slope. Additionally, there was greater variance in the measured CO_2 data collected where expected CO_2 was above the physiological range for $ETCO_2$ (Figures 5–10). This may be due to both pressure and collision broadening. Despite this, the EMMA device demonstrated a statistically significant linear relationship between measured CO_2 and expected CO_2 for the oxygen/CO_2 test gas mixes and the air/CO_2 test gas mixes in the hyperbaric environment. The EMMA device potentially could be used to monitor trends in the hyperbaric environment.

We chose to use test gases, as opposed to test subjects, to remove the additional variables of inter-person variability in CO_2 production and elimination, and the challenges created by sample dilution and gas bypass in non-intubated patients. Our study cannot be considered an accurate representation of what would occur in a human subject.

The test gases were supplied as a specific concentration of CO_2, so it is expected with increased pressure, the percentage of CO_2 would stay the same, but the measured CO_2 partial pressure would increase (Dalton's Law). The use of test gases is not the same as for a human subject exhaling CO_2. A stable patient should produce CO_2 at a constant rate, and a consistent partial pressure of $ETCO_2$ at 101 kPa. At higher chamber pressures, metabolic CO_2 would be diluted in the alveoli by the extra molecules of oxygen (+/- nitrogen). Theoretically, this would reduce alveolar CO_2 but maintain a measured $ETCO_2$ within physiological ranges. Additionally, the test gases used during the study were dry. In humans, exhaled gas is normally saturated in water vapour, at body temperature. Its presence would likely further reduce measured $ETCO_2$ when used clinically.[18] Therefore, $ETCO_2$ at all pressures should remain in a range that could be measured by the EMMA capnometer (< 99 mmHg). EMMA measurements in the physiological range had less deviation from expected CO_2, so the EMMA device may be useful clinically, particularly to monitor trends.

One group tested the SpaceLabs Medical 90369G mainstream capnometer using a range of test gases with known CO_2 concentrations (in oxygen only) under hyperbaric conditions.[25] This study tested the device at only one experimental pressure of 243 kPa (2.4 atm abs), a typical hyperbaric oxygen treatment pressure. Five test gas concentrations were selected to reflect an expected range of PCO_2 from 20.1–78.3 mmHg at this experimental pressure. Our findings with the EMMA capnometer, were similar to theirs. Their device also read erroneously high under hyperbaric conditions, which they presumed related to calibration issues, pressure broadening and collision broadening from oxygen. They identified a correction equation applicable to their device, but only under specific conditions of oxygen and pressure.

We used test gases of known CO_2 concentration in either an 'air' or 'oxygen' mix. Patients undergoing HBOT are treated with 100% oxygen periods with intermittent air breaks, to reduce the risk of oxygen toxicity.[26] Our test gases were chosen to represent typical $ETCO_2$ in HBOT patients throughout their treatment period. We chose a range of CO_2 percentages to represent a range of $ETCO_2$ of 20–60 mmHg at 101 kPa. These values represent normal physiological $ETCO_2$ extended to include levels expected with possible hyper- or hypoventilation. Our study was limited by the inability to assess all test gases to our maximum experimental pressure of 281 kPa as some test gas expected CO_2 pressures were higher than the display capability (99 mmHg) of the EMMA device.

It is possible that a collision broadening effect may influence capnometer-measured CO_2 readings. This phenomenon affects the sensitivity of infrared analysers which leads to erroneous CO_2 readings.[27] It results in the broadening of spectral absorption peaks of a gas (e.g., CO_2) due to the collision or proximity of molecules of another gas, e.g., N_2O, oxygen. Typically, the addition of molecules such as He, N_2O and H_2O to a gas tends to cause erroneously higher CO_2 readings because the energy absorbed by a carbon dioxide molecule is transferred to the larger (additional) molecule when the two collide, permitting the carbon dioxide molecule to absorb more infrared energy, resulting in less infrared reaching the capnometer detector and a higher CO_2 reading.[28] This effect is less pronounced with homonuclear diatomic gases such as oxygen and nitrogen.[28] Capnometers are usually calibrated with known concentrations of CO_2 in nitrogen and oxygen at 101 kPa.[28] The practical consequences of collision broadening influencing the EMMA CO_2 readings for either our 'air' or 'oxygen' test mixes should be low, but data are limited from devices used at higher ambient pressures. In our study, both air and oxygen test gas mixes demonstrated a linear relationship between measured and expected CO_2 under normobaric and hyperbaric conditions with conversion equations able to be derived for both. However, there was a statistically significant difference between the air versus the oxygen test gas mixes under hyperbaric conditions, with more consistent readings (compared with expected) and a shallower slope with the oxygen test gases (Figures 7, 9–10). This could be due to the collision broadening effect of oxygen versus nitrogen. Even though this is statistically significant, the difference is so small, it would be unlikely to be clinically significant, and for practical purposes, the EMMA device can be used to monitor trends both during oxygen treatment periods and air breaks.

One group demonstrated that $ETCO_2$ readings were erroneously high in hyperbaric conditions, which was attributed to the pressure broadening effect of increased gas density.[15,16] This is a similar concept to collision broadening described above, however, it is the density of the molecules that increases collisions and ergo energy absorption and

alters the infrared detection causing erroneously high measured CO_2 at higher pressures. Pressure broadening could be the main influence from nitrogen and oxygen on $ETCO_2$ measurements when measured at pressures > 101 kPa. Again, despite this phenomenon, a linear relationship could be derived for both groups of test gases.

Data collection was limited by the EMMA device having a maximum displayed value of 99 mmHg CO_2. With increased pressures, there was an expected increase in CO_2 of the test gases. As per Table 2, we predicted that the expected CO_2 would surpass 99 mmHg for test gases C1 and C2 at 281 kPa, D1 and D2 by 201 kPa and for E1 and E2 by 181kPa. However, during our study, we observed that as the pressure increased, the EMMA device consistently overread at pressures higher than 141 kPa as well as having greater variance (Tables 3 and 4). The maximum reading of 99 mmHg was surpassed earlier than expected. As a result, measurable data could only be obtained to 141 kPa or 161 kPa for some test gases. Only test gases A1 and A2 were able to be tested to our maximum test pressure of 281 kPa. A possible solution for the maximum display value would be increasing the display capabilities of the EMMA device from two to three digits. However, as per previous reasoning, the EMMA capnometer display range should be adequate for clinical monitoring in the hyperbaric environment.

Conclusion

This study validated the function of the EMMA capnometer in the hyperbaric environment. The device over-read CO_2 measurements at test pressures > 141 kPa, however there was a linear relationship between expected and measured CO_2. These data suggests that the EMMA capnometer may be clinically useful for monitoring $ETCO_2$ trends in patients undergoing hyperbaric oxygen treatment.

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Hyperbaric oxygen and treadmill exercise partially prevented bone loss and bone microarchitecture deterioration in ovariectomized rats

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Keywords
Animal model; Endocrinology; Hyperbaric oxygen treatment; Osteoporosis; Ovariectomy

Abstract
(Peng X, Gao B, Wang X, Qin X, Peng M, Zeng X. Hyperbaric oxygen and treadmill exercise partially prevented bone loss and bone microarchitecture deterioration in ovariectomized rats. Diving and Hyperbaric Medicine. 2023 June 30;53(2):111–119. doi: 10.28920/dhm53.2.111-119. PMID: 37365128.)

Introduction: Previous studies have demonstrated the beneficial effects of treadmill exercise (EX) on osteoporosis, and of hyperbaric oxygen (HBO) on osteoblast and osteoclast formation *in vitro*. We investigated the effects of HBO and the combination of HBO and EX on osteoporosis in ovariectomized rats.

Methods: Forty 3-month-old female Sprague-Dawley rats were randomly divided into 5 groups (n = 8): a sham control group (Control); an ovariectomy group; an ovariectomy with treadmill exercise treatment group; an ovariectomy with HBO treatment group; and an ovariectomy with HBO treatment combined with treadmill exercise group. The HBO exposures were 203 kPa, 85–90% O_2, 90 min and the exercise regimen was 20 m·min^{-1}, 40 min·day^{1}, 5º slope. Both treatments were administered once daily, five days a week for 12 weeks until the rats were sacrificed.

Results: All three treatments (HBO, exercise, and both combined) significantly promoted the expression of the osteoblast-related gene and oxidative metabolism-related gene (PGC-1α). They also exerted significant inhibitory effects on the osteoclast-related mRNA expression (RANKL) and bone resorption marker CTX-I. Additionally, exercise and the combination exercise-HBO treatment increased serum superoxide dysmutase (SOD) and sclerostin expression. No significant between-group difference was observed.

Conclusions: Hyperbaric oxygen, exercise, and the combination ameliorated bone microarchitecture deterioration and ovariectomy-induced bone loss in rats, and these inhibitory effects may be associated with the increased SOD and up-regulated PGC-1α.

Introduction

Osteoporosis, a systemic bone disease characterised by bone mass reduction and bone microstructure destruction, can increase bone fragility and predispose to fractures. With the aging population, the incidence of osteoporosis is increasing which in turn increases the medical expenditure on fractures worldwide.[1] It is expected that the number of osteoporotic fractures will double by 2050.[2] Therefore, the prevention and treatment of osteoporosis is clinically important.

Osteoporosis can be treated by drug and non-drug therapies. In drug therapy, bone resorption inhibitors and bone formation promoters are commonly applied. Though used widely with good results in clinical practice, drug therapy has certain disadvantages, such as long medication cycles, high treatment costs, poor patient compliance, and adverse drug reactions.[3,4] Therefore, developing effective non-drug therapies for osteoporosis, such as pulsed electromagnetic field, ultrasound, extracorporeal shock wave, electroacupuncture, exercise therapy and hyperbaric oxygen (HBO) treatment, has become a focus of current research.

One study showed that a combination of mildly elevated pressure and inspired oxygen fraction (133 kPa, 40% O_2) exerted protection against osteoporosis induced by hindlimb unloading.[5] Ultra-early HBO (223 kPa, 97–99% O_2) can promote bone conversion, bone formation, improve bone mass and inhibit bone resorption in rats with osteoporosis induced by complete spinal cord injury.[6,7] Osteogenic-differentiating mesenchymal stem cells exposed to HBO (243 kPa, 100% O_2) under *in vitro* simulated inflammatory conditions exhibited enhanced differentiation towards the

osteogenic phenotype.[8] Clinically, 95–100% O_2 at 203 kPa (2 atmospheres absolute [atm abs]) is a commonly used HBO protocol in humans. The effects of HBO may vary if evaluated with different protocols.

Exercise therapy, another well-studied non-drug treatment in recent years, has been shown to exert a positive influence on the skeleton, and subsequently prevent or improve osteoporosis.[9] Exercise (EX) has been shown to benefit bone modeling by promoting bone marrow mesenchymal stromal cells, and to improve the bone metabolism of ovariectomized rats with osteoporosis.[10,11] Several studies[12,13] have investigated the effects of combining physical exercise and drugs for the treatment of osteoporosis. However, reports regarding the effects and mechanism of treadmill EX plus HBO, or HBO alone, on osteoporosis in ovariectomized rats are limited.

The purpose of this study was to evaluate the effect of HBO (203 kPa, 85–90% O_2, 90 min), EX (20 m·min^{-1}, 40 min·day^{-1}, 5-degree slope) and combining HBO and EX on osteoporosis in ovariectomized rats and to explore the mechanism of any benefit to provide a mechanistic basis for the clinical application of these therapies.

Methods

These procedures were carried out in accordance with the Animal Protection Law of the People's Republic of China (2019/315) and approved by the Ethics Committee of Sichuan Provincial People's Hospital (protocol code: 2019/315, date of approval 28 November 2019).

EXPERIMENTAL ANIMALS AND OVARIECTOMIZED MODELS

Forty 3-month-old female Sprague-Dawley rats, each weighing about 270 g, were purchased from Si Chuan Chengdu Dasuo Experimental Animal Co. Ltd. (license key: syxk (chuan) 2013-110). Prior to the experiment, all rats were housed in cages at room temperature (20–25°C) in an atmosphere of 60–70% humidity under a 12/12 h light/dark cycle for one week to minimise the physiological and psychological effects which might result from a new environment. Access to water and food was unrestricted. According to a random digit table, the rats were divided into five groups (n = 8 each): a sham-operated control group ('control'), ovariectomy group ('OVX'), ovariectomy with treadmill exercise treatment group ('OVX+EX'), ovariectomy with HBO treatment group ('OVX+HBO'), ovariectomy with HBO treatment combined with treadmill exercise group ('OVX+HBO+EX').

All rats underwent either a sham surgery or bilateral ovariectomy following a standard protocol.[14] All treatments were carried out 14 days post-ovariectomy. The rats were weighed monthly and euthanised at the end of the intervention. Blood was collected from the abdominal aorta prior to sacrifice. The uterus was carefully dissected and weighed for each rat to evaluate the estrogen agonistic activity.

HYPERBARIC OXYGEN (HBO) TREATMENT

The animal hyperbaric oxygen chamber (Yantai Hong Yuan Hyperbaric Oxygen Chamber Co. Ltd., China) was provided by Sichuan Provincial People's Hospital. The rats were compressed over 30 minutes to a pressure of 203 kPa (2 atm abs) where they remained for 40 minutes before being depressurised over 20 minutes. Rats breathed 85% oxygen for the duration of the treatment cycle (90 minutes).

The HBO exposure was applied once per day, 5 days per week for a total of 12 weeks. Rats not receiving hyperbaric treatment received air at atmospheric pressure.

EXERCISE PROTOCOLS

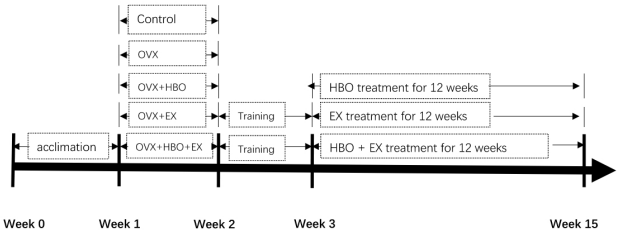
All animals ran on a six-channel motor-drive treadmill (Anhui Zheng Hua Co. Ltd, China) at a speed of 12–16 m·min^{-1} for 40 min·day^{-1} for the first week in order to reduce stress during the training period. The rats did not receive any electric stimulus to run, but manual stimulation was applied. The exercise groups participated in a running program at a constant speed of 20 m·min^{-1} for 40 min, five days per week, with a 5-degree angle for 12 weeks, 14 days post-ovariectomy. The nontrained rats were placed on the switched-off treadmill with the same duration. The experimental flowchart of the present study is shown in Figure 1.

SERUM PARAMETERS FOR BONE METABOLISM

Serum bone formation biomarker procollagen type I N-terminal propeptide (PINP) (Elabscience, China) and bone resorption biomarker C-terminal telopeptides of

Figure 1

The experimental flowchart of the present study; Week 0 – rats acquired; Week 1 – modeling rats and allocation to five groups (n = 8 each); week 15 – euthanasia at the end of the intervention; Control – sham-operated control group; OVX – ovariectomy group; OVX+EX – ovariectomy with exercise treatment group; OVX+HBO – ovariectomy with hyperbaric oxygen treatment group; OVX+HBO+EX – ovariectomy with hyperbaric oxygen treatment combined with exercise group



type I collagen (CTX) (Elabscience, China) were used to evaluate bone turnover. Serum superoxide dismutase (SOD) (Elabscience, China) was used to evaluate antioxidant capacity.

MICRO-CT FOR BONE MASS AND MICROSTRUCTURE

The left femurs of the rats were fixed with 10% formalin for 24 h and scanned using a laboratory micro-CT scanner (Quantum GX; PerkinElmer, Waltham, MA). The basic parameters of the scanner were as follows: X-ray energy, 80 kV; current intensity, 88 μA; scan time, 14 min; pixel size, 50 μm. A total of 512 slices were scanned for each femur. Then, three-dimensional images were reconstructed by PerkinElmer Analyze 12.0 software (PerkinElmer, Waltham, MA) and a series of planar cross-sectional images were generated. Regions of interest were manually selected to define the subchondral bone plate and subchondral trabecular bone of the femur. The bone microstructure parameters included bone volume to tissue volume ratio (BV/TV), trabecular number (Tb. N), trabecular thickness (Tb. Th), and trabecular thickness separation (Tb. Sp).

BONE HISTOLOGICAL ANALYSIS

Following micro-CT scan, the left femurs were decalcified for 2–3 months in 20% ethylene diamine tetra-acetic acid, processed and embedded in paraffin wax. Coronal sections from the middle of the femur (5 μm) were stained with hematoxylin eosin (H&E) and tartrate-resistant acid phosphatase (TRAP) (Sigma-Aldrich, St. Louis, MO) for histological analysis. TRAP-positive multinucleated cells (three or more nuclei as osteoclasts) were observed in the regions 2 mm beneath the growth plate.

REAL-TIME POLYMERASE CHAIN REACTION (PCR) FOR BONE-RELATED GENE EXPRESSION

After bone marrow removal, total RNA was extracted from the distal metaphyses of right femurs using Trizol reagent, following the protocol of the Eastep Super Total RNA Extraction Kit (Promega, Shanghai, China). Then, the PrimeScript RT reagent kit (Takara-Bio, Otsu, Japan) was used to synthesize cDNA from RNA. Polymerase chain reaction tests were conducted using an ABI 7300 Real-Time PCR system using the SYBR Premix Ex Taq II kit (Takara-Bio, Otsu, Japan). The primers were synthesized (Qinke Biotech, Beijing, China) and the sequences are listed in Table 1. Each RNA quantification was carried out in triplicates in a 96-well plate, and the operation was performed on each sample three times. The relative mRNA expression levels were normalised to the glyceraldehyde-3-phosphate dehydrogenase (GAPDH) for each sample and analysed using the $2-\Delta\Delta Ct$ relative quantification method.

STATISTICAL ANALYSES

All data were expressed as mean (standard deviation [SD]) and statistical analyses were performed using IBM SPSS Statistics 19 software and GraphPad Prism 8 (GraphPad Software, San Diego, CA). Statistically significant differences were assessed by one-way analysis of variance (ANOVA). A P-value of < 0.05 was considered significant.

Results

EFFECTS OF HBO AND EX ON BODY WEIGHT AND UTERINE MASS IN OVARIECTOMIZED RATS

The body weight of rats in all groups during the experimental period is shown in Figure 2. No significant differences in

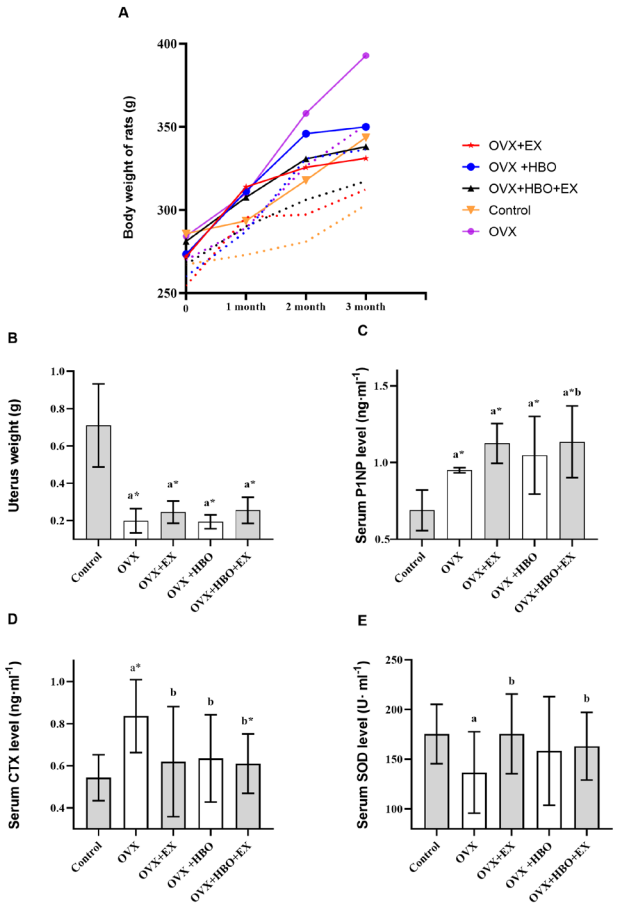
Table 1

Sequence of primers for real-time fluorescence quantitative polymerase chain reaction; GAPDH–glyceraldehyde-3-phosphate dehydrogenase; OCN – osteocalcin; PGC-1α – peroxisome proliferator-activated receptor γ coactivator 1α; RANKL – receptor activator of nuclear factor κB ligand; SOST – sclerostin

Gene	(5′–3′)	Sequence
GAPDH	Forward	TGC ACC ACC AAC TGC TTA G
	Reverse	GGA TGC AGG GAT GAT GTT C
OCN	Forward	ACC CTC TCT CTG CTC ACT CTG CT
	Reverse	GCT GGG GCT CCA AGT CCA TT
SOST	Forward	GAG TAC CCA GAG CCT CCT CA
	Reverse	AGC ACA CCA ACT CGG TGA
RANKL	Forward	TGC TCA CCT CAC CAT CAA TGC
	Reverse	GTT GCT TAA CGT CAT GTT AGA GAT C
PGC-1α	Forward	CGA TGA CCC TCC TCA CAC CA
	Reverse	TTG GCT TGA GCA TGT TGC G

Figure 2

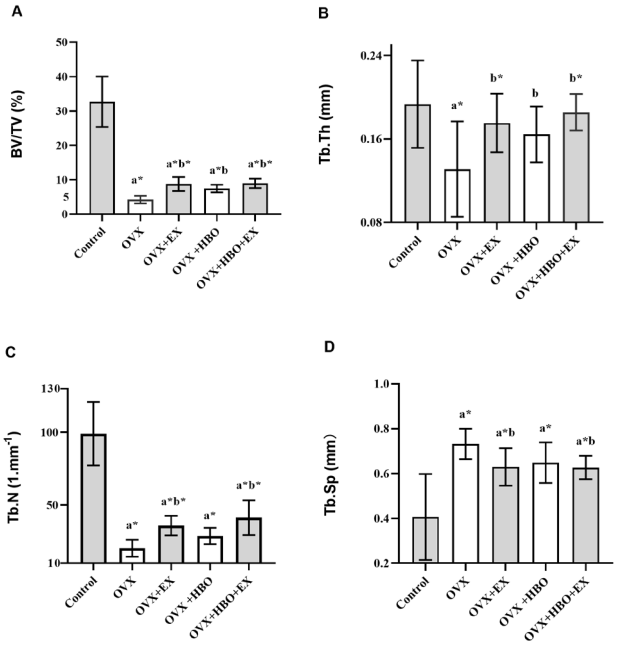
Changes in metabolic parameters (n = 8 each group); data are mean (SD); A – monthly body weight of OVX rats; B – uterus weight after sacrifice; C – serum N-terminal propeptide of type 1 procollagen (P1NP); D – C-terminal cross-linked telopeptides of type I collagen (a bone resorption marker) (CTX-I); E – serum superoxide dismutase (SOD); a – $P < 0.05$ or $^*P < 0.01$ versus Control; b – $P < 0.05$ or $^*P < 0.01$ versus OVX; Control – sham operated control group; OVX – ovariectomy group; OVX+EX – ovariectomy with exercise treatment group; OVX+HBO – ovariectomy with hyperbaric oxygen treatment group; OVX+HBO+EX – ovariectomy with hyperbaric oxygen treatment combined with exercise group



body weight were found among groups at beginning of the experiment. The body weight of rats in the OVX group was significantly increased at two months (11.24%, 358.1 (31.8) g vs. 317.9 (36.9) g, $P < 0.05$; Figure 2A) and 3 months (14.33% 392.9 (41.7) g vs. 343.6 (40.5) g, $P < 0.05$; Figure 2A) after the ovariectomy compared with the control group. However, an inhibitory effect on OVX-induced body weight gain was found in all the intervention groups when compared to the OVX group at 3 months, and the differences were significant ($P < 0.01$ Figure 2A). Furthermore, significantly lowered uterine weight was found in groups receiving ovariectomy compared to the control group ($P < 0.01$; Figure 2B), indicating the successful establishment of the estrogen withdrawal model.

Figure 3

Changes of micro-CT parameters of subchondral trabecular bone in left femur; (n = 8 each group); data are mean (SD); A – bone volume fraction (BV/TV); B – trabecular thickness (Tb.Th); C – trabecular number (Tb.N); D – trabecular separation (Tb.Sp); a – $P < 0.05$ or $^*P < 0.01$ versus Control; b – $P < 0.05$ or $^*P < 0.01$ versus OVX; Control – sham operated control group; OVX – ovariectomy group; OVX+EX – ovariectomy with exercise treatment group; OVX+HBO – ovariectomy with hyperbaric oxygen treatment group; OVX+HBO+EX – ovariectomy with hyperbaric oxygen treatment combined with exercise group



SERUM BIOCHEMICAL ANALYSIS

The serum biomarkers for bone formation and bone resorption are shown in Figures 2C and 2D. Ovariectomy resulted in an increase in serum P1NP (a bone formation marker) ($P < 0.01$) compared with the control. After the 12-week intervention, the serum P1NP was significantly increased in the OVX+HBO+EX combination group ($P < 0.01$, Figure 2C). Although the levels of P1NP in the HBO or EX rats were higher than that in the OVX rats, no significant differences were observed. Furthermore, the serum CTX-I was significantly higher in the OVX group than in the control group ($P < 0.01$, Figure 2D). The OVX rats subjected to HBO, EX and the combination therapy showed significantly decreased serum CTX-I concentration compared with the OVX group ($P < 0.05$, $P < 0.05$, $P < 0.01$, respectively, Figure 2D). The serum SOD, an important scavenger of oxygen free radicals, was significantly decreased in OVX rats compared with the control group ($P < 0.05$, Figure 2E), but increased in the groups receiving EX and the combination therapy compared with the OVX group ($P < 0.05$ Figure 2E). No significant difference was observed between the HBO and OVX rats ($P > 0.05$, Figure 2E).

Figure 4

Histological appearance (hematoxylin and eosin staining) of the left femur samples; all images are shown at ×100 magnification; Control – sham operated control group; OVX – ovariectomy group; OVX+EX – ovariectomy with exercise treatment group; OVX+HBO – ovariectomy with hyperbaric oxygen treatment group; OVX+HBO+EX – ovariectomy with hyperbaric oxygen treatment combined with exercise group

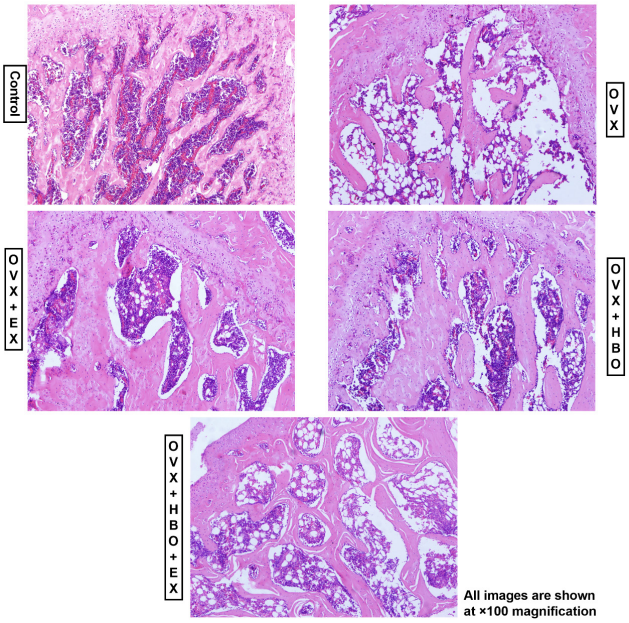
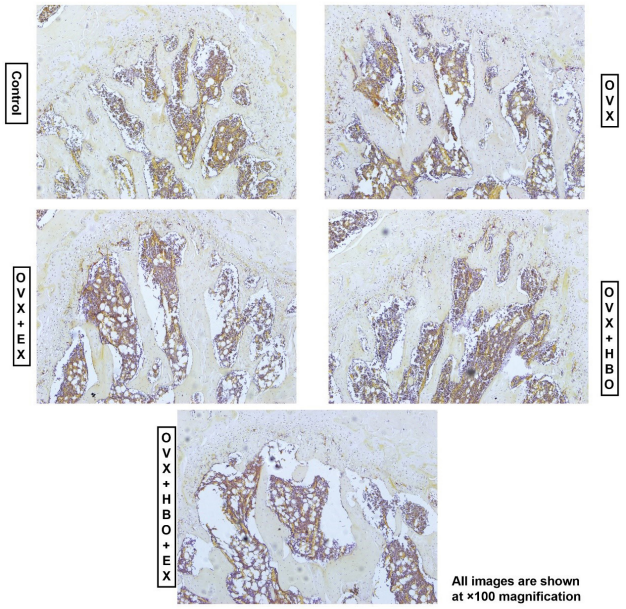


Figure 5

Histological appearance (tartrate-resistant acid phosphatase staining) of the left femur samples; all images are shown at ×100 magnification; Control – sham operated control group; OVX – ovariectomy group; OVX+EX – ovariectomy with exercise treatment group; OVX+HBO – ovariectomy with hyperbaric oxygen treatment group; OVX+HBO+EX – ovariectomy with hyperbaric oxygen treatment combined with exercise group



MICRO-CT ANALYSIS

Micro-CT analysis showed that the OVX group had an 87.1% reduction in BV/TV ($P < 0.05$, Figure 3A), a 32.2% reduction in Tb.Th ($P < 0.05$, Figure 3B), a 79.5% reduction in Tb.N ($P < 0.01$, Figure 3C), and a 180.1% growth in Tb. Sp ($P < 0.05$, Figure 3D) after the surgery, in comparison to the control group. However, the parameters Tb.Th were not altered in all the intervention groups. In comparison to the OVX group, the OVX+EX, OVX+HBO, and OVX+HBO+EX groups showed 2.07-, 1.77- and 2.12-fold higher values in BV/TV respectively ($P < 0.01$, $P < 0.05$, $P < 0.01$). Similar changes were seen in Tb.Th (1.34-, 1.25-, 1.42-fold higher, $P < 0.01$, $P < 0.05$, $P < 0.01$ respectively) and Tb.N (1.77-, 1.42-, and 2.04-fold higher, $P < 0.01$, $P > 0.05$, $P < 0.01$, respectively), but Tb.Sp was lower in the OVX+EX, OVX+HBO, and OVX+HBO+EX groups (0.86-, 0.89-, and 0.86-fold lower, $P < 0.05$, $P > 0.05$, $P < 0.05$ respectively) (Figure 3).

HISTOLOGICAL ANALYSIS

Hematoxylin and eosin staining (Figure 4) showed that in the OVX group, under the growth plate, trabeculae were thinner, less abundant, and spaced at greater distances. In the control group, the trabeculae appeared normal. In all the intervention groups, the trabeculae exhibited a near-normal histological appearance with increased trabecular bone area and trabecular number and decreased marrow cavity.

Tartrate-resistant acid phosphatase staining (Figure 5) showed significantly increased number and size of TRAP-positive multinucleated cells in the OVX group ($P < 0.01$) compared with the control group. In contrast, the number of TRAP-positive multinucleated cells in the rats from the intervention groups was less than that of OVX group (Figure 6), and the differences between them were obvious ($P < 0.01$). Moreover, a significant difference was observed in the TRAP-positive multinucleated cells between the EX group and OVX+HBO+EX group ($P < 0.01$).

REAL-TIME PCR ANALYSIS

The quantification results of rat femur gene expression via real-time PCR analysis are shown in Figure 7. The OVX group showed increased mRNA levels of osteocalcin (OCN) compared with the control group (Figure 7A). All the intervention groups demonstrated significantly upregulated femur OCN gene expression compared with the OVX group. We also found a significantly increased mRNA expression of receptor activator of nuclear factor κB ligand (RANKL) and sclerostin (SOST) in the OVX group compared with the control group (Figure 7B–C), and all the intervention groups had significantly downregulated femur SOST and RANKL gene expression compared with the OVX group. As for the expression of the peroxisome proliferator-activated receptor γ coactivator-1α (PGC-1α) gene, a significant decrease was observed in the OVX group compared with the control group, while all the intervention groups showed

Figure 6

Numbers of tartrate-resistant acid phosphatase staining positive cells per unit bone sample by treatment group; a – $P < 0.05$ or $^*P < 0.01$ versus Control; b – $P < 0.05$ or $^*P < 0.01$ versus OVX; c – $P < 0.05$ or $^*P < 0.01$ versus OVX+HBO+EX; Control – sham operated control group; OVX – ovariectomy group; OVX+EX – ovariectomy with exercise treatment group; OVX+HBO – ovariectomy with hyperbaric oxygen treatment group; OVX+HBO+EX – ovariectomy with hyperbaric oxygen treatment combined with exercise group

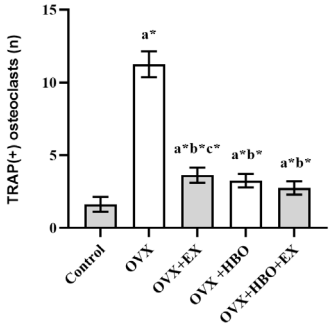
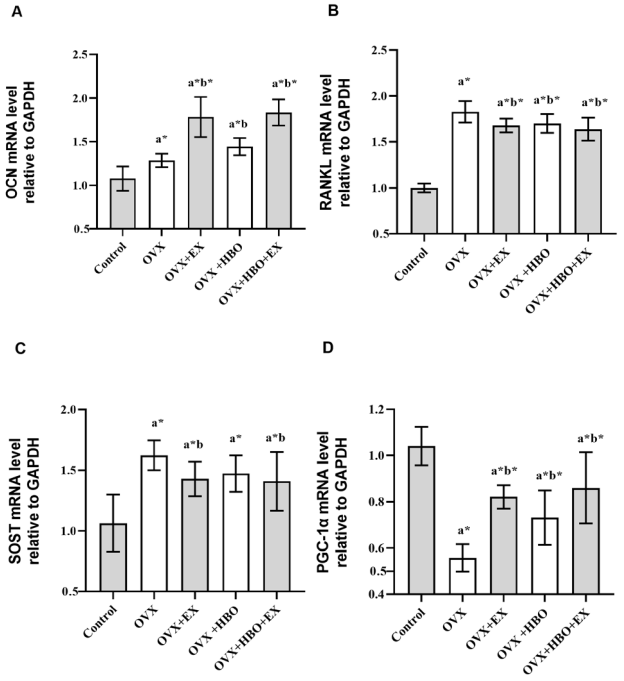


Figure 7

Relative mRNA expressions in the distal metaphyses of right femurs with bone marrow removal in OVX rats via real-time PCR analysis; A – Osteocalcin (OCN); B – receptor activator of nuclear factor kB ligand (RANKL); C – sclerostin (SOST); D – Peroxisome Proliferator-Activated Receptor γ coactivator 1α (PGC-1α); Control – sham operated control group; OVX – ovariectomy group; OVX+EX – ovariectomy with exercise treatment group; OVX+HBO – ovariectomy with hyperbaric oxygen treatment group; OVX+HBO+EX – ovariectomy with hyperbaric oxygen treatment combined with exercise group



significantly upregulated PGC-1α gene expression compared with the OVX group.

Discussion

In this study, we investigated whether HBO, EX, and a combination of HBO and EX could inhibit ovariectomy-induced bone loss and osteoporosis in rats and explored the underlying mechanisms. We found that HBO, EX and the combination treatment significantly ameliorated the deterioration of bone microarchitecture and the ovariectomy-induced bone loss in OVX rats. This inhibitory effect may be associated with the increased SOD and up-regulated PGC-1α. Exercise has been reported to inhibit bone loss after ovariectomy in rats.[15,16] Hyperbaric oxygen has also been reported to exert similar inhibitory effects on osteoporosis rats,[5–7] but in these studies, different osteoporosis models and HBO parameters were used. One study examined the effects of 'mild HBO' (134 kPa [1.32 atm abs] pressure with 40% oxygen) on unloading-induced osteoporosis rats,[5] and another group used rats with complete spinal transection as osteoporosis models with HBO delivered at 223 kPa (2.2 atm abs) with an oxygen concentration of 97–99%.[6,7] In the current study, we used 3-month-old ovariectomized rats as the postmenopausal model because these rats are reproductively mature and capable of responding appropriately to estrogen deficiency. The experimental intervention lasted for three months to establish the standard osteoporotic animal model.[17] We performed interventions at 14 days after OVX, because bone loss consistently occurred at 14 days after OVX.[43] Hyperbaric oxygen was applied at 203 kPa (2.0 atm abs) and an oxygen concentration of 85–90% because these parameters are commonly used in studying and treating clinical indications for HBO. We sampled the left femur rather than the lumbar spine for Micro-CT and histological analyses because weight-bearing bones such as tibia and femur have a higher sensitivity to treadmill exercise than the lumbar spine in rats.[18–22] Treadmill exercise was applied at 20 m·min^{-1}, 40 min·day^{-1} for 12 weeks, because previous studies have shown that moderate exercise can prevent the bone loss of the tibia and femur in ovariectomized rats.[22,23]

In our study, marked body weight gain was observed in OVX rats in the 12-week experimental period, which was consistent with the previous reports on OVX animal models.[24,25] This significant OVX-induced weight gain can be attributed to decreased energy consumption and lipid metabolism and increased fat deposition in adipose tissues in OVX animal models.[26–28] Our study also revealed that HBO, EX, and the combined intervention of HBO and EX could suppress the OVX-induced weight gain in rats. This result was in line with previous findings.[28,29] Exercise can inhibit the significant increase in body weight induced by OVX in rats. Therefore, our findings indicate that the 3-month HBO and/or EX intervention after ovariectomy may partially prevent OVX-induced weight gain by regulating energy metabolism. Furthermore, we found that endogenous estrogen production was ceased by ovariectomy, which was manifested as marked uterine atrophy in the OVX group, indicating that estrogen withdrawal was successfully

achieved in the OVX model. Comparatively, the uterine mass of OVX rats in the three intervention groups was not affected, suggesting that the interventions can inhibit bone loss without affecting the uterus after ovariectomy.

The maintenance of bone mass depends on continuous bone remodeling activity, which involves the balanced effects of osteoblastic bone formation and osteoclastic bone resorption. Yet, ovariectomy can disturb this balance, biasing the process towards bone resorption and thus leading to bone loss.[30,31] Previous *in vitro* studies reported that HBO (243 kPa, 97% O_2, 90 min, 14 HBO sessions) can accelerate human osteoblast differentiation, promote bone formation, and suppress human osteoclast formation and bone resorption in hypoxic conditions.[32,33] Another study suggests that *in vivo* HBO (243 kPa, 100% O_2, 90 min, 25 HBO sessions) suppresses osteoclast formation and bone resorption from circulating human monocytes.[34] Our study showed that osteogenesis-related gene (OCN) and serum P1NP levels were increased in the OVX group while HBO alone, EX alone and their combination increased bone formation after the 12-week intervention. Furthermore, serum CTX-I markers, the indicators of bone resorption, were significantly upregulated in OVX rats, which was consistent with the results of a previous study.[17,35] Increased expressions of osteoclastogenesis-related genes (SOST, RANKL) were also found in OVX rats. The results in the intervention groups implied that HBO alone, EX alone and their combination can significantly inhibit bone resorption. Together, our results showed that the levels of bone formation markers were significantly higher and the levels of bone resorption markers were markedly lower in the intervention groups than in the OVX group, implying that HBO alone, EX alone and their combination can enhance osteoblast genesis and inhibit osteoclast genesis in OVX rats. This was also indicated by the decreased marrow cavity, increased trabecular bone area, trabecular number, and increased Tb-N, BV/TV, and decreased Tb-Sp in intervention groups in comparison to the OVX group. The values of the combined group were superior to those of the single intervention group but without statistical difference. The above findings suggest that EX alone, HBO alone and their combination are effective in preventing ovariectomy-associated bone loss in rats, and therefore are promising alternatives for postmenopausal osteoporosis management.

Oxygen free radicals play an important role in the pathogenesis of osteoporosis by inhibiting osteoblast genesis and activating osteoclast differentiation. Therefore, the application of antioxidants might be beneficial for bone health.[36] Superoxide dismutase can scavenge oxygen free radicals and thus block the damage they cause. It has been shown that ovariectomized rats have significantly reduced estrogen and antioxidant enzyme activity, remarkably increased oxidative stress response, and thus the balance of bone metabolism is disturbed.[37] Hyperbaric oxygen has a strong antioxidant capacity and can significantly increase the activity of antioxidant enzymes in the serum.[38–41] The anti-oxidative effect of exercise can counter the aging progress in aged skeletal muscles.[42] Our study found that serum SOD level in the OVX group was significantly lower than that in the sham group ($P < 0.05$). After the 12-week intervention, the serum SOD level in the EX and combination groups was significantly increased compared to the OVX group ($P < 0.05$). PGC-1α, a master regulator of oxidative metabolism (including oxidative enzyme activity), plays a critical role in bone metabolism, and PGC-1α deficiency reduces bone mass.[5,44,45] In this present study (203 kPa, 85–90% O_2), the level of PGC-1α mRNA in the OVX group was significantly lower than that in the control group ($P < 0.01$). After a 12-week intervention, the levels in the HBO, EX and combination groups were significantly increased compared to that in the OVX group ($P < 0.05$). The results regarding SOD and PGC-1α mRNA suggest that HBO, EX and their combination can up-regulate PGC-1 α to increase SOD, improve the oxidative capacity, and thus inhibit bone loss.

Conclusions

Our findings demonstrate that HBO (203 kPa, oxygen concentration of 85–90%) and EX (20 $m \cdot min^{-1}$, 40 $min \cdot day^{-1}$, five days per week with a 5-degree slope for 12 weeks) could partially prevent bone loss and bone microarchitecture deterioration in OVX rats, and the mechanism may be associated with the increased SOD and up-regulated PGC-1α.

However, the present study failed to detect any significant difference in beneficial effects on osteoporosis between the combination treatment and monotherapy. Further investigations are needed in the future to explore the beneficial effects and underlying mechanism by the combination treatment on osteoporosis.

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How fit are military hyperbaric personnel after an asymptomatic or mild symptomatic COVID-19 infection? A retrospective study

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Abstract
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Introduction: In the diving community there is a special need to know if asymptomatic or mild COVID-19 disease impacts the cardiopulmonary functioning of individuals with occupational exposure to extreme environments. To date, no controlled studies have been conducted comparing COVID-19-infected hyperbaric employees and non-COVID-19-infected peers in a military setting.
Methods: Between June 2020 and June 2021, healthy, hyperbaric, military personnel aged between 18 and 54 years old, who had recovered from asymptomatic or subclinical COVID-19 disease at least one month earlier, were analysed. Non-COVID-infected peers with medical assessments during the same period were used as the control group. Somatometry, spirometry, VO_2 max, and DLCO were measured for each group.
Results: No clinically relevant differences in somatometry, lung function tests, and exercise testing were found between the COVID-19 group and the controls. However, the percentage of individuals with a decrease in estimated VO_2-max of 10% or more was significantly greater in the COVID group than in the control group (24 vs. 7.8%, $P = 0.004$).
Conclusions: After asymptomatic or mild symptomatic COVID-19 infections, military hyperbaric employees are as fit as those who had not encountered COVID-19. As this research was based on a military population, it cannot be extrapolated to a nonmilitary population. Further studies in nonmilitary populations are necessary to determine the medical relevance of the present findings.

Introduction

Since December 2019, the world has faced the spread of the SARS-CoV-2 virus, which causes the coronavirus disease known as COVID-19.[1] Coronaviruses infect epithelial cells throughout the respiratory tract. The majority of patients recover at home with only minor symptoms.[2] Histological research indicates that in patients hospitalised with COVID-19, infection can result in different types of pulmonary damage.[3] Whether this damage is permanent is still a point of debate. High-resolution computed tomography (HRCT) scans three months after discharge still showed radiological evidence of COVID-19 in 78% of severe cases, declining to 48% after six months.[4] Studies have demonstrated decreases in predicted diffusing capacity for carbon monoxide (DLCO), restrictive ventilatory defects, and small airway dysfunction in COVID-19 patients.[5]

Little is known about the extent to which these pathophysiological changes and changes in physiological pulmonary parameters also occur in asymptomatic or subclinical COVID-19 patients. One study reports a reduction in diffusion capacity of carbon monoxide (DLCO) as the main effect,[5] whereas other studies suggest radiological evidence of COVID-19 in asymptomatic and mild disease.[6–9] Furthermore, several studies relating to non-severe COVID-19 found a maximal oxygen uptake (VO_2-max) lower than expected after illness,[10–12] although others report no impairment.[13]

Multiple guidelines for the medical assessment of post-COVID-19 divers have been developed because of the major concern in the diving industry about the risk of COVID-19-induced pulmonary barotrauma (PBt).[14–16] Pathological predispositions to PBt include bullae and blebs.[17–19] Although there have been no published cases of divers

with PBt following COVID-19, numerous case reports have linked COVID-19 to bullae-associated pneumothorax,[20–23] even in mild cases of COVID-19.[24] Based upon the latter, and following DMAC 33 regulations,[15] a tailormade post-COVID-19 dive medical assessment of divers was implemented at the Royal Netherlands Navy Diving and Submarine Medical Centre (DMC).

Since many previous studies investigating the effect of mild COVID-19 on pulmonary function did not include baseline function data, it was not possible to ascertain whether abnormalities found were pre-existing or due to COVID-19. Consequently, there is still uncertainty regarding the long-term effects of COVID-19 on pulmonary function in both military divers and the general public.

The DMC has a large (historical) database of medical assessments relating to different diving- and hyperbaric-related occupations. This database was used to provide matched, non-COVID-19-infected individuals to act as the control group. This retrospective, cross-sectional study aimed to investigate whether changes in cardiopulmonary function occur in hyperbaric personnel after mild and asymptomatic COVID-19.

Methods

According to national law and legislation, retrospective analyses are not required to be evaluated by a medical ethics committee. Nevertheless, this study has been evaluated and granted permission by the Surgeon General of the Netherlands Armed Forces (reference number DOSCO2020036245). Furthermore, the methods used to handle personal details and privacy were in agreement with the guidelines of the Association of Universities in the Netherlands and the Declaration of Helsinki.

Medical assessments of military hyperbaric personnel, i.e., divers, chamber attendants, and submariners, of the Netherlands Armed Forces are performed annually at the DMC. All fitness-to-dive assessments are performed according to European Diving Technology Committee (EDTC) guidelines,[25] except that pulmonary function testing (PFT) results are interpreted relative to the Global Lung Initiative (GLI)-2012 reference set.[26] In June 2020, a post-COVID-19 dive medical assessment, adapted from the DMAC-33 guideline,[15] was adopted by DMC. In this study, we use the term 'hyperbaric' which refers to divers, chamber attendants and submariners.

SUBJECTS

The data used in this study were from participants who underwent a post-COVID-19 dive medical assessment for hyperbaric work in the period from June 2020 to June 2021. The data of subjects who tested positive, using either the reverse transcriptase-polymerase chain reaction test (RT-PCR) or serology for SARS-CoV-2, but were neither hypoxic nor hospitalised were included. Thus, only asymptomatic and mildly symptomatic patients were included. Furthermore, active disease or the absence of a previous record of relevant testing were also reasons for exclusion.

The intention was to couple two to three age-, sex-, and occupation-matched controls, who had never tested positive for COVID-19 but who had undergone their last medical in the same period as the COVID-19 group, to every person included in the COVID-19 group. This ensured that the control group had experienced similar disadvantages (e.g., closure of sporting facilities and working at home) as the COVID-19-positive group during the COVID-19 lockdown periods. In terms of age, a one year difference in birth year was accepted (e.g., someone born in 1990 could be coupled to someone born in 1989, 1990, or 1991).

DIVE MEDICAL ASSESSMENT

Regular annual dive medical assessments typically consist of somatometry, spirometry, and exercise testing, along with an interview and physical examination by the dive medical officer, as specified by EDTC guidelines. Post-COVID-19 dive medical assessment additionally included measurements of diffusing capacity and plethysmography. All personnel who came for a COVID-19 dive medical assessment were asked if their results could, anonymously, be used for research purposes. Only the results of those who agreed were included in this study database.

Spirometry, plethysmography, and diffusion capacity

Forced expiratory volume in one second (FEV_1), forced vital capacity (FVC), and DL_{CO} were measured on MS-PFT PRO (Vyaire, the Netherlands). All measurements were carried out by qualified respiratory technicians according to the European Respiratory Society / American Thoracic Society task force guidelines.[27,28] For DLCO, the single-breath method was used and is reported as DLCO corrected for haemoglobin. The predicted FEV_1 and FVC were calculated using the GLI reference values of 2012.[29] Residual volume (RV), vital capacity (VC), and total lung capacity (TLC) plethysmography measurements were carried out in a Vyaire Vyntus Body box (Vyaire, the Netherlands). For plethysmography, the current GLI reference values of 2021 were used.[30] For DL_{CO} predicted values, the GLI 2017 reference values were used.[27] Calibration was performed following the manufacturer's instructions.

Exercise testing

Maximal exercise capacity tests were performed with an Excalibur Sport ergometer (Lode, the Netherlands) based on a weight-dependent protocol in which the test starts with a power of 1 watt·kg^{-1} body weight during the first minute, with incremental increases of 0.5 watt·kg^{-1} body weight and 0.25 watt·kg^{-1} body weight in the second and

third minutes, respectively, until exhaustion. The load was raised once per minute. All testing was done as advised in the American Thoracic Society protocol.[31] In this paper, the term estimated VO_2-max (eVO_2-max) will be used to express the calculated VO_2-max. All electrocardiographs (ECGs) were taken with a Vyaire Vyntus ECG (Vyaire, the Netherlands). Due to COVID-19 regulations, it was not possible to use breath analysis for testing carried out at the beginning of the pandemic. Thus, it was decided to calculate VO_2-max using the formula below developed by the DMC where W_{MAX} = maximal load (Watts), A = age (years), W = weight (kg).

$$Male: VO_2max\ (ml\ O_2.kg^{-1}.min^{-1}) = \frac{(10.00 * Wmax) - (7.71 * A) + 738.77}{W}$$

$$Female: VO_2max\ (ml\ O_2.kg^{-1}.min^{-1}) = \frac{(10.78 * Wmax) + 175.76}{W}$$

STATISTICAL ANALYSIS

As only a handful of studies have been published with subclinical COVID-19 patients, the sample size estimation was rather difficult. The threshold for the COVID-19 group was set at a minimum of 50 subjects since the sample size of other studies at the time of data gathering was at least 33.[5]

Analysis of data was blinded and performed with SPSS Statistics 23 (IBM Corporation), VassarStats (http://vassarstats.net) and SISA (https://www.quantitativeskills.com/sisa/). The latter two were used for their Freeman-Halton extension of Fisher's exact test. All data were first tested for normality using the Shapiro-Wilk test. Continuous variables were compared using an independent or two-tailed-sample, *t*-test, or a rank-sum test in the case of nonparametric data. Finally, a linear regression analysis was performed to test whether changes were time-dependent. A probability (*P*) value of less than 0.05 was considered significant.

Results

Fifty-one post-COVID-19 subjects and 125 healthy controls were included in this study. The baseline characteristics of the post-COVID-19 and control groups were not statistically different (Table 1); however, those in the post-COVID-19 group had their latest dive medical assessment significantly later than those in the control group (1.5 y [IQR 1.75 y] vs 1 y [IQR 1 y], respectively; $P = 0.023$). On average, the post-COVID-19 group had their post-COVID-19 dive medical assessment 1 month (IQR 2.0 months) after their positive COVID-19 test.

SOMATOMETRY

The post-COVID-19 group had a significantly higher body fat percentage (Δfat percentage 2.1% [SD 3.1]) than the control group (0.6% [2.4]; $P = 0.002$). However, the skinfold thickness measurement method has an error of up to 3.5% for women and up to 5% for men.[32]

LUNG FUNCTION TESTING

None of the pre-post parameters for diffusion capacity and plethysmography varied significantly between the COVID-19 and control groups. This was also the case for spirometry except for the change in FEV_1 (ΔFEV_1), which had a significantly greater decline in the control group than in the post-COVID-19 group (Tables 2 and 3). However, this difference in ΔFEV_1 falls within the coefficient of variation.[33] Finally, there was no correlation between DLCO % of predicted or change in DLCO % of predicted (ΔDL_{CO}) and either age or months after illness.

EXERCISE TESTING

The absolute value of eVO_2-max did not differ significantly between the two groups. By contrast, compared with baseline, there was a significant decline in eVO_2-max (ΔeVO_2-max) in the post-COVID-19 group (-0.99 $mL{\cdot}kg^{-1}{\cdot}min^{-1}$, IQR 5.93) when compared to the control group (+0.13 $mL{\cdot}kg^{-1}{\cdot}min^{-1}$, IQR 3.12; $P = 0.020$). This was also the case for the percentage change (post-COVID-19: -2.4%, IQR 13.9; control: +0.3%, IQR 3.12; $P = 0.028$). Although these differences are statistically significant, they are nevertheless small in absolute terms. Interestingly, the proportion of subjects with a decrease of more than 10% in eVO_2-max was significantly higher in the post-COVID-19 group than in the control group (24% vs 7.8%, $P = 0.004$). It is important to mention that although 24% of our COVID-19 positives had a decrement of more than 10% in eVO_2-max, all were still fit enough to pass their assessment for hyperbaric work. All other categories and parameters were statistically equivalent between the groups. Finally, there was no correlation between ΔeVO_2-max and the time elapsed since COVID-19 infection (Figure 1).

Discussion

The present study showed no clinically relevant differences in somatometry, lung function, or exercise testing between a post-COVID-19 group and a previously uninfected control group. However, the number of subjects with more than a 10% reduction in their eVO_2-max relative to their baseline data was significantly higher in the post-COVID-19-group than in the control group.

To the best of our knowledge, this is the first study to compare the pre-post effect of COVID-19 on lung function and exercise testing in hyperbaric workers previously infected with COVID-19 and in a previously uninfected control group. By contrast, several studies on the effect of COVID-19 on lung function parameters in patients or athletes have been published. Pre- and post-COVID spirometry data showed no significant difference in FEV_1 or FVC between COVID-19 and uninfected controls.[12,13,34,35] Indeed, in a previous review paper, it was stated that spirometry indices appear to be well preserved.[5] Thus, the results of our study align with these previous findings.

Table 1

Main baseline characteristics of the two study groups; data are mean (SD) for normally divided parameters and median (IQR, μ = mean) for non-normal parameters; DLCO – diffusing capacity for carbon monoxide; FEV_1 – forced expiratory volume in one second; FVC – forced vital capacity; HRmax – maximum heart rate; IQR – interquartile range; SD – standard deviation; TLC – total lung capacity; VC – vital capacity; eVO_2max – estimated maximum rate of oxygen consumption

Parameter	**COVID+ (*n* = 51)**	**CONTROL (*n* = 125)**	***P***
Age	30.00 (IQR 8, μ = 31.78)	31.00 (IQR 11, μ = 32.66)	0.497
Male Female	49 (96.1%) 2 (3.9%)	123 (98.4%) 2 (1.6%)	0.581
Height (cm)	183 (SD 7.6)	184 (SD 6.8)	0.726
Weight (kg)	86.0 (SD 12.5)	86.6 (SD 11.5)	0.767
Body mass index ($kg \cdot m^{-2}$)	25.4 (SD 2.4)	25.6 (SD 2.7)	0.962
Fat percentage *n*	15.8 (IQR 5.5, μ = 16.4) 48	15.8 (IQR 6.2, μ = 16.5) 111	0.754
Occupation *Diver* *Submariner* *Special Forces* *Other*	 32 (62.7%) 10 (19.6%) 7 (13.7%) 2 (3.9%)	 77 (61.6%) 27 (21.6%) 19 (15.2%) 2 (1.6%)	0.804
Smoking status *Never* *Active* *Former*	 32 (62.7%) 10 (19.6%) 9 (17.6%)	 71 (56.8%) 22 (17.6%) 32 (25.6%)	0.527
Smoking pack years *n*	3.5 (IQR 6.45, μ = 4.19) 19	4.05 (IQR 5.63, μ = 5.33) 54	0.330
FEV_1 (L) FEV_1 Z-score *n*	4.63 (SD 0.58) -0.14 (SD 0.78) 51	4.74 (SD 0.61) 0.0 (SD 0.80) 123	0.316 0.275
FVC (L) FVC Z-score *n*	5.96 (SD 0.82) 0.25 (SD 0.82) 51	6.01 (SD 0.76) 0.27 (SD 0.83) 123	0.679 0.841
FEV_1/FVC FEV_1/FVC Z-score *n*	77.78 (SD 4.48) -0.65 (SD 0.64) 51	78.90 (SD 4.55) -0.48 (SD 0.68) 123	0.141 0.118
RV (L) *n*	1.65 (SD 0.48) 27	1.53 (SD 0.31) 21	0.314
VCmax (L) *n*	6.17 (SD 0.77) 30	6.51 (SD 0.83) 21	0.138
TLC (L) *n* TLC % of predicted *n*	7.90 (SD 1.09) 27 105.5 (IQR 14.6, μ = 104.9) 26	8.03 (SD 1.00) 21 104.6 (IQR 17.0, μ = 105.0) 21	0.666 0.856
DLCO ($mmol \cdot min^{-1} \cdot kPa^{-1}$) DLCO % of predicted *n*	11.67 (SD 1.90) 101.7 (SD 14.4) 32	12.13 (SD 1.68) 102.3 (SD 8.7) 33	0.306 0.838
Wmax (watt) *n*	324 (SD 50) 50	324 (SD 45) 122	0.920
eVO_2-max ($ml \cdot kg^{-1} \cdot min^{-1}$) *n*	43.69 (IQR 6.52, μ = 43.78) 50	42.68 (IQR 6.63, μ = 43.10) 122	0.284
HRmax ($beats \cdot min^{-1}$) *n*	186 (IQR 20, μ = 184) 50	185 (IQR 11, μ = 186) 122	0.550

Table 2

Main post-measurement characteristics of the two study groups; data are mean (SD) for normally divided parameters and median (IQR, μ = mean) for non-normal parameters; DLCO – diffusing capacity for carbon monoxide; FEV_1 – forced expiratory volume in one second; FVC – forced vital capacity; HRmax – maximum heart rate; IQR – interquartile range; SD – standard deviation; TLC – total lung capacity; VC – vital capacity; eVO_2max – estimated maximum rate of oxygen consumption

Parameter	COVID+ (*n* = 51)	CONTROL (*n* = 125)	*P*
Age	31.0 (IQR 9, μ = 33.9)	32.0 (IQR 10, μ = 34.5)	0.609
Years since previous medical *n*	1.50 (IQR 1.75, μ = 2.17) 51	1.00 (IQR 1.00, μ = 1.69) 124	0.023
Months since infection	1.0 (IQR 2.0, μ = 2.5)	N/A	N/A
Weight (kg)	87.4 (SD 12.3)	86.6 (SD 11.6)	0.773
Body mass index ($kg \cdot m^{-2}$)	26.3 (IQR 3.4, μ = 25.9)	25.4 (IQR 3.4, μ = 25.6)	0.340
Fat percentage *n* Δ Fat percentage *n*	18.4 (IQR 4.4, μ = 18.0) 47 2.1 (SD 3.1) 46	16.8 (IQR 6.2, μ = 17.5) 117 0.6 (SD 2.4) 105	0.165 **0.002**
FEV_1 (L) *n* FEV_1 Z-score *n* ΔFEV_1 (L) *n*	4.62 (SD 0.57) 50 -0.04 (SD 0.81) 50 -0.01 (SD 0.31) 50	4.62 (SD 0.57) 123 -0.13 (SD 0.74) 123 -0.12 (SD 0.21) 121	0.953 0.476 **0.023**
FVC (L) *n* FVC Z-score *n*	5.94 (SD 0.75) 50 0.31 (SD 0.78) 50	5.92 (SD 0.72) 123 0.17 (SD 0.80) 123	0.898 0.320
FEV_1/FVC *n* FEV_1/FVC Z-score *n*	77.76 (SD 5.67) 50 -0.58 (SD 0.82) 50	78.25 (SD 5.17) 123 -0.51 (SD 0.76) 123	0.583 0.619
RV (L) *n*	1.59 (SD 0.39) 50	1.56 (SD 0.45) 13	0.815
VCmax (L) *n*	6.04 (SD 0.77) 50	6.08 (SD 1.00) 13	0.891
TLC (L) *n*	7.62 (SD 0.95) 50	7.63 (SD 1.24) 13	0.959
DLCO ($mmol \cdot min^{-1} \cdot kPa^{-1}$) *n*	11.26 (SD 1.69) 50	11.70 (SD 2.66) 18	0.519
Wmax (Watt) *n*	320 (SD 53) 51	328 (SD 44) 119	0.325
eVO_2-max ($ml \cdot kg^{-1} \cdot min^{-1}$) *n* Δ eVO_2-max ($ml \cdot kg^{-1} \cdot min^{-1}$) *n* % change eVO_2-max Categorial % change eVO_2-max *>-10%* *-10 to 0%* *0 to 10%* *>10%*	42.41 (IQR 4.88, μ = 42.23) 46 -0.99 (IQR 5.93, μ = -1.57) 50 -2.4% (IQR 13.9, μ = -3.0) 12 (24%) 18 (36%) 17 (34%) 3 (6%)	42.20 (IQR 6.74, μ = 43.35) 118 0.13 (IQR 3.12, μ = 0.22) 115 0.3% (IQR 7.3, μ = 0.59) 9 (7.8%) 39 (33.9%) 58 (50.4%) 9 (7.8%)	0.435 **0.020** **0.028** **0.004** 0.795 0.053 0.683
HRmax ($beats \cdot min^{-1}$) *n*	185 (IQR 16, μ = 183) 51	184 (IQR 11, μ = 183) 118	0.742

Table 3
Before and after spirometric parameters for COVID-19+ subjects; data are mean (SD); FEV_1 – forced expiratory volume in one second; FVC – forced vital capacity; TLC – total lung capacity

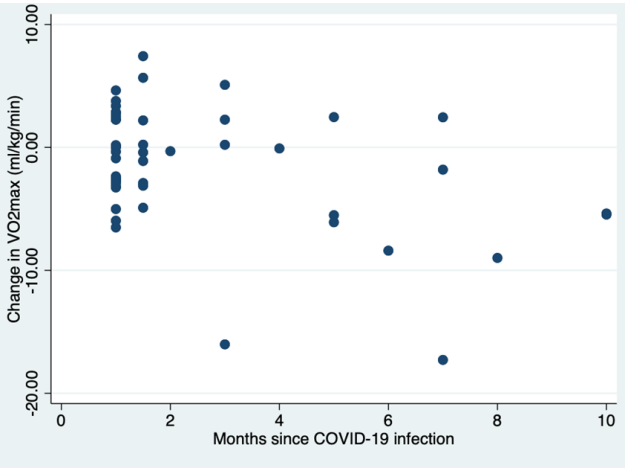
Parameter	COVID +		*P*
	Pre	Post	
FEV_1 (n = 50)	4.63 (0.59)	4.62 (0.57)	0.852
FVC (n = 50)	5.94 (0.81)	5.94 (0.75)	0.298
TLC (n = 26)	7.80 (0.96)	7.85 (0.83)	0.358

Consistent with the spirometry data, we did not find any indication of restriction or alteration in TLC after mild or asymptomatic COVID-19. This is in contrast to recent studies that found restriction and decrease in TLC as the most common abnormalities.[5] However, the subjects in those studies were severe, hospitalised patients, whereas our subjects consisted of asymptomatic or only mild symptomatic COVID-19 patients, which suggests that mild COVID-19 does not generate any restriction or changes in TLC. This has been supported by studies that suggest that restriction worsens with increasing severity of COVID-19.[5]

In the present study, DL_{CO} was unaffected by COVID-19 infection, which is comparable to those of some previous studies.[36,37] Although this seems to contradict the common finding that DL_{CO} is decreased in COVID-19 patients one has to keep in mind that the percentage of DL_{CO} predicted (%DL_{CO} pred) was unaffected in patients with mild illness but was lower in patients with severe illness.[37] Furthermore, in most studies, the mean age of subjects was higher than that of the subjects in our study, which could have influenced DL_{CO} values since increased age is associated with a graver course of COVID-19.[38] In addition, most studies were conducted at the time of patient discharge or within 30 days after discharge. It is possible that DL_{CO} improves most in the months immediately after COVID-19 infection.[39,40] Our study does not support this as we found no linearity or statistical significance in %DL_{CO} pred or difference between individual pre-COVID-19 and post-COVID-19 values, for any period of time after infection or for any age.

In the present study, we did not find a significant difference in eVO_2-max between the COVID-19 population and the control group. This is in line with a recent study that found that mild or asymptomatic COVID-19 does not influence average VO_2-max.[13] One can hypothesize that the mild course of the disease in asymptomatic or mild symptomatic patients may have enabled them to restart training faster and recuperate more quickly from the detraining effect of COVID-19 illness than severe symptomatic patients. Indeed, a previous study reported that post-COVID-19 subjects who received six weeks of rehabilitation improved their six-minute walking distance (6MWD) by 50 metres, while no improvement was observed for the group who did not undergo rehabilitation, which supports the detraining hypothesis.[40]

Figure 1
Correlation between ΔVO_2-max (ml $O_2 \cdot kg^{-1} \cdot min^{-1}$) and months since infection with COVID-19 in the COVID+ group



Recently, it was reported that the percentage of recruits who had a decline of more than 10% in VO_2-max was significantly higher in a COVID-19 symptomatic group than in either COVID-19 asymptomatic or noninfected groups (18.8% vs 1.9% and 0%).[11] The reverse applied to recruits with an increase of more than 10% in their VO2-max; while none of the COVID-19 symptomatic recruits had an increase of more than 10%, the noninfected and asymptomatic groups had increases of 13.9% and 7.6%, respectively. Similar results were obtained in the present study; while 24% of the COVID-19-positive group had a decreased eVO_2-max of more than 10%, only 7.8% of the COVID-19-negative group had a decreased eVO2-max of 10% or more. By contrast, the proportion of individuals with an eVO_2-max increase of more than 10% was not statistically significantly different between the two groups. We suggest that the non-significant increase in VO_2-max could be because subjects did not engage in sports on a daily basis, which is in contrast to military recruits who perform basic military training regularly.[41]

Based on the above, we hypothesize that the significant increase in the proportion of hyperbaric personnel who showed a > 10% decrease in eVO_2-max in the COVID-19 group in this study was not due to COVID-19 directly but to a detraining effect. Dive medical assessments in the future are needed to test this hypothesis. Yet, more importantly, the present results underline the importance to use exercise capacity as a parameter for returning to hyperbaric work as has been suggested by others.[42]

The strengths of this study include its study design, sample size, the inclusion of only asymptomatic or mild

symptomatic hyperbaric personnel, the use of a control group, and practical relevance for professional hyperbaric work. Nevertheless, the study has some limitations.

First, our subjects were stratified based on the result of their PCR test. Asymptomatic subjects were only included in the COVID-19 group if they had undergone a PCR test for other purposes (e.g., travel or part of contact tracing research) and had tested positive. Asymptomatically infected individuals who had not undergone a PCR test could have been erroneously assigned to the control group. As we do not know to what extent this occurred, it is possible that this oversight could have biased the results of the control group. For the purpose of this study, we had to rely on the honesty of divers to report any illnesses and the results of their PCR tests.

Second, for some outcome measures, data were unavailable for subjects in the control group because not all measurements are standardly performed at every regular dive medical assessment. For example, data for only a small group of control subjects were available for determining the pre-post values of both DL_{CO} and plethysmography.

Third, it should be considered that our data gathering was performed during the period when the Alpha and Delta SARS-Cov-2 variants were most prevalent, and most people were not vaccinated. Vaccination status and virus variants could influence clinical outcomes and thus lung function outcomes. A follow-up study with enhanced knowledge of patient infection and vaccination status may shed light on this limitation.

Finally, the vast majority of our study population was male, with only two females in the COVID-19 group and two female matched-controls. We do not think this biased our results as it was reported that age, but not gender, had an impact on the recovery of mild-to-moderate COVID-19 patients.[43] However, to investigate any gender influence, a more balanced male-female population would be needed in future studies.

Conclusion

The present study is the first to measure pre-post changes, relating to lung function and exercise testing, in asymptomatic or mild symptomatic COVID-19 hyperbaric personnel and a matched control group. Based on the results of this study, we concluded that there are no negative effects on either lung function or exercise testing due to asymptomatic or mild symptomatic COVID-19 infection. Since our study population primarily comprised male, hyperbaric employees who were in good physical condition before being infected by COVID-19, this conclusion cannot be generalised to the whole hyperbaric population. Further studies with different populations will be necessary to determine the dive medical relevance of the present findings.

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Technical report

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

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

Abstract
(Wang C, Xue L, Yu Q, Liu Y, Ren Z, Liu Y. Evaluation of a new hyperbaric oxygen ventilator during volume-controlled ventilation. Diving and Hyperbaric Medicine. 2023 June 30;53(2):129–137. doi: 10.28920/dhm53.2.129-137. PMID: 37365130.)
Introduction: The performance of the Shangrila590 hyperbaric ventilator (Beijing Aeonmed Company, Beijing, China) was evaluated during volume-controlled ventilation.
Methods: Experiments were conducted in a multiplace hyperbaric chamber at 101, 152, 203, and 284 kPa (1.0, 1.5, 2.0 and 2.8 atmospheres absolute [atm abs]). With the ventilator in volume control ventilation (VCV) mode and connected to a test lung, comparison was made of the set tidal volume (VTset) versus delivered tidal volume (VT) and minute volume (MV) at VTset between 400 and 1,000 mL. Peak inspiratory pressure was also recorded. All measurements were made across 20 respiratory cycles.
Results: Across all ambient pressures and ventilator settings the difference between VTset and actual VT and between predicted MV and actual MV were small and clinically insignificant despite reaching statistical significance. Predictably, Ppeak increased at higher ambient pressures. With VTset 1,000 mL at 2.8 atm abs the ventilator produced significantly greater VT, MV and Ppeak.
Conclusions: This new ventilator designed for use in hyperbaric environments performs well. It provides relatively stable VT and MV during VCV with VTset from 400 mL to 800 mL at ambient pressures from 1.0 to 2.8 atm abs, as well as VTset 1,000 mL at ambient pressures from 1.0 to 2.0 atm abs.

Introduction

Hyperbaric oxygen treatment (HBOT) involves administration of 100% inspired oxygen at elevated ambient pressure. It is widely used in disorders such as acute carbon monoxide poisoning, decompression sickness, and arterial gas embolism which occasionally require intensive care.[1,2] It is a safe intervention within the common treatment pressure range 203–284 kPa (2–2.8 atmospheres absolute [atm abs]).[3] In a normobaric environment, the arterial partial pressure of oxygen (PaO_2) can only be raised by increasing the fraction of inspired oxygen (FiO_2) in a limited manner.[4] In a hyperbaric environment, the PaO_2 can be further enhanced by increasing ambient pressure and FiO_2.

Administering HBOT in ventilated intensive care unit (ICU) patients can be challenging because ordinary ICU ventilators may not work well at increased ambient pressures. Indeed, many medical devices cannot be used in hyperbaric chambers including life support technologies such as haemofiltration, electrical defibrillators and extracorporeal membrane oxygenation systems.[5,6] In recent years, a series of bench tests have been carried out on ventilators under hyperbaric conditions during basic ventilation modes, volume-controlled ventilation (VCV) and pressure-controlled ventilation (PCV).[7,8]

Pneumatical ventilators can operate safely in hyperbaric environments, but they cannot provide stable tidal volume (VT), respiratory rate (f) or minute volume (MV) without considerable user intervention.[9,10] Similarly, most electro-pneumatical ventilators cannot function well in hyperbaric chambers. In the early stage, researchers focused on empirically predicting changes in ventilation parameters under high pressures and then adjusted the parameters of the ventilator to manually compensate for the changes. With improved understanding of respiratory mechanics in hyperbaric environments, HBOT ventilators have been developed. These ventilators can automatically adjust performance when the ambient pressure changes, for example, the Siaretron 1000 Iper (Bologna, Italy).[10,11] However, these devices are expensive and not widely available in China. We tested a locally developed HBOT

ventilator to evaluate the stability of VT and MV during VCV in a hyperbaric chamber.

Methods

ETHICS APPROVAL

This study did not involve human participants, human material, or human data so ethical approval was not required.

THE VENTILATOR

The Shangrila590 ventilator is an electropneumatic ventilator from Beijing Aeonmed Company that is commonly used in the ICU in China. To comply with the safety regulations of medical hyperbaric chambers in China, the pneumatic part was placed in the chamber, and the electronic part was positioned outside the chamber (Figure 1A and B).[12,13] The two parts of the ventilator were connected through a penetrator in the chamber bulkhead, allowing doctors to operate the ventilator from outside the hyperbaric chamber. Ventilator engineers improved the control algorithm to make the ventilator work reliably and safely in a hyperbaric environment.

THE TEST LUNG

We used a Michigan Instruments PneuView®3 System (Grand Rapids, MI, USA) to measure the ventilation parameters. The detection system comprised a test lung and PneuView®3.3 software; the latter processed the test lung data which was recorded electronically.

THE CRITICAL CARE MULTIPLACE HYPERBARIC CHAMBER

Multiplace hyperbaric chambers are generally better suited for HBOT in critically ill patients than monoplace hyperbaric chambers.[14] The critical care hyperbaric chamber (GY3800-A / GY3800 M2-D) (Yantai Hongyuan Oxygen Industrial Inc., Yantai, China) is a multiplace hyperbaric chamber with an automated operation system equipped with electrocardiogram monitors, ventilators, transcutaneous oxygen and carbon dioxide tension monitors, syringe drivers, and infusion pumps to ensure continuity of treatment for ICU patients. The chambers have three compartments; two ICU chambers and a prechamber between them. These have the capacity for 24 seated people or eight gurneys.

EXPERIMENTAL CONFIGURATION

We calibrated the ventilator and the test lung at atmospheric pressure before the experiments. The test lung was located inside the hyperbaric chamber and connected to the pneumatic component of the ventilator. The digital data detected by the test lung and the ventilator were passed by penetration wires through the bulkhead to a personal computer and the electronic component of the ventilator outside the chamber (Figure 2). The ventilator was adjusted by doctors outside the chamber, and the resistance and compliance of the test lung were regulated by staff inside the chamber (Table 1). Respiratory resistance includes lung compliance and airway resistance, which needs to be matched with tidal volume to ensure safe airway pressure. Under normal physiological conditions and positive-pressure ventilation, higher compliance and lower resistance may produce larger tidal volumes, and result in stable airway pressure. So, in this study, compliance and airway resistance of the test lung were set differently between VTset

Figure 1
A – ventilator electronic component external to the chamber; B – ventilator pneumatic component inside the chamber

Figure 2
Schematic of the experimental configuration

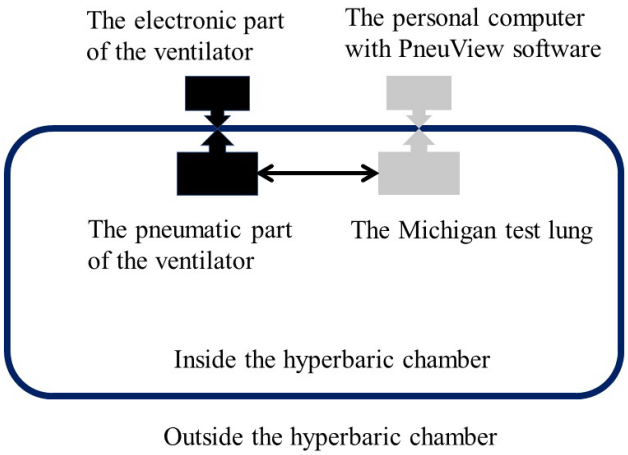


Table 1
Experimental settings for the ventilator and the test lung during volume- controlled ventilation at different ambient pressures

Ventilator settings	Volume-controlled ventilation (VCV), Respiratory rate (f) = 20 breaths per minute (BPM), Inspiratory/expiratory ratio (I/E) = 1:2, Positive end-expiratory pressure (PEEP) = 0.2 kPa, Fraction of inspired oxygen (FiO_2) = 40 %				
Ventilator VTset (mL)	400	500	600	800	1,000
Test lung compliance ($mL \cdot kPa^{-1}$)	200	200	200	500	500
Test lung resistance ($kPa \cdot L^{-1} \cdot s^{-1}$)	2	2	2	0.5	0.5

Figure 3
A – changes in tidal volume (VT) during volume-controlled ventilation (VCV) with preset tidal volume (VTset) 400–600 mL at different ambient pressures; B – changes in tidal volume (VT) during volume-controlled ventilation (VCV) with preset tidal volume (VTset) 800–1,000 mL at different ambient pressures

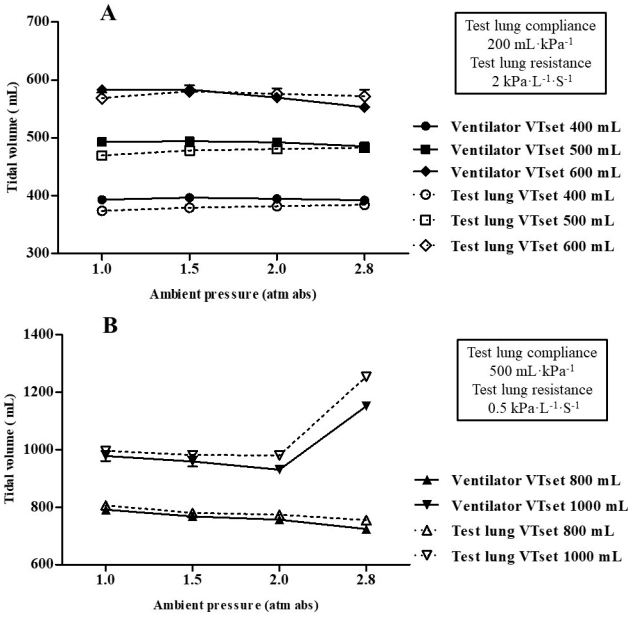
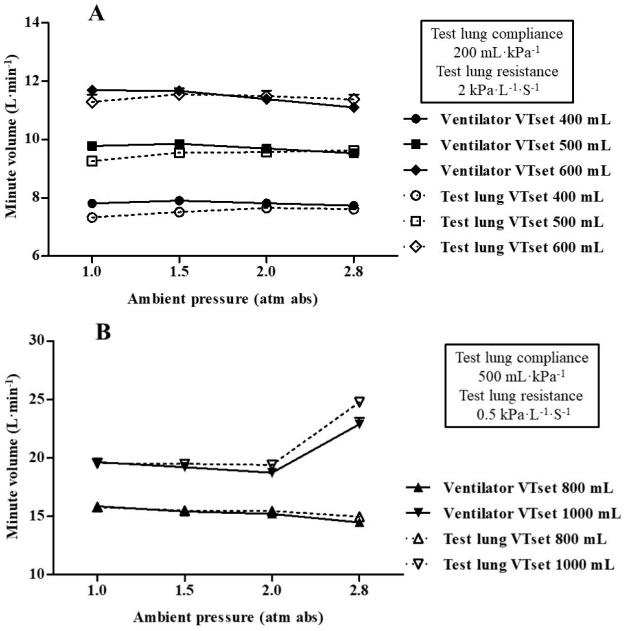


Figure 4
A – changes in minute volume (MV) during volume-controlled ventilation (VCV) with preset tidal volume (VTset) 400–600 mL at different ambient pressures; B – changes in minute volume (MV) during volume-controlled ventilation (VCV) with preset tidal volume (VTset) 800–1,000 mL at different ambient pressures



400–600 mL and VTset 800–1,000 mL according to the calibration specification in for ventilators in China.[15,16]

EXPERIMENTAL PROCEDURE

The hyperbaric chamber ambient pressure was sequentially increased from 101 kPa to 152, 203, and 284 kPa (from 1.0 to 1.5, 2.0 and 2.8 atm abs) with testing occurring at these different ambient pressures. At every pressure stage, the ventilator was operated in VCV mode at different preset tidal volumes (VTset) (400, 500, 600, 800 and 1,000 mL) and the following parameters; 20 breaths per minute (BPM), inspiratory/expiratory (I:E) ratio 1:2, positive end-expiratory pressure (PEEP) 0.2 kPa, and FiO_2 40%. The corresponding resistance and compliance of the test lung is provided in Table 1. The steady state of the ventilator after regulation was two minutes. The VT, MV, peak inspiratory pressure (Ppeak) and PEEP values were collected by the ventilator and the test lung for 20 respiratory cycles at every ambient pressure and VTset. Static lung compliance (Cs) and airway resistance (Raw) were measured by the ventilator. The temperature in the hyperbaric chamber was maintained at 24°C to 26°C.

STATISTICAL ANALYSIS

For the five values of VTset multiple factor analysis of variance was used to evaluate VT, MV, Ppeak and PEEP. The effects of the four ambient pressures and two test methods on

Table 2

Tidal volume (VT) during volume-controlled ventilation (VCV) at different ambient pressures, data are mean (standard deviation); $^{*}P < 0.05$, ventilator vs. test lung; $^{a}P < 0.05$ vs. 1.0 atm abs group, $^{b}P < 0.05$ vs. 1.5 atm abs group, $^{c}P < 0.05$ vs. 2.0 atm abs group

VTset (mL)	Equipment	VT (mL)			
		1.0 atm abs	1.5 atm abs	2.0 atm abs	2.8 atm abs
400	Ventilator	393.2 (2.4)	396.8 (2.3)^{a}	394.8 (2.3)^{a}	392.3 (3.0)^{a}
	Test lung	373.8 (3.3)*	379.3 (4.4)^{*a}	381.7 (6.1)^{*a}	384.4 (6.8)^{*a}
500	Ventilator	493.1 (3.2)	494.3 (4.7)^{a}	492.3 (2.6)^{a}	485.3 (3.8)
	Test lung	469.4 (6.0)*	478.2 (7.2)^{*a}	480.7 (6.0)^{*a}	482.9 (5.5)*
600	Ventilator	583.3 (9.2)	583.7 (8.6)	569.6 (7.8)^{b}	553.1 (7.3)^{abc}
	Test lung	568.6 (9.5)	580.5 (10.0)	576.0 (9.3)^{b}	571.9 (11.3)^{abc}
800	Ventilator	791.8 (12.9)	768.3 (12.8)^{a}	756.7 (12.5)^{ab}	724.1 (11.0)^{abc}
	Test lung	806.1 (9.2)*	780.3 (11.2)^{*a}	773.6 (9.6)^{*ab}	754.6 (11.6)^{*abc}
1,000	Ventilator	978.2 (17.3)	959.2 (17.0)^{a}	931.0 (12.9)^{ab}	1,152.0 (8.9)^{abc}
	Test lung	996.4 (13.7)*	981.6 (12.9)^{*a}	980.2 (11.8)^{*ab}	1,254.0 (7.5)^{*abc}

Table 3

Minute volume (MV) during volume-controlled ventilation (VCV) at different ambient pressures, data are mean (standard deviation); $^{*}P < 0.05$, ventilator vs. test lung; $^{a}P < 0.05$ vs. 1.0 atm abs group, $^{b}P < 0.05$ vs. 1.5 atm abs group, $^{c}P < 0.05$ vs. 2.0 atm abs group

VTset (mL)	Equipment	MV (L·min^{-1})			
		1.0 atm abs	1.5 atm abs	2.0 atm abs	2.8 atm abs
400	Ventilator	7.81 (0.02)	7.90 (0.01)^{a}	7.82 (0.01)^{a}	7.73 (0.01)^{a}
	Test lung	7.33 (0.09)*	7.51 (0.10)^{*a}	7.65 (0.08)^{*a}	7.61 (0.14)^{*a}
500	Ventilator	9.78 (0.04)	9.85 (0.11)^{a}	9.69 (0.07)^{a}	9.53 (0.06)^{b}
	Test lung	9.26 (0.14)*	9.54 (0.16)^{*a}	9.57 (0.11)^{*a}	9.63 (0.13)^{*b}
600	Ventilator	11.70 (0.05)	11.67 (0.13)^{a}	11.39 (0.08)^{b}	11.10 (0.01)^{abc}
	Test lung	11.28 (0.25)	11.55 (0.21)^{a}	11.49 (0.19)^{b}	11.37 (0.17)^{abc}
800	Ventilator	15.86 (0.05)	15.41 (0.02)^{a}	15.20 (0.01)^{ab}	14.47 (0.05)^{abc}
	Test lung	15.79 (0.16)*	15.48 (0.23)^{*a}	15.44 (0.22)^{*ab}	14.97 (0.23)^{*abc}
1,000	Ventilator	19.63 (0.12)	19.20 (0.01)	18.73 (0.05)^{a}	22.89 (0.54)^{abc}
	Test lung	19.54 (0.33)*	19.48 (0.37)*	19.39 (0.33)^{*a}	24.77 (0.19)^{*abc}

VT, MV, Ppeak and PEEP were analysed. A *P*-value smaller than 0.05 was considered significant. We used SPSS19.0 to perform the statistical analysis and GraphPad Prism 5 to prepare graphs.

Results

At the same ambient pressure, VT and MV displayed by the ventilator and the test lung were compared. With increasing ambient pressure, the change trend at VTset 400–600 mL detected by the ventilator decreased, but the change trend detected by the test lung increased; the change trend at VTset 800–1,000 mL detected by the ventilator was in accordance with the test lung (Figures 3 and 4). There was a significant difference between the ventilator and the test lung at VTset 400–1,000 mL (Tables 2 and 3). Surprisingly, VT and MV increased sharply at VTset 1,000 mL at 2.8 atm abs.

Meanwhile, at every VTset, the Ppeak displayed by the ventilator and the test lung were almost identical at each fixed ambient pressure, except for the significant differences of Ppeak at VTset 500 mL and 800 mL at 2.0–2.8 atm abs (Table 4). However, when the ambient pressure increased, Ppeak increased obviously at VTset 400–1,000 mL (Figure 5).

Table 4

Peak inspiratory pressure (Ppeak) during volume-controlled ventilation (VCV) at different ambient pressures, data are mean (standard deviation); $^{*}P < 0.05$, ventilator vs. test lung; $^{a}P < 0.05$ vs. 1.0 atm abs group, $^{b}P < 0.05$ vs. 1.5 atm abs group, $^{c}P < 0.05$ vs. 2.0 atm abs group

VTset (mL)	Equipment	Ppeak (kPa)			
		1.0 atm abs	1.5 atm abs	2.0 atm abs	2.8 atm abs
400	Ventilator	2.44 (0.02)	2.58 (0.03)^{a}	2.71 (0.04)^{ab}	2.96 (0.05)^{abc}
	Test lung	2.47 (0.02)	2.60 (0.02)^{a}	2.73 (0.03)^{ab}	2.94 (0.04)^{abc}
500	Ventilator	3.06 (0.03)	3.28 (0.04)^{a}	3.50 (0.05)^{ab}	3.87 (0.06)^{abc}
	Test lung	3.06 (0.03)*	3.28 (0.03)^{*a}	3.48 (0.04)^{*ab}	3.78 (0.04)^{*abc}
600	Ventilator	3.67 (0.04)	4.02 (0.05)^{a}	4.24 (0.04)^{ab}	4.63 (0.05)^{abc}
	Test lung	3.68 (0.03)	4.02 (0.04)^{a}	4.23 (0.05)^{ab}	4.58 (0.07)^{abc}
800	Ventilator	2.00 (0.03)	2.04 (0.03)^{a}	2.13 (0.04)^{ab}	2.24 (0.04)^{abc}
	Test lung	2.02 (0.02)*	2.07 (0.02)^{*a}	2.16 (0.02)^{*ab}	2.25 (0.02)^{*abc}
1,000	Ventilator	2.46 (0.04)	2.59 (0.04)^{a}	2.72 (0.03)^{ab}	4.35 (0.05)^{abc}
	Test lung	2.48 (0.02)	2.61 (0.02)^{a}	2.75 (0.02)^{ab}	4.29 (0.02)^{abc}

Figure 5

A – changes in peak inspiratory pressure (Ppeak) during volume-controlled ventilation (VCV) with preset tidal volume (VTset) 400–600 mL at different ambient pressures; B – changes in peak inspiratory pressure (Ppeak) during volume-controlled ventilation (VCV) with preset tidal volume (VTset) 800–1,000 mL at different ambient pressures

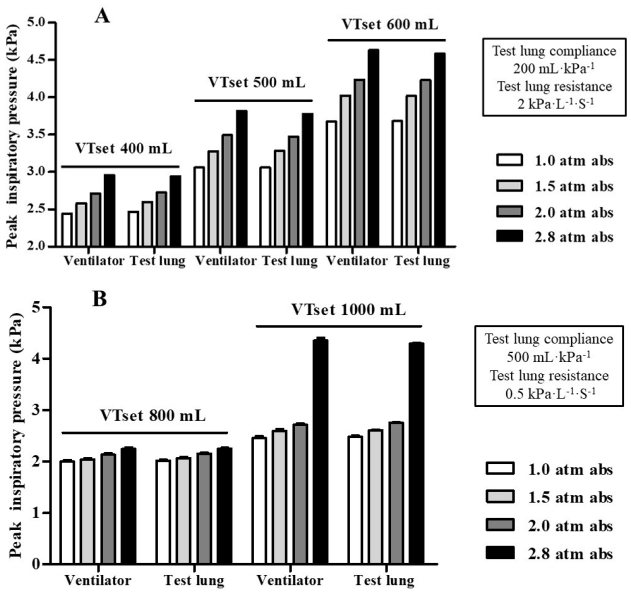
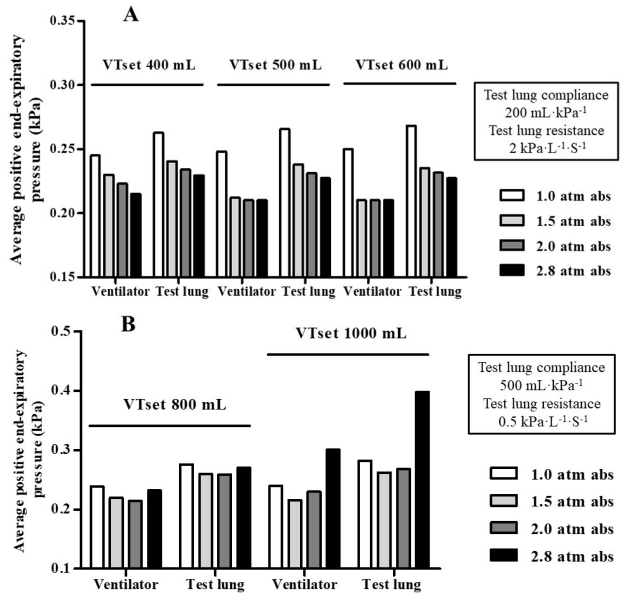


Figure 6

A – changes in positive end-expiratory pressure (PEEP) during volume-controlled ventilation (VCV) with preset tidal volume (VTset) 400–600 mL at different ambient pressures; B – changes in positive end-expiratory pressure (PEEP) during volume-controlled ventilation (VCV) with preset tidal volume (VTset) 800–1,000 mL at different ambient pressures



Positive end-expiratory pressure (PEEP) detected by the ventilator and test lung showed the same decreasing trend at VTset 400–600 mL at 1.0–2.8 atm abs (Figure 6A) and at VTset 800–1,000 mL at 1.0–2.0 atm abs. It increased only at VTset 800–1,000 mL at 2.8 atm abs (Figure 6B).

At each fixed VTset the static lung compliance (Cs) seemed to decrease as ambient pressure increased. There was a significant difference at VTset 400–1,000 mL among different ambient pressures (Table 5 and Figure 7). Airway resistance increased with increasing ambient pressure, and there was a significant difference at VTset 400–1,000 mL (Table 6 and Figure 8).

Table 5

Static lung compliance (Cs) detected by the ventilator during volume-controlled ventilation (VCV) at different ambient pressures, data are mean (standard deviation); $^{*}P < 0.05$ vs. 400 mL, $^{\#}P < 0.05$ 800 mL vs. 1,000 mL, $^{a}P < 0.05$ vs. 1.0 atm abs group, $^{b}P < 0.05$ vs. 1.5 atm abs group, $^{c}P < 0.05$ vs. 2.0 atm abs group

VTset (mL)	Static compliance (mL·kPa^{-1})			
	1.0 atm abs	1.5 atm abs	2.0 atm abs	2.8 atm abs
400	181.4 (48.8)	179.8 (35.8)	118.2 (64.1)^{ab}	83.2 (32.3)^{ab}
500	200.6 (13.7)	175.8 (38.0)	140.4 (41.1)^{ab}	107.6 (30.9)^{ab}
600	201.0 (17.1)	181.8 (35.0)	141.2 (41.8)^{ab}	127.2 (41.5)^{ab}
800	449.0 (25.7)	423.6 (106.5)	382.2 (133.3)	288.2 (85.9)^{ab}
1,000	513.8 (116.2)$^{\#}$	473.7 (123.5)$^{\#}$	484.2 (115.9)$^{\#}$	373.5 (113.0)^{ab}

Table 6

Airway resistance (Raw) detected by the ventilator during volume-controlled ventilation (VCV) at different ambient pressures, data are mean (standard deviation); $^{*}P < 0.05$ vs. 400 mL, $^{\Delta}P < 0.05$ vs. 500 mL, $^{\#}P < 0.05$ 800 mL vs. 1,000 mL, $^{a}P < 0.05$ vs. 1.0 atm abs group, $^{b}P < 0.05$ vs. 1.5 atm abs group, $^{c}P < 0.05$ vs. 2.0 atm abs group

VTset (mL)	Airway resistance (kPa·L^{-1}·S^{-1})			
	1.0 atm abs	1.5 atm abs	2.0 atm abs	2.8 atm abs
400	0.29 (0.07)	0.45 (0.10)	0.51 (0.04)^{a}	0.62 (0.16)^{ab}
500	0.53 (0.03)*	0.61 (0.18)*	0.66 (0.16)^{*a}	0.83(0.36)^{*ab}
600	0.67 (0.07)$^{*\Delta}$	0.69 (0.05)$^{*\Delta}$	1.05 (0.18)$^{*\Delta a}$	1.31 (0.30)$^{\Delta ab}$
800	0.22 (0.05)	0.27 (0.04)	0.28 (0.04)	0.39 (0.08)^{abc}
1,000	0.27 (0.05)$^{\#}$	0.35 (0.03)$^{\#}$	0.32(0.11)$^{\#}$	0.70 (0.20)$^{\#abc}$

Figure 7

Changes in static lung compliance (Cs) detected by the ventilator during volume-controlled ventilation (VCV) at different ambient pressure; error bars represent standard deviation

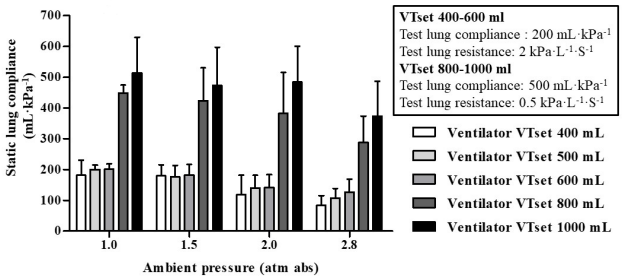
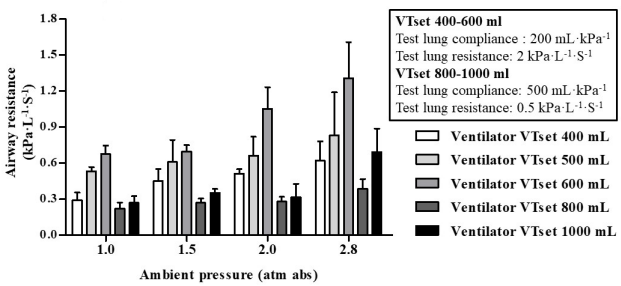


Figure 8

Changes in airway resistance (Raw) detected by the ventilator during volume-controlled ventilation (VCV) at different ambient pressures; error bars represent standard deviation



Interestingly, it was observed that inspiratory flow increased suddenly at VTset 1,000 mL at 2.8 atm abs, associated with increased VT, MV and Ppeak. The flow setting was a square wave in the ventilator setting. Therefore, the flow displayed by the ventilator was approximately equal to the maximum inspiratory flow measured by the test lung. Inspiratory time (Ti) and I:E ratio will affect inspiratory flow at a fixed VTset. When the theoretical inspiratory flow was less than 60 L·min^{-1}, the inspiratory flow detected by the ventilator and test lung was stable and near the theoretical value. When the theoretical inspiratory flow was more than 60 L·min^{-1}, inspiratory flow measured by the ventilator decreased between 1.0 to 2.8 atm abs, but when measured by the test lung, it increased (Table 7).

Discussion

Previous research has shown that ordinary ventilators normally used at atmospheric pressure cannot maintain a stable VT during VCV when operated at higher pressures.

Table 7
Inspiratory flow measured by the ventilator and the test lung at different respiratory rates and inspiratory:expiratory (I:E) ratios; atm abs – atmospheres absolute; Ti – inspiratory time

Breaths per min	Inspiratory time (I:E ratio)	Theoretical value of inspiratory flow (L·min^{-1})	Ventilator / Test lung Maximum inspiratory flow (L·min^{-1})	
			1.0 atm abs	2.8 atm abs
10	Ti = 1.0 s (1:5)	60.0	61.0 / 147.2	53.0 / 209.7
	Ti = 1.5 s (1:3)	40.0	50.8 / 33.0	40.0 / 41.0
	Ti = 2.0 s (1:2)	30.0	30.0 / 32.3	30.0 / 38.5
15	Ti = 1.0 s (1:3)	60.0	60.0 / 138.5	60.0 / 190.9
	Ti = 1.3 s (1:2)	46.2	45.0 / 50.0	46.0 / 46.5
20	Ti = 0.8 s (1:2.8)	75.0	72.0 / 79.1	52.0 / 84.1
	Ti = 0.9 s (1:2.3)	66.7	65.0 / 94.1	52.0 / 96.6
	Ti = 1.0 s (1:2)	60.0	60.0 / 148.7	51.0 / 218.8
	Ti = 1.2 s (1:1.5)	50.0	50.0 / 57.5	60.0 / 56.1

Inspiratory flow provided by the ventilator will decrease with increasing ambient pressure.[7–11] The reason is that during HBOT the respired gas density becomes higher and produces more turbulent flow in airways and external circuits.[17] To obtain stable inspiratory flow, more driving pressure (△P) must be provided by the ventilator to overcome the increased Raw produced by the increased turbulent flow.[11] To maintain stable VT a ventilator used in the hyperbaric chamber must autoregulate △P to compensate for this change.

EVALUATION OF VT DURING VCV AT HIGH AMBIENT PRESSURE

During VCV, the Shangrila590 ventilator can achieve constant VT and MV, even though VT and MV decreased within a narrow range compared with VTset, except at VTset 1,000 mL at 2.8 atm abs. Measured by the test lung, the range of VT was less than 5% for VTset of 400–800 mL from 1.0 to 2.8 atm abs. The range of VT was 2–5% for VTset 1,000 mL from 1.0 to 2.0 atm abs. In contrast, in non-adapted ICU ventilators during VCV in hyperbaric environments the fall in VT at the same ambient pressures was greater than 50%.[7,9] The Siaretron IPER 1000 hyperbaric ventilator is CE-certified for hyperbaric use in Europe. In tests of this device during VCV (Ti 1.0 s), a 4–10% increase in VT at VTset 500 mL at ambient pressures between 2.2–2.8 atm abs and an 11–21% decrease at VTset 750 mL at ambient pressures between 2.0–2.8 atm abs was seen.[10] A modified Penlon Nuffield 200 used in a monoplace hyperbaric chamber and fixed outside the chamber showed a 40% decrease in VT at ambient pressures from 1.0 to 2.3 atm abs (flow setting: 0.25–1 L·s^{-1}).[18]

In the present study, we were surprised to find that VT and MV increased by 27% at 2.8 atm abs with VTset at 1,000 mL, and we carried out complementary tests (Table 7). The relationship between inspiratory valve opening and volume flow is constant only for a specified gas density.[7] When the theoretical inspiratory flow is more than 60 L·min^{-1}, the inspiratory flow provided by the ventilator is unstable. If the inspiratory valve cannot close immediately at the end of inspiration, more inspiratory flow will be detected by the test lung. The opening degree and closing speed of the ventilator valve may be influenced by the high inspiratory flow, especially at high pressure.

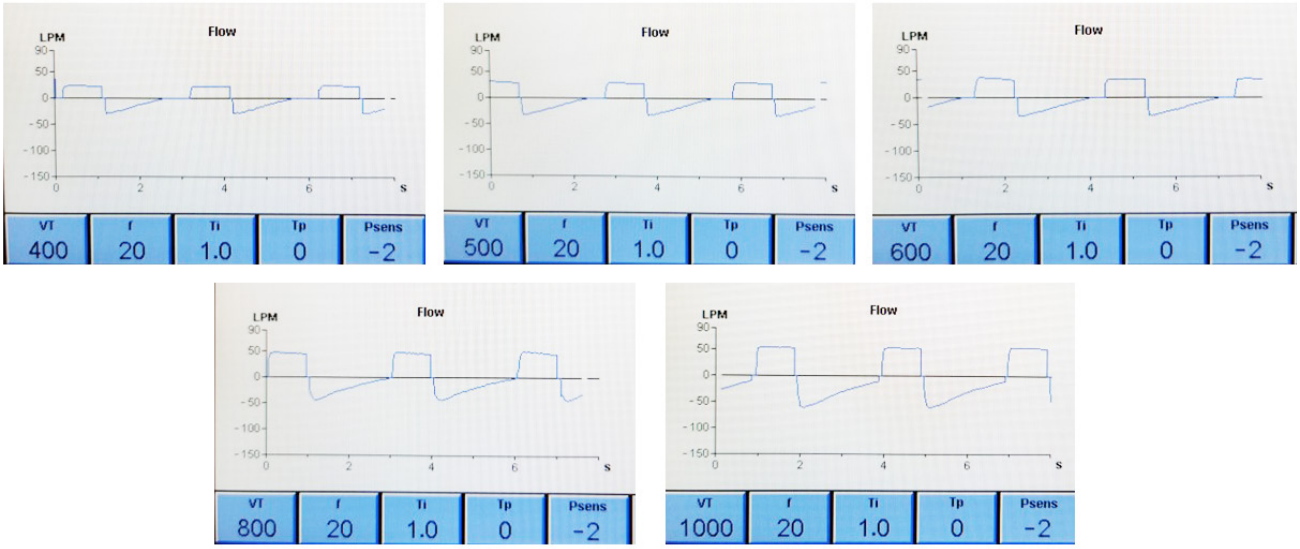
CHANGES IN Ppeak DURING VCV AT HIGH AMBIENT PRESSURE DUE TO HIGHER INSPIRATORY RESISTANCE

Peak inspiratory pressure primarily reflects inspiratory resistance and △P indirectly, as our results show in Table 4 and Figure 5. In clinical use, attention must be given to Ppeak increases associated with increases in ambient pressure. These increases cannot be avoided, though some patient-centered strategies may help such as ensuring adequate paralysis, sputum aspiration, bronchodilation (if applicable). Similarly, environmental factors such as reducing ambient pressure and use of lower density respired gases (such as a helium oxygen mixture) can help if the clinical circumstances allow it.

CHANGES IN PEEP DURING VCV AT HIGH AMBIENT PRESSURE BECAUSE OF HIGHER EXPIRATORY RESISTANCE

Higher expiratory resistance may occur, and expiratory flow may decrease during HBO.[17] In a previous study, PEEP was set to 0.1–0.2 kPa in an ICU ventilator (EVITA 4), and PEEP decreased to zero at 1.9 and 2.8 atm abs.[7] The valve that regulates the PEEP is controlled pneumatically by the ventilator and is likely to be affected by the higher density of driving gas.[7] As shown in Figure 6, the PEEP at VTset 400–1,000 mL decreased with increasing

Figure 9
Flow-time curve displayed by the ventilator during volume-controlled ventilation (VCV) with preset tidal volume (VTset) 400–1,000 mL at 2.8 atm abs



ambient pressure, but at 2.8 atm abs, the PEEP at VTset 800–1,000 mL increased. Figure 9 shows that at 2.8 atm abs, the expiratory flow at VTset 400–600 mL returned to zero (baseline) and remained static until the next inspiration; the expiratory flow at VTset 800 mL returned to baseline without any buffer time, which resulted in a slight increase in PEEP; the expiratory flow at VTset 1,000 mL did not return to baseline before the next inspiration; and incomplete expiration resulted in an obvious increase in PEEP.

CHANGES IN Cs AND Raw DURING VCV AT HIGH AMBIENT PRESSURE

Volume control ventilation emphasises stable volume flow, but at higher ambient pressure, the stability of volume flow accompanies the increased mass flow because of increased gas density. According to the resistance formula, Cs and Raw can directly influence the work of breathing.[19] Combined with the breathing equipment itself, the work of breathing will be increased compared with that in a normobaric environment.[6,19] In the ICU, the endotracheal tube diameter is a critical factor in breathing work.[20] Additionally, we can decrease airway resistance by appropriately prolonging the inspiratory time, using a helium oxygen mixture to decrease the gas density, or down regulating ambient pressure.[19]

LIMITATIONS

A limitation of this research is the narrow VTset levels of 400–1,000 mL, and small VTset volumes relevant to paediatric practice were not included in this work. We will conduct additional research using a small VTset of 50–300 mL in the future to comprehensively check the performance of the ventilator in a hyperbaric chamber.

Conclusions

In summary, over a range of ambient pressures from 1.0 to 2.8 atm abs, the new hyperbaric oxygen ventilator (Shangrila590) made in China can provide relatively stable VT and MV during VCV with VTset levels from 400 to 1,000 mL, except at VTset 1,000 mL at 2.8 atm abs. The changes in VT are acceptable with VTset from 400 to 800 mL at 1.0–2.8 atm abs and 1,000 mL at 1.0–2.0 atm abs. During VCV, Ppeak unavoidably increases, and PEEP may be influenced at high ambient pressure.

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Short communication

Hyperbaric medicine in Canadian undergraduate medical school curriculum

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Education; Hyperbaric oxygen treatment; Teaching

Abstract
(Talbot Z, Lee A, Boet S. Hyperbaric medicine in Canadian undergraduate medical school curriculum. Diving and Hyperbaric Medicine. 2023 June 30;53(2):138–141. doi: 10.28920/dhm53.2.138-141. PMID: 37365131.)
Introduction: Hyperbaric oxygen treatment (HBOT) has fourteen approved indications in the management of acute and chronic diseases in various medical specialties. However, lack of physician knowledge and exposure to hyperbaric medicine may hinder the ability of patients to access this treatment option for approved indications. We aimed to determine the prevalence and nature of HBOT-related learning objectives in Canadian undergraduate medical education programs.
Methods: Pre-clerkship and clerkship learning objectives from responding Canadian medical schools' curricula were reviewed. These were acquired through the school websites or by emailing the faculties. Descriptive statistics were used to summarise the number of hyperbaric medicine objectives taught in Canadian medical schools, and within each institution.
Results: Learning objectives from seven of the 17 Canadian medical schools were received and reviewed. From the curriculum of the responding schools, only one objective was found to be related to hyperbaric medicine. Hyperbaric medicine was absent from the other six schools' objectives.
Conclusions: Based on the responding Canadian medical schools, hyperbaric medicine objectives were mostly absent from undergraduate medical curricula. These findings illustrate a possible gap in HBOT education and the need for discussion regarding the design and implementation of HBOT educational initiatives in medical training.

Introduction

Hyperbaric oxygen treatment (HBOT) is a well-established and approved treatment modality for a range of elective and urgent conditions in North America.[1] However, the physiological and biochemical mechanisms require a deeper understanding to be able to fully appreciate the actions of oxygen as a drug under hyperbaric conditions.[2–4]

In Canada there are currently 14 approved indications for HBOT[5] in correlation to the Undersea and Hyperbaric Medical Society (UHMS) list[1] (Table 1), which relate to various medical specialities, as well as multiple other emerging indications still under study.[5,6] Despite this, evidence suggests that physicians are unaware of HBOT indications and thus may not refer patients for treatment.[7,8] This becomes critical in situations where treatment needs to be initiated urgently, such as with air/gas embolism. For patients in Canada to access HBOT, their physician must refer them to a hyperbaric medicine physician. Therefore, referring physicians must be aware of the indications for HBOT and educated on the potential benefits of this treatment modality.

The lack of physician knowledge on HBOT[7,8] and potential benefits for patients with approved indications[1] highlight the importance of exploring the barriers to HBOT education. Medical school is the foundation of physicians' training and exposes students to a large amount of information which is consolidated throughout their career. By assessing the presence of HBOT learning objectives in medical school curricula, an area of possible improvement for

Table 1
Approved indications for HBOT

Undersea and Hyperbaric Medical Society 2019 [1]	Indications in Canada [6]
Air or gas embolism	Air/gas embolism
Carbon monoxide poisoning	Carbon monoxide poisoning
Clostridial myonecrosis (gas gangrene)	Gas gangrene
Acute traumatic ischaemias	Crush injury, compartment syndrome and other acute traumatic problems where blood flow is reduced or cut off
Decompression sickness	Decompression sickness
Arterial insufficiencies: A. Central retinal artery occlusion B. Selected problem wounds	Enhancement of healing for wounds ie diabetic foot ulcers
Severe anaemia	Exceptional blood loss
Intracranial abscess	Intracranial abscess
Necrotizing soft tissue infections	Necrotizing soft tissue infections
Refractory osteomyelitis	Osteomyelitis
Delayed radiation injuries (soft tissue and bony necrosis)	Delayed radiation injury
Compromised grafts and flaps	Skin grafts and flaps that are not healing well
Thermal burns	Thermal burns
Sudden sensorineural hearing loss	Idiopathic sudden sensorineural hearing loss (ISSHL)

HBOT education could be uncovered. This report aimed to determine the prevalence of HBOT content in the pre-clerkship and clerkship learning objectives of undergraduate medical education in Canada.

Methods

ETHICAL ISSUES

This report followed Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines[9] and was exempted from ethics review by the Ottawa Health Science Network Research Ethics Board.

SETTING

A standardised email was sent to all 17 Canadian medical schools accredited by the Committee on Accreditation of Canadian Medical Schools (CACMS).[10] It was directed to their undergraduate medical offices or to the schools' resource contact. In the email, pre-clerkship and clerkship learning objectives were requested. A search through the faculties' websites was also performed to collect the learning objectives that were reported online to the public. We followed up with a phone call to non-responders.

DATA COLLECTION

We conducted a direct content analysis. Learning objectives relevant to hyperbaric medicine were identified from the collected curricula. The 'command search' tool was used to search for the term 'hyperbaric' in the objective repository collected from the school or through their website. Several schools did not agree to share their objectives but were willing to conduct the search themselves. In these cases, confirmation emails reporting their findings were accepted. Identified learning objectives on hyperbaric medicine were extracted and included in the analysis.

DATA ANALYSIS

Descriptive statistics were used to summarise the number of hyperbaric medicine objectives taught in Canada and within each medical school. Microsoft Excel (version 16.36, Microsoft® Corporation, Redmond Washington, USA) was used for the statistical analysis.

Results

Learning objectives from seven out of the 17 Canadian medical school were reviewed for mention of hyperbaric

Table 2
Learning objectives relating to hyperbaric medicine in Canadian medical schools; East of Canada is composed of New Brunswick, Newfoundland and Labrador, Nova Scotia, Nunavut, Prince Edward Island; West of Canada is composed of Alberta, British Columbia, Manitoba, Saskatchewan, Northwest Territories, Yukon

Medical school	Location	Number of relevant learning objectives	Obtention of objectives
1	East of Canada	None	By university staff
2	Québec	Non responder	–
3	Ontario	None	By university staff
4	East of Canada	None	By university staff
5	Ontario	Non responder	–
6	Ontario	None	By university staff
7	Québec	Non responder	–
8	Québec	Non responder	–
9	Québec	One objective: "*Explain the treatments of carbon monoxide intoxication including the general indications of hyperbaric oxygen therapy*"	By university staff
10	West of Canada	Non responder	–
11	West of Canada	Non responder	–
12	West of Canada	Non responder	–
13	West of Canada	Non responder	–
14	Ontario	None	By researcher through the university's website
15	West of Canada	None	By researcher through the university's website
16	Ontario	Non responder	–
17	Ontario	Non responder	–

medicine. Objectives from 10 schools were unobtainable due to inability to access/privacy restrictions or due to lack of response from the corresponding undergraduate medical offices despite follow-up (Table 2).

Objectives from two medical school were searched online through their website while five other schools confirmed that they searched their curricula for hyperbaric medicine. Out of these seven medical schools who responded, only one of them had a learning objective regarding hyperbaric medicine. The learning objective related to carbon monoxide intoxication and the main indications for hyperbaric oxygen therapy (Table 2).

Discussion

In this report, we aimed to investigate the prevalence and nature of hyperbaric medicine objectives in the Canadian medical schools' curricula. Learning objectives from seven out of the 17 Canadian medical school were searched for mention of hyperbaric medicine. A single relevant objective at a single school was found.

The near absence of learning objectives in medical school curricula suggests that hyperbaric medicine is probably not formally taught to medical students in Canadian institutions. This may be surprising given its use for decades as an approved treatment modality for several illnesses. However, access to HBOT is still limited in many parts of Canada, as well as in regions around the world. According to the Canadian Undersea and Hyperbaric Medical Association (CUHMA) only 14 Canadian cities have hyperbaric chambers, half of these being in Ontario. In fact, two territories and four provinces do not have a single hyperbaric facility.[11] The lack of wide availability of HBOT to most patients may be a reason for the absence of this topic in undergraduate medical education.

The next step may be to determine the best time, location and instructional design for teaching basic understanding of HBOT and its indications to trainees and physicians. Medical school is where physicians learn the most common and life-threatening diseases as well as their main treatments. Indications such as gas embolism or decompression sickness may fall under life-threatening disease management, while others may fall under common elective conditions, such as

delayed radiation-induced injury or diabetic foot ulcers.[5,6] Indications of HBOT are diverse and are relevant to many different medical practices/specialities. Additionally, medical school is the only time in the continuum of education where all (future) physicians are exposed to a common base of knowledge. Therefore, one may consider that HBOT, with its overlap on a variety of specialities, should be included in the undergraduate medical school curricula. However, with the obvious exceptions of decompression sickness and gas embolism, hyperbaric medicine remains a supplemental treatment in most of its indications.[1] It is also possible that HBOT exposure is more suited to post-graduate medical education or continuing professional development as this is where learners enhance the basic skills learned in medical school to better treat a certain population of patients. Learning at this stage also considers the resources available in their location of practice. Therefore, physicians who will likely work with patients who could access and benefit from HBOT would be exposed to it.

While this report suggests a lack of education on HBOT, some limitations should be noted. First, only seven out of the 17 medical schools participated. Therefore, we cannot know whether the missing schools have learning objectives related to hyperbaric medicine, although it seems plausible that they do not. A different approach may be necessary to further explore undergraduate medical education related to HBOT. Perhaps, the use of surveys sent to students, or a qualitative approach is needed. Second, while learning objectives are the most specific information we can retrieve on curricula, they may not be comprehensive. Many topics are covered during medical school lectures without being explicitly stated in learning objectives. Therefore, our approach could underestimate the presence of this topic in undergraduate medical education. Finally, there may have been inconsistencies to the methodology as some schools opted to conduct their own search for hyperbaric medicine within their learning objectives. However, the keyword 'hyperbaric' is quite specific to HBOT in learning objectives.

Conclusions

Hyperbaric medicine was mostly absent from the undergraduate medical education learning objectives of the sample of Canadian medical school curricula reviewed. These findings may represent a potential barrier for patients to benefit from HBOT when indicated. Given its effectiveness for approved indications, the identified educational gaps related to HBOT should warrant meaningful objectives incorporation and effective educational design and implementation.

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Rebreather Forum Four consensus statements

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Abstract

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Closed circuit rebreathers have been widely adopted by technical divers as tools for reducing gas consumption and extending depth and duration capabilities. Rebreathers are technologically complex with many failure points, and their use appears associated with a higher accident rate than open circuit scuba. Rebreather Forum Four (RF4) was held in Malta in April 2023 attracting approximately 300 attendees and representatives of multiple manufacturers and training agencies. Over two and a half days a series of lectures was given by influential divers, engineers, researchers and educators on topics of contemporary relevance to rebreather diving safety. Each lecture was followed by a discussion session with audience participation. Potential consensus statements were drafted by the authors (SJM and NWP) during the course of the meeting. These were worded to be confluent with some important messages emerging from the presentations and subsequent discussions. The statements were presented one by one in a half-day plenary session of participants, and discussion was invited on each. After discussion and any necessary revision, the participants voted on whether to adopt the statement as a position of the forum. A clear majority was required for acceptance. Twenty-eight statements embracing thematic areas designated 'safety', 'research', 'operational issues', 'education and training', and 'engineering' were adopted. Those statements are presented along with contextualising narrative where necessary. The statements may help shape research and teaching initiatives, and research and development strategies over subsequent years.

Introduction

The use of rebreather devices to facilitate deeper and longer dives has become common among so-called 'technical divers'.[1] Rebreathers offer several important advantages, including minimisation of gas consumption and optimisation of inspired pressure of oxygen throughout a dive. These are important advantages when attempting to reduce the use of expensive gases like helium, and to optimise the efficiency of decompression. Rebreathers are complex devices with more failure points than typical open circuit scuba, and it is not surprising that their use seems associated with a significantly higher accident rate.[2]

Such is the importance of rebreather technology to the technical diving community that over several decades this single item of equipment has been the subject of four focused conferences designated Rebreather Forums One through Four. Presentations and discussions at these forums have focussed primarily on technological developments, relevant research, and training and safety issues. Rebreather Forum Three (RF3) held in Orlando, Florida, in 2012 was arguably the first of the four meetings held in an era of widespread adoption of rebreathers. Rebreather Forum Four (RF4) was held in Malta in April 2023 attracting approximately 300 attendees and representatives of multiple manufacturers and training agencies. Over two and a half days a series of lectures was given by influential divers, engineers, researchers and educators on topics of contemporary relevance. Each lecture was followed by a discussion session with audience participation.

Following a precedent set at RF3[3] the final half day of RF4 was dedicated to discussion of a series of consensus statements intended to reflect current evidence and strongly supported opinion of the presenters and participants on important contemporary issues.

Methods

Potential consensus statements were drafted by the authors (SJM and NWP) during the course of the meeting. These

were worded to be confluent with important messages emerging from the presentations and subsequent discussions. The statements were not intended to capture all important messages to emerge from the forum; the authors focused on matters that seemed important, widely supported and relatively non-controversial, and that would therefore lend themselves to meaningful consensus.

The statements were presented one by one to a half day plenary session of participants and discussion was invited on each. The amount of discussion was variable with some statements attracting little, and others requiring more debate and wordsmithing which was performed live. As is the case with sessions of this nature some degree of directive chairmanship was required in order to work through the list of statements within the allocated time. For this reason, discussion of some statements had to be truncated but there was substantial discussion of all statements that emerged as controversial for any reason. For transparency, a transcript of the discussion will be presented in the forum proceedings. After discussing each statement a show of hands was taken to gauge agreement and disagreement with the statement. It was announced prospectively that a clear majority of participants would need to agree for a statement to make the published list. Ultimately, after discussion and rewording where necessary, all draft statements were accepted; most unanimously and never with more than 5–10% in disagreement.

The 28 statements are presented in thematic areas designated 'safety,' 'research,' 'operational issues,' 'education and training,' and 'engineering.' The authors acknowledge that some of these statements seem relevant to multiple themes. Most are self-explanatory, but some are accompanied by contextualising narrative from the authors where necessary.

Statements

THEMATIC AREA 'SAFETY'

Accident data

Analysis of contemporary rebreather accident data indicates a continued need for integrated effort to reduce the rates of injury, morbidity, and mortality associated with rebreather diving.

Cardiac health surveillance

The forum endorses the principle of periodic cardiac health surveillance for all rebreather divers with an emphasis on targeted annual or biennial evaluation for divers older than 45 years even in apparent good health.

Contextualising narrative: the forum resolved that this statement should be accompanied by citation of relevant supportive medical literature. Various studies have identified the importance of cardiac events as the disabling injury in recreational diving fatalities,[4,5] and an expert consensus guideline for cardiac evaluation of divers was recently published.[6]

Accident analysis

The analysis of accident, incident, and injury data from rebreather incidents should consider wider contextual elements and error-producing conditions and not just immediate contributory factors.

Solo diving

The forum recognises that solo diving may increase the likelihood of a fatality in the event of a rebreather diving incident.

Pre-entry checklists

The forum strongly advocates the use of a pre-entry checklist (in a check and response format if practicable) administered just prior to water entry. This should be a brief checklist addressing contextually relevant critical safety items such as "*rebreather switched on*," "*oxygen cylinder on*," "*diluent cylinder on*," "*wing/buoyancy device/dry suit inflation connected and working*."

THEMATIC AREA 'RESEARCH'

Training and sales data

The forum strongly endorses continued collection of anonymised rebreather diver training and rebreather unit sales data by the Divers Alert Network (DAN) Research Department as an adjunct to interpreting diver accident statistics.

Mishap and near-miss reporting

The forum advocates self-reporting of diving mishaps and near-misses, and reporting of fatalities, to the DAN diving incident reporting system.

Contextualising narrative: The DAN diving incident reporting system was nominated in this statement because of its high visibility, global scope, and accessibility for divers anywhere in the world. However, the forum also acknowledged the value of national or regional systems of relevant data collection and analysis (such as that run by the British Sub-Aqua Club [BSAC]) and also advocates for maintenance of diver reporting to such systems. Data sharing between DAN and regional groups was also discussed and was supported.

End-tidal CO_2 monitoring

The forum identifies as a research priority/goal the development of capnography and accurate end-tidal CO_2 monitoring for rebreathers.

Regenerating CO_2 absorption technology

The forum identifies as a research priority the development of regenerating CO_2 absorption technologies.

Full-face masks

In relation to a documented RF3 research priority, the forum recognises the emergence of data pertaining to the efficacy of full-face masks in preventing water aspiration in unconscious subjects.[7] This strengthens the argument for considering their use in scenarios associated with an elevated risk of oxygen toxicity such as in-water recompression.

Real-time physiological monitoring

The forum endorses ongoing research into strategies for real-time diver physiological monitoring.

THEMATIC AREA 'OPERATIONAL ISSUES'

Bailout rebreathers

The forum identifies as a priority or goal the development and documentation of practices and/or monitoring for optimising bailout rebreather use.

Mouthpiece retaining straps

The forum recognises the use of correctly deployed mouthpiece retaining straps as a strategy for avoiding loss of the mouthpiece and minimisation of water aspiration in the event of loss of consciousness underwater.

Bailout valves

The forum recognises the potential advantage of a bailout valve for transitioning from closed- to open-circuit in the event of hypercapnia or other events requiring bailout; this advantage requires a high performance open-circuit breathing system.

Mixed mode diving

The forum recognises mixed mode diving as a legitimate buddy option in dives of appropriate scope but recommends a mixed mode briefing, and pre-establishment of strategies for gas sharing.

Contextualising narrative: 'Mixed mode' in this context refers to divers using different underwater breathing apparatus types working as a buddy pair, for example, an open-circuit diver diving with a rebreather diver.

Mixed platform diving

The forum recognises mixed platform diving as a legitimate buddy option and recommends at least a mixed platform briefing with emphasis on emergency procedures.

Contextualising narrative: 'Mixed platform' in this context refers to divers using different brands or models of the same underwater breathing apparatus type working as a buddy pair, for example, two divers using different brands of rebreather.

Bailout rebreather symmetry

The forum recognises symmetric (same rebreather unit) or asymmetric (different rebreather unit) multiple rebreather systems as options for an alternative breathing or bailout system.

Contextualising narrative: 'Symmetric' in this context refers to multiple rebreathers of the same make and type, and 'asymmetric' refers to multiple rebreathers of different makes or types.

Head-up Display

The forum recommends the display of safety-critical information such as loop oxygen status on a head-up display.

Expedition standard operating procedures and emergency action plan documentation

The forum endorses the compilation of a contextually tailored and detailed dive plan/standard operating procedures document and emergency action plan prior to rebreather diving expeditions.

Emergency preparedness

The forum endorses the importance of emergency preparedness including a validated emergency action plan, oxygen supplies, access to appropriate medical support with adequate medical supplies, and evacuation plans during rebreather diving expeditions; particularly to remote locations.

In-water recompression

The forum recognises the recent medical endorsement of emergency in-water recompression of selected divers by appropriately equipped teams trained in oxygen decompression.[8,9]

THEMATIC AREA 'EDUCATION AND TRAINING'

Manufacturer-training agency coordination

The forum recognises the challenges for training agencies in maintaining confluence between course content/availability and emergence of new rebreather technologies. The forum endorses close liaison between training agencies and manufacturers (including factory trainers) to share information about emerging technologies and manufacturer expectations on training approaches using their platforms.

Knowledge gap targets

The forum identifies the following as common knowledge gaps that constitute educational opportunities for rebreather instructors and leaders to address:

– Predispositions, symptoms, and frequency of immersion pulmonary oedema
– Increasing risk of deeper dives executed perfectly on the same decompression algorithm (i.e., these are not iso-risk exposures)
– Scope of variability in venous gas emboli counts in individual divers serially performing identical dives and the associated implications for interpretation of individual monitoring of venous gas emboli post-dive
– The difference between CO_2 inhalation and hypoventilation as the two mechanisms of hypercapnia in rebreather diving
– Correct management of ingestion/inhalation of caustic scrubber by-product (i.e., a 'caustic cocktail')
– Functional characteristics of CO_2 scrubbers

Contextualising narrative: It is emphasized that this list is not intended to define all relevant knowledge gaps. Rather, it contains items that emerged as obvious educational opportunities in the various presentations and discussion at Rebreather Forum 4.

Diver retraining/updating

The forum recognises the potential for skill and knowledge degradation over time or during periods of diving inactivity and encourages training agency initiatives to promote continuing education and training, refresher options, and/or recertification as appropriate.

THEMATIC AREA 'ENGINEERING'

Oxygen sensor replacement warning

The forum recommends that manufacturer's consider incorporating oxygen sensor replacement warnings in rebreather operating systems.

Contextualising narrative: The context in which this discussion took place was that these warnings would be based on elapsed time since sensor manufacture.

Gas density warning

The forum recommends that rebreather manufacturers consider incorporating gas density displays and/or alarms in the user interface.

Contextualising narrative: The discussion around this statement included strong advocacy for viewing gas density as a dive planning and operational concern that requires careful consideration. Reference was made to recently published data identifying an inspired density threshold of 6 $g \cdot L^{-1}$ beyond which the risk of CO_2 retention rises significantly, especially during exercise.[10]

Orientation monitoring

The forum identifies optimally positioned accelerometers or inclinometers within rebreathers as an opportunity for capturing diver trim and movement data that could be used for training, performance, and forensic evaluation.

Inspired CO_2 monitoring

The forum recognises the potential safety advantage of inhale side CO_2 or scrubber monitors, but acknowledges that they may fail to detect some causes of hypercapnia.

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Professors Mitchell and Pollock are members of the *Diving and Hyperbaric Medicine* Editorial Board. However, this publication is a straightforward replication of the consensus statements agreed upon by the RF4 delegates and not a report of original work by the authors.

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Case reports

Successful hyperbaric oxygen treatment of a patient with a HeartMate III left ventricular assist device

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Abstract

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A 53-year-old woman with a HeartMate III left ventricular assist device (LVAD) was successfully treated under hyperbaric conditions for haemorrhagic cystitis. The HeartMate III LVAD inserted in this patient had not previously been tested or certified for use under hyperbaric conditions. To our knowledge this is the first report of the HeartMate III LVAD being used to support a patient undergoing hyperbaric treatment. The overview detailed here of the safety and technical aspects of managing this patient for hyperbaric treatment was possible due to the collaboration of a multi-disciplinary team. We believe that our experience has demonstrated a pathway to safe hyperbaric treatment of patients dependent upon a HeartMate III LVAD.

Introduction

There are significant limitations to the safe use of electromedical devices in patients receiving hyperbaric oxygen treatment (HBOT). Few medical devices are manufacturer approved, marketed and licensed for use in the unique environmental conditions that exist in hyperbaric chambers. Devices must be tolerant of the pressure changes involved, and the use of medical devices in the chamber must not present any hazard to the patient or others. In particular, the device must not create any fire ignition hazard, which can be more likely due to the altered operating environment, and which creates the risk of a potentially catastrophic fire given the increased oxygen availability under pressure.[1]

To our knowledge, there are only two prior published reports of patients with a left ventricular assist device (LVAD) being treated with HBOT. One group treated their patient by housing the external parts of the LVAD in a pressure resistant housing[2] while another performed some modifications to a HeartMate II LVAD that were similar to our approach in order to treat their LVAD-dependent patient.[3]

The patient reported here was referred for hyperbaric treatment of haemorrhagic cystitis (HC); a dysfunction of the bladder mucosa leading to bleeding which can be acute or insidious. It can be a complex disease process to manage. There are a number of potential causative factors and multiple aetiologies can be involved in some cases. Common causative factors are radiation injury,[4] chemotherapy,[4] haematopoietic stem cell transplantation,[5] infection (bacterial/viral/fungal), systemic disease, anticoagulation or idiopathic causes.[6]

Conventional management strategies for HC have included options such as clot extraction, continuous bladder irrigation, bladder instillations of haemostatic factors, formalin, arterial embolisation or salvage surgery.[6] In recent years, HBOT has been increasingly used in the treatment of HC with growing evidence of efficacy.[1,7,8]

Currently in Australia it is estimated that there are about 110,000 people living with heart failure based upon census data.[9] A proportion of these become dependent upon implanted ventricular assist devices which are mechanical pumps that augment the function of the damaged ventricle so as to restore normal haemodynamics and end-organ blood flow. An external power source / controller device is connected to the intra-thoracic pump via an electrical cable through the skin. The assistance provided by an LVAD allows the heart to rest and recover its function and in those who are not expected to recover adequate cardiac function; the LVAD can provide a bridge to transplantation.[10]

Case report

CLINICAL FACTORS

The patient consented to the publication of her case details. She is a 53-year-old woman who was referred to the Alfred Hospital's Hyperbaric Unit (HBU) for management of refractory HC. She presented a complicated past medical history, with localised cervical cancer diagnosed in 2005 and treated with chemotherapy, radiotherapy and a subsequent hysterectomy. She subsequently developed a non-ischaemic dilated cardiomyopathy (NIDCM) that led to severe heart failure, manifested on one occasion by an out-of-hospital cardiac arrest that she survived in 2015. Following this episode, she had an implantable cardiac defibrillator (ICD) inserted. The ICD is a Medtronic Amplia MRI Quad Biventricular ICD implanted in November 2018. Her ICD was approved by Medtronic to be pressurised to 4 atmospheres absolute (atm abs).[11] In late 2019 she presented to hospital with decompensated heart failure and this required insertion of a permanent LVAD and a temporary right ventricular assistance device. She was therapeutically anti-coagulated with a heparin infusion for these device insertions and sustained a kidney injury requiring continuous renal replacement therapy post operatively.

On day seven post-insertion of her cardiac assist devices, she experienced onset of frank haematuria. Imaging of the urinary tract with ultrasound and computed tomography (CT) demonstrated no anatomical cause for the haematuria. Haematuria continued despite conventional therapies including continuous bladder washout and treatment of potential urinary tract infections. She required multiple blood transfusions to combat anaemia resulting from the ongoing haematuria. A rigid cystoscopy demonstrated severe cystitis with multiple dilated and tortuous mucosal capillary vessels that were bleeding. Clot evacuation was performed and visible bleeding vessels were cauterised. She continued to have persistent bleeding for many weeks sufficient to require ongoing blood product transfusions. She was transitioned to anticoagulation with warfarin during this time in view of her LVAD. Further rigid cystoscopies were performed with injection of intra-vesical alum and further diathermy. Intra-vesical prostaglandin was ruled out as a viable option due to her brittle asthma. Viral causes such as cytomegalovirus and adenovirus infections were investigated and ruled out as potential contributors. Therapeutic anticoagulation was eventually ceased to moderate HC; requiring acceptance that this carried an increased risk of stroke. Her multiple comorbidities made her medically unsuitable to undergo a radical cystectomy or an urgent heart transplant and she was referred to the HBU for consideration of HBOT three months after onset of her HC.

She was considered a likely responder to HBOT given that the features of her HC were consistent with being a late radiation side effect.[5] Significant considerations arose during her assessment and consent for HBOT however. These included the tolerance and reliability of her ICD in the chamber at pressure, the need for her LVAD to work at pressure without risk of catastrophic cardiac failure if it failed and the safety risks these devices might pose in chamber. Her brittle asthma also needed consideration. These issues led to a significant delay of eight weeks whilst the LVAD technology issue was addressed. She had temporising bilateral nephrostomies performed during this period to minimise her bleeding risk by urinary diversion. She commenced HBOT in late December 2019 and successfully received 38 treatments in total, each at involving oxygen breathing at 243 kPa (2.4 atmospheres absolute) for a duration of approximately 90 minutes.

ADAPTION OF THE HEARTMATE III DEVICE

The HeartMate III LVAD is a widely used LVAD which is manufactured by Abbott Cardiovascular. Its components are:

- The implanted pump unit, consisting of a fully sealed electronically controlled motor driving a blood flow impeller via magnetic coupling;
- a power cable that connects the implanted pump with its external controller (termed '*the driveline*' by Abbott); this cable can be separated external to the patient's body should the controller need replacing;
- a pump controller which incorporates a liquid crystal display (LCD) screen and which has two power input cables;
- a mains power supply that can be used to power the LVAD when the patient will remain close to an electrical power outlet; and
- battery units to allow mobility.

Normally, one of the two power supply inputs should remain connected whenever the other is changed so that the LVAD is never without power. The controller incorporates a small battery which can power the LVAD should both of the power inputs be disconnected simultaneously or fail for any reason however the duration of operation enabled by this controller battery is a few minutes only.

The following summarises the evaluation and modification process for enabling use of the HeartMate III within the hyperbaric chamber at The Alfred hospital, an institution that has a strong history of assessing, developing and modifying medical devices for hyperbaric use. Several Alfred HBU hyperbaric technical officers hold qualifications in biomedical engineering.

The process commenced with an overview evaluation of the HeartMate III LVAD, to determine potential suitability for use within the hyperbaric chamber. These controllers were not certified from the manufacturer as hyperbaric compatible, therefore a detailed physical inspection, functional testing and failure modes evaluation was warranted. Abbott cardiovascular do not approve these units for use in hyperbaric chambers; however, due to the urgency of the clinical situation they agreed to supply the Alfred

Figure 1
Lithium ion battery of the LVAD controller

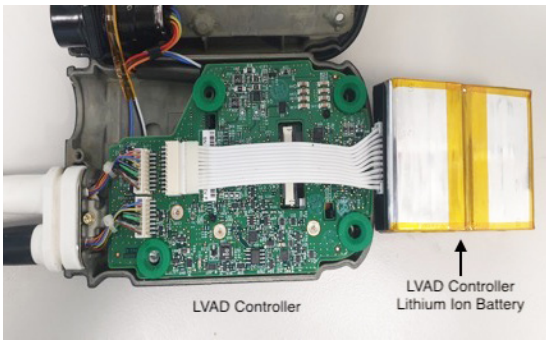


Figure 2
Hyperbaric modified LVAD controller

Figure 3
LVAD controller circuit board with minimum components

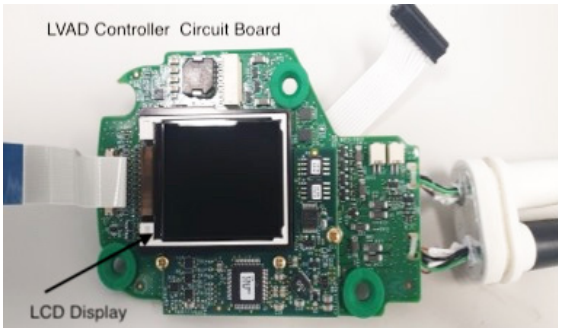
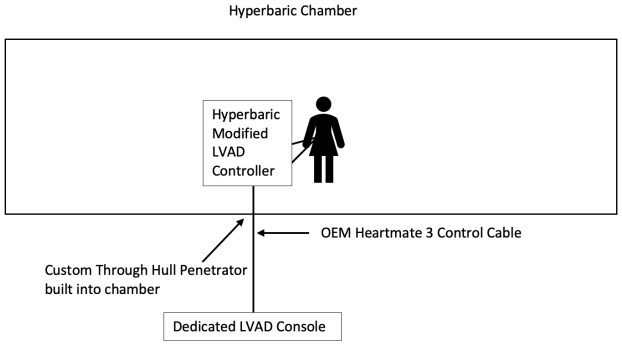


Figure 4
Schematic of patient and hyperbaric modified controller in chamber with components present external to chamber



HBU with a new implantable pump system and supporting documentation to facilitate our evaluation and testing processes. The evaluation included a review of all available documentation that included the operating and technical service manuals. The sample pump unit was subject to a detailed evaluation of its internal components. Discussions were undertaken with Abbott's engineering department and the cardiovascular team at the Alfred, to develop a deeper mutual understandings of the proposed plan for hyperbaric treatment of patients with an LVAD and the options for ensuring continuity of LVAD function. A literature review was also conducted to determine whether previous testing has been reported under hyperbaric conditions.

Power supply continuity is an essential feature of safe LVAD use and this was constrained by the safety related policy of the Alfred HBU that mains electrical power (240 volt AC) is not available in the hyperbaric chamber.

An early conclusion from evaluation of the device was that the controller unit would require modification. The controller's internal 3 min backup lithium battery was assessed as both at risk of failure and a potential fire hazard if repetitively compressed in the chamber. Rather than attempting to rigorously determine if these concerns were valid, a decision was made to remove this battery (Figure 1), with the consequence that the LVAD would only operate if the controller was continuously connected to external power. We removed the batteries from two LVAD controllers which became dedicated for hyperbaric use only as a primary and backup controller. These were clearly identified with 'Hyperbaric Modified' stickers (Figure 2). The controllers were completely stripped down to the bare essentials that would allow us to safely compress them in the chamber (Figure 3).

The department is equipped with highly redundant medical grade, cardiac protected AC supplies, accompanied by an UPS (uninterruptible power supply) to provide power to the LVAD system controller when under pressure from the outside.

A dedicated LVAD chamber penetrator was constructed using an OEM HeartMate III control cable. This custom through-hull penetrator was independently certified to two and a half times the maximum working pressure of the chamber to validate it for pressure and electrical integrity (Figure 4). The cable provided all communication and redundant power to the controller from the outside of the chamber and enabled the Abbott engineers and the VAD coordinators to manipulate controller setting under pressure and download the event logs after each hyperbaric treatment for analysis back at head office. The power provided to the controller via the cable is approximately 14 Volt and 4.8 Amp. In the event of a catastrophic mains power failure and UPS failure, the dedicated VAD console also contained a backup battery system. This provided triple redundancy in the event of a mains power failure.

The removal of the internal battery required a modification to a control circuit to inactivate the normal alarm feature that identifies failure of the internal battery. All other alarms and fault logs were assessed as functioning normally during testing.

Pressure testing was undertaken to evaluate the tolerance of the controllers and a sample pump unit to a maximum pressure of 300 kPa gauge (400 kPa absolute, ~4 atm abs) and to pressurisation and depressurisation rates not exceeding 180 kPa·min^{-1}. The controllers were designated as not to be transferred through the medical lock due to the excessive pressurisation rate, thermal and humidity changes that occur during medical lock operation.

A dedicated flow loop was built which enabled comparison of real time sample LVAD flow measurements with flow output indicated on the VAD controller during various hyperbaric pressure profiles. The controller and pump assembly performed according to manufacturer's specifications during all pressure conditions tested.

Protocols were developed for transfer of the patient to the hyperbaric modified LVAD system in the chamber, with staff training undertaken around both anticipated and emergency operating procedures. For hyperbaric treatments, the patient was transferred from her normal controller to one of the hyperbaric modified controllers prior to her first treatment, and this unit remained in use for the patient for the whole course of her hyperbaric treatment course.

OUTCOME

The patient was successfully treated in our multi-place chamber with no adverse complications or consequences as a result of her HBOT. There were no LVAD functional abnormalities identifies and no alarms were logged throughout the course of 38 hyperbaric treatment sessions.

Hyperbaric treatment reduced, but unfortunately did not eliminate transfusion requirements. Subsequent interventions included discontinuation of warfarin and right iliac artery embolisation. She was eventually discharged to palliative care in May 2020, nine months after her initial admission. At the time of writing this report, she remains well and has only needed intermittent iron and blood transfusions. She has been recommenced on warfarin with manageable HC symptoms.

Conclusion

The HeartMate III LVAD had not previously been tested or certified for use under hyperbaric conditions. To our knowledge this is the first report of the HeartMate III LVAD being used to support a patient undergoing hyperbaric treatment. The safety and management of the patient and her device would not have been possible without the collaboration of the Alfred Hospital's heart failure team, intensive care and key representatives from Abbot Cardiovascular. We believe that our experience has demonstrated a pathway to safe hyperbaric treatment of patients dependent upon a HeartMate III LVAD.

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Successful delayed treatment of acute glans penis ischaemia after adult circumcision: a case report

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Abstract

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Penile glans ischaemia post-circumcision is very rare. A 20-year-old male presented with glans ischaemia following an elective circumcision and was successfully treated with a combination of subcutaneous injection of low molecular weight heparin 0.5 mg·kg^{-1} twice-daily, oral Tadalafil 5 mg once-daily for three days and 12 hyperbaric oxygen treatments at 243 kPa (2.4 atmospheres absolute) beginning 48 hours after the onset of ischaemia.

Introduction

Circumcision is a commonly performed surgical procedure in Australia. A 2010 survey estimated 33% of Australian males under 30 years of age were circumcised.[1] Circumcision is a safe procedure with an associated complication rate of 1% when performed by specialist physicians.[2] Complications include bleeding, wound infection, and cosmetic concerns. Ischaemia or necrosis of the glans penis is considered one of the rarest complications.[3] Reported precipitants for glans penis ischaemia include vasoconstrictor infiltration with local anaesthetics, arterial vasospasm during dorsal nerve block application, excessive use of monopolar diathermy, inadvertent blood vessel ligature, and a tourniquet effect from tight dressings or suture lines.

We present a case of a 20-year-old with acute glans ischaemia following an elective circumcision which, despite presenting 48 hours after the onset of ischaemia, was successfully treated with a combination of subcutaneous injection of low molecular weight heparin (LMWH), oral tadalafil, and hyperbaric oxygen treatment (HBOT).

Case report

The patient gave consent for the case and photos to be presented in this report.

An otherwise fit and well 20-year-old man was referred to the hyperbaric medicine department with dusky discolouration of the glans penis two days following an elective circumcision for phimosis. The operation had been performed by a urological surgeon with a subspecialty interest in andrology and penile surgery and was very experienced in circumcision technique. It was performed via an athermal sleeve technique under general anaesthesia. Bipolar diathermy was only used for haemostasis. Shortly after the foreskin was excised and before sutures were placed, discolouration developed on the glans and residual inner layer of foreskin. The wound was closed with interrupted tension-free 4-0 Vicryl Rapide™ sutures in two layers (dartos and skin). A ring block using 15 ml of 0.75% ropivacaine with no adrenaline was administered following closure of the wound. The blue-purple discolouration progressed over the next hour and was noted to be present on discharge despite no dressing being placed. At all times the glans were warm to touch and the patient expressed no significant discomfort.

The patient was advised to contact the surgeon within 24–48 hours if the discolouration had not improved. At 48 hours, it was persistent and had not changed appreciably compared to the immediate post-operative period (Figure 1). The glans were still warm to touch with no significant discomfort noted. At that point, the patient was referred to the local HBOT unit.

Figure 1
Image of penis pre-treatment with signs of oedema and ischaemia

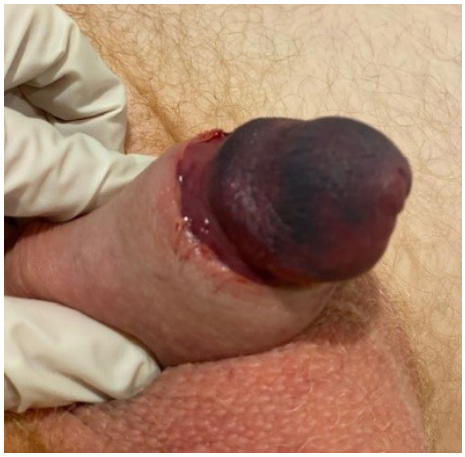


Figure 2
Image of penis immediately following the first HBOT session

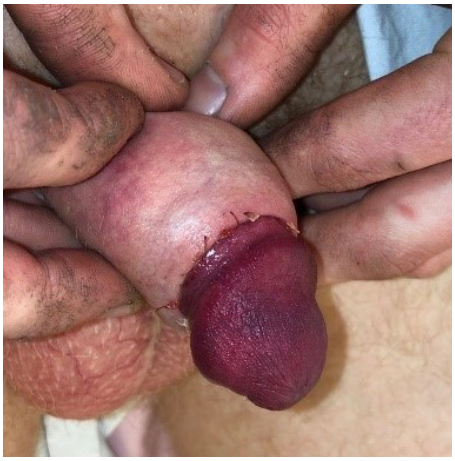


Figure 3
Image of penis following 12 HBOT sessions with residual areas of discolouration

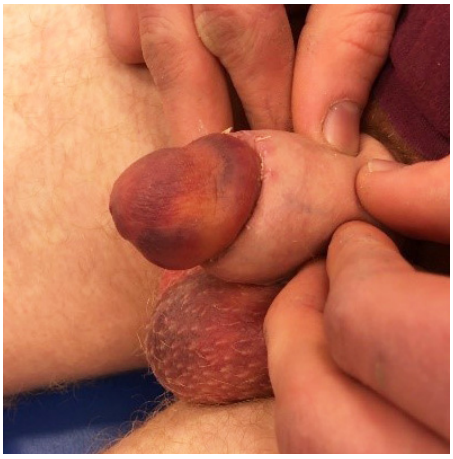
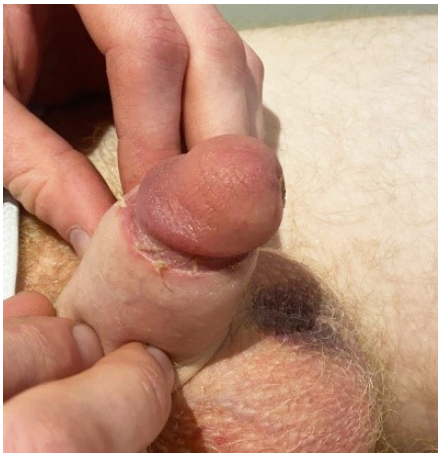


Figure 4
Image of penis at one week clinic follow-up with marked improvement



The decision was taken to commence immediate HBOT with twice-daily 0.5 mg·kg^{-1} subcutaneous LMWH injections (enoxaparin) and once-daily oral 5 mg tadalafil. On breathing oxygen after pressurization to 243 kPa (2.4 atmospheres absolute), the appearance of the glans significantly improved, although the improvement was initially patchy (Figure 2).

The patient received three hyperbaric treatments in the next 24 hours, two the following day with daily treatment thereafter. A total of 12 hyperbaric treatments were given in a monoplace chamber over eight days. Tadalafil was ceased after three days. Twice-daily LMWH was continued until the end of treatment.

At discharge, the patient had residual but much reduced patches of discolouration on the glans with an overall markedly improved appearance (Figure 3). On review in the urology clinic after one week, there was near total resolution of glans discoloration (Figure 4) with no areas of skin loss. The patient reported no erectile or sexual dysfunction.

Discussion

Although penile glans ischaemia is a rare but well described complication of circumcision, the aetiology is unclear. Many of the reports and studies in the literature involve paediatric patients rather than adults. Reported outcomes of glans ischaemia ranges from transient superficial ischaemia to glans necrosis and amputation with subsequent meatal stenosis and urethral stricture.[4] The resultant functional and cosmetic impact to patients may be devastating.

The possible aetiologies of penile glans ischaemia may relate to the use of the local anaesthetic agents, surgical technique, cautery type, and infection.

A dorsal penile nerve block (DPNB) is usually achieved with a ring block or infiltration at the pubic symphysis level or a combination of both techniques. Although DPNB has had a reported complication rate as low 0.23%,[5] subsequent glans ischaemia has been reported.[6,7] Potential causes include large volume local anaesthetic or haematoma formation compressing the dorsal penile arteries, endothelial damage and vasospasm resulting from needle insertion as well as the concurrent infiltration of vasoconstrictor agents with local anaesthetic.

A number of circumcision techniques are described. Clamping techniques, more commonly used in the paediatric population include the Morgen clamp, Gomco clamp or Plastibell devices, whilst open surgical techniques used in any age group include the guillotine technique, dorsal slit or sleeve technique. Less common techniques include thermal energy or laser cutting. Glans necrosis has been reported with cautery using Gomco clamps or an incorrectly sized Plastibell ring.[8] Tight suture lines and compression dressings may exacerbate ischaemia. Monopolar cautery delivers more electrical energy compared with bipolar cautery with reported cases of penile necrosis in paediatric patients following monopolar cautery. A large retrospective study examining 100,157 male paediatric circumcisions performed in US army hospitals reported infection as an uncommon complication (0.06%).[9]

The circumcision technique in our patient was an athermal sleeve technique using bipolar electrocautery with an appropriate volume ring block and there was no haematoma evident. At all times the glans felt warm to touch. The sutures were placed in a tension-free, interrupted fashion without any clinical signs of infection initially and throughout his recovery.

Unfortunately, despite all due care and diligence to reduce the risk of ischaemia, penile glans ischemia still occurred in our patient. Without any readily identifiable risk factors, and no deviation from standard operative technique, the cause of ischaemia in our patient is unknown.

There is a paucity of good evidence-based guidance and consensus on the treatment of glans ischaemia. Reported treatments in case reports and case series include HBOT,[10] phosphodiesterase inhibitors such as pentoxifylline[11] and tadalafil,[12] intravenous alprostadil,[13] antiplatelets, corticosteroids, LMWH, topical testosterone,[14] and caudal anaesthesia.[15,16] One study reported success with LMWH treatment for a paediatric patient with severe glans ischemia 24 hours post-circumcision.[16] Although no adult literature existed for this intervention, we considered it reasonable to treat our patient with LMWH.

We initially commenced tadalafil, a phosphodiesterase-5 (PDE5) inhibitor in our patient based on its reported success in treatment of penile glans necrosis in conjunction with IV pentoxifylline.[12] We ceased this after three doses because of apparent visual improvement in perfusion of the glans penis following HBOT. It is possible that some of the initial improvement in perfusion could be attributed to the PDE5 inhibitor.

There is a large body of experimental and clinical evidence on the use of HBOT to support and enhance the survival of compromised grafts and flaps. Mechanisms of action include reduction of hypoxic insult, enhancement of fibroblast and collagen synthesis, neovascularisation, closure of arteriovenous shunts and positive effects on microcirculation.[17,18] A review on the use of HBOT for flaps and grafts included 957 HBOT patients with 583 control patients encompassing 23 clinical trials (16 controlled trials and 12 randomised controlled trials). The results showed a strong positive result favoring survival in HBOT compared to controls, especially in patients treated 72 hours post-surgery. Of note, there have been reports of success with HBOT in treating ischaemia in other at-risk areas similar to the glans penis. Case reports of necrosis of the nasal areas following trauma or cosmetic hyaluronic filler injection,[19] or amputated finger tips[20] highlight the adjunctive benefit of HBOT in tissue salvage, especially in conjunction with surgery.

Our patient was commenced on treatment 48 hours after the onset of ischaemia. Whilst there is a paucity of published literature on delayed HBOT for penile glans ischaemia, experimental work on animal models demonstrated improvement in the survivability of skin flaps with delayed HBOT.[21] A retrospective analysis showing improvement of ischaemic mastectomy flaps with HBOT had a median time to start HBOT of three days.[22]

Although there are reports of the use of HBOT in conjunction with surgery or pentoxifylline,[10] there are no reports of successful treatment of penile glans ischaemia utilising a combination of HBOT, LMWH and a PDE5 inhibitor. This case demonstrates that despite a treatment delay of 48 hours, this combination of therapies has been associated with a full resolution.

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Hemiplegia resulting from acute carbon monoxide poisoning

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Keywords
Case reports; Hyperbaric oxygen treatment; Neurological manifestations; Recovery of function

Abstract

(Turgut B, Canarslan Demir K, Sarıyerli Dursun GB, Zaman T. Hemiplegia resulting from acute carbon monoxide poisoning. Diving and Hyperbaric Medicine. 2023 June 30;53(2):155–157. doi: 10.28920/dhm53.2.155-157. PMID: 37365135.)
Carbon monoxide (CO) poisoning can cause neurological complications such as movement disorders and cognitive impairment through hypoxic brain damage. Although peripheral neuropathy of the lower extremities is a known complication of CO poisoning, hemiplegia is very rare. In our case, a patient who developed left hemiplegia due to acute CO poisoning received early hyperbaric oxygen treatment (HBOT). The patient had left hemiplegia and anisocoria at the beginning of HBOT. Her Glasgow coma score was 8. A total of five sessions of HBOT at 243.2 kPa for 120 minutes were provided. At the end of the 5th session, the patient's hemiplegia and anisocoria were completely resolved. Her Glasgow coma score was 15. After nine months of follow-up, she continues to live independently with no sequelae, including delayed neurological sequelae. Clinicians should be aware that CO poisoning can (rarely) present with hemiplegia.

Introduction

Carbon monoxide (CO) poisoning can cause neurological complications such as movement disorders and cognitive impairment through hypoxic brain damage. Besides peripheral neuropathy of the lower extremities, a known complication of CO poisoning, hemiplegia due to CO poisoning, is very rare.[1] We present a patient with left hemiplegia caused by acute CO poisoning and whose neurological symptoms were completely resolved after five sessions of hyperbaric oxygen treatment (HBOT).

Case report

The patient consented to her case details being reported.

An 82-year-old female with prior diagnoses of hypertension and Parkinson's disease and who lives alone was found unconscious in her coal-burning stove-heated home by her relatives and transferred to the local hospital's emergency unit. On admission the Glasgow Coma Scale (GCS) was 6 ($E_2V_1M_3$), and her blood pressure was 80/50 mmHg. Arterial blood gas test results were as follows: carboxyhemoglobin (COHb): 36%; pH: 7.18; lactate: 14.4 (mmol·L^{-1}) and the white blood cell count was 21.38 (10^9·L^{-1}).

The patient was not intubated because her peripheral oxygen saturation was 96–98% while breathing oxygen with a reservoir face mask at the rate of 10 L·min^{-1}. Her $PaCO_2$ was 3.54 kPa, respiratory rate was 18·min^{-1} and no pathological respiratory pattern was observed. Her cardiac evaluation was normal. The patient was given a preliminary diagnosis of carbon monoxide poisoning and HBOT was recommended.

In the first examination of the patient at the hyperbaric department, redness and abrasion were observed in the face's left forehead and left cheek area. She had spontaneous respiration, and while breathing 100% oxygen with a mask, her oxygen saturation was 99%, pulse rate was 105–110·min^{-1}, blood pressure was 130/90 mmHg, and GCS was 8 ($E_2V_2M_4$). In the neurological examination the pupils were anisochoric; the left pupil was more dilated than the right pupil. There was no pupillary light reflex on the left side. Additionally, a left hemiplegia was noted with a positive Babinski sign on the left side. Anisochoric pupils and positive Babinski sign suggested central nervous system pathology rather than peripheral neuropathy. There was no response to painful stimulus in the left lower and upper extremities. Bilateral respiratory sounds were natural, and no pathological sounds were heard. The relatives of the patient were interviewed, and it was learned there was no pre-existing mobility issues. Before the patient was accepted for HBOT, a brain computed tomography (CT) scan was performed to exclude intracranial haemorrhage. No pathology was detected on the CT scan. The patient began HBOT at the 5th hour of her admission to the emergency service.

In the examination performed after the first HBOT session she opened her eyes when called by her name, made meaningless sounds, and withdrew to pain. The GCS was 9 ($E_3V_2M_4$). The patient was advised to continue HBOT sessions beginning the next day. In the neurological examination performed after the second session, GCS was 13 ($E_4V_4M_5$); in the motor examination, the strength in the left lower and upper extremities was 0/5, and the pupils were isochoric. Bilateral pupillary light reflex was obtained. Brain diffusion MRI and neurology consultation were requested. No diffusion restriction was detected. Carotid Doppler ultrasound was recommended for differential diagnosis and was normal. No brain pathology was detected in the neurological examination performed by the neurologist. In the examination performed after the third session, GCS was 14 ($E_4V_4M_6$). Pupils were isochoric and the light reflex was detected bilaterally. Left lower and upper extremity motor strength was 2/5. The eyes opened spontaneously and in response to voice. In the examination performed after the fourth session, the motor strength was 4/5; after the fifth session, the motor strength was 4/5, and GCS was 15 ($E_4V_5M_6$). HBOT was terminated after neurological symptoms were completely resolved after five sessions.

Discussion

Carbon monoxide is a colourless, odourless, highly toxic gas with an affinity for haemoglobin 200 to 250 times greater than oxygen. It may cause tissue hypoxia and inhibition of mitochondrial function.[2,3] By reducing oxygen delivery and mitochondrial oxidative phosphorylation, CO can cause ischaemic brain damage and cognitive dysfunction in survivors.[4] Associated excitotoxicity, acidosis, ion imbalance, depolarisation, oxidative stress, nitrative stress, inflammation, and apoptosis can result in brain damage.[5] When cerebral oedema and focal necrosis are seen, more degenerative and demyelinating changes could develop in the brain. The corpus callosum, hippocampus, and substantia nigra are the most affected.[6,7] Demyelination in the cerebral cortex, which has perivascular spread, is a common neurological finding.[6] Also, neuropathy can be seen in peripheral nerves.[7]

Although the brain constitutes approximately 2% of the body mass, it uses 20% of all oxygen taken into the body.[8] The brain needs continuous and sufficient oxygen to perform its functions.[9] For this reason, the brain is extremely sensitive to hypoxia and ischaemia. Hypoxic brain injury, also called hypoxic-ischaemic encephalopathy, is a serious consequence of cerebral ischaemia due to carbon monoxide poisoning or other causes (such as myocardial infarction or cerebrovascular event).[10] Carbon monoxide poisoning may result in serious morbidity and mortality.[11] As a result, deterioration in brain function causes motor, cognitive, behavioral, and functional disorders.[12] In the case we present, we believe that the patient developed left hemiplegia and anisocoria due to hypoxic injury and mitrochondrial impairment with consequent injury in the brain stem. Other common causes of the presentation were excluded by the various investigations performed. Very few cases of hemiplegia due to acute CO poisoning are reported.[1,13] In our case, early treatment with HBOT was associated with a complete recovery. After nine months of follow-up, she continues to live independently with no sequelae, including delayed neurological sequelae. We report this rare presentation of CO poisoning, which can easily be misattributed to other causes, to raise awareness of the possibility among clinicians.

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Obituary – Professor Michael Heywood Bennett AM

It was with great sadness and shock that I received a call on Sunday 30 April advising me of the sudden and unexpected death of our friend and colleague Professor Mike Bennett, AM. Mike was like a father to many of us and to our specialty and was a mentor to a new generation of diving and hyperbaric medicine physicians.

Mike had recently commenced his well-earned retirement, completing his last clinical shift at the Prince of Wales Hospital (POWH), Sydney on the 16 March 2023 – barely six weeks before. He had grand plans for his retirement, cycling around France with his wife Sue and a group of friends to attend the Rugby World Cup as part of their itinerary and making a dent in his collection of cellared red wine. Playing on his new Bocce court (built by him and Sue) at his holiday estate in Mudgee, was high on his 'to do list'. Mike was full of excitement telling me about these plans when we last met at the Australian and New Zealand Hyperbaric Medicine Group (ANZHMG) Introductory Course in Diving and Hyperbaric Medicine Course Dinner in Fremantle in early March. Sadly, none of this will come to pass.

We will miss Mike's ongoing dedication to our field. He had only retired from clinical work but chose to continue his academic commitments. Mike was a giant in the field of Diving and Hyperbaric Medicine and Evidence Based Medicine as detailed in his Bio in the March edition of *Diving and Hyperbaric Medicine*. Read his Bio here.

Mike's academic achievements are too numerous to list again here, so I will cover but a few. He was President of our Society from 2008–2014 and was elected to Life Membership in 2020. His first SPUMS Annual Scientific Meeting was in Rabaul, Papua New Guinea (PNG) in 1994 and he has attended most since, being an enthusiastic diver, presenter and listener. Mike was Scientific Convenor in 2003 (Palau, with Convenor Cathy Meehan), 2010 (Redang Island, with Convenor Glen Hawkins), 2012 (Madang PNG, with Convenor Cathy Meehan), 2013 (Scientific Committee Chair for Tricon, Reunion), 2015 (Palau, with Convenor Cathy Meehan) and 2018 (Scientific Committee Chair for Tricon, Durban).

Mike was a great supporter of our journal *Diving and Hyperbaric Medicine*, authoring numerous research papers and was a longstanding, invaluable member of the Editorial Board. Other notable publications include a chapter 'Hyperbaric and Diving Medicine' in *Harrison's Principles of Internal Medicine*, 21st edition (with Professor Simon Mitchell) and 'Decompression Sickness and Arterial Gas Embolism' in the *New England Journal of Medicine* in 2022 with Simon Mitchell and Richard Moon. He was a co-author and co-editor of the pre-eminent textbook on diving medicine *Diving and Subaquatic Medicine 5th Edition,* along with the late Carl Edmonds, John Lippmann and Simon Mitchell.

Mike's other 'baby' was "*HBO Evidence*" – a web database reviewing all the clinical trials published on diving and hyperbaric medicine. We are endeavouring to find a suitable person to continue this legacy.

Mike was a founding father of the Special Interest Group in Diving and Hyperbaric Medicine of the Australian and New Zealand College of Anaesthetists (ANZCA) and was pivotal in the development and ongoing success of the ANZCA Diploma of Advanced Diving and Hyperbaric Medicine. He was understandably very proud of these achievements, which are crucial to the ongoing growth and development of this specialty.

Mike developed and ran the first ANZHMG Introductory Course in Diving and Hyperbaric Medicine at the Prince of Wales Hospital, Sydney, in 2000 and has taught on every edition of the course since, although he had planned that the February–March 2023 course in Fremantle would be his last. As such, Course Faculty held a 'last supper' in his honour and renamed the prize for the best candidate at each course the '*Unsworth-Bennett Prize*', which Mike was very chuffed with! He loved his annual visits to Fremantle – the town of his birth (across the road from my house, I believe!). As well as teaching on the course, Mike and '*Wilko*' (David

Wilkinson) would spend their evenings in their favourite small bars and restaurants, the owners of which knew them by name.

Mike, along with David Smart, was one of the "*wise men from the east*" whom I frequently had need to contact for advice regarding difficult or unusual cases. They always provided prompt and sage advice.

Since Mike's passing just a few days ago, I have received many emails and calls from members of the diving and hyperbaric medicine community from around the world expressing sadness for his loss. He has left our profession a lasting legacy which we can only hope to emulate and he has left us far too soon.

Mike had an incisive, erudite and witty sense of humour and was a gentlemen and scholar and bloody good fun. On behalf of SPUMS members past and present, I am honoured to be able to say "*Thank you, Mike*" for your enormous contributions, warmth and friendship and to express our deepest sympathy to his wife Sue and his family. Please raise a toast to Mike!

Neil Banham
SPUMS President

Notices and news

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

EUBS2023 Scientific Meeting on Diving and Hyperbaric Medicine

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 major benefit from meeting in person, both during lectures, discussions, in and outside of the meeting room and during social events.

Registration and abstract submission are open now, so please visit the conference website www.eubs2023.com and book your EUBS annual meeting experience as soon as possible.

Even though the 'early bird' registration rates are no longer available, there is still time for you to register for EUBS2023, so go ahead and book your flight/train/hotel and register now. Your friends will be there too.

Ukraine war position statement on EUBS website

While science should be and remain apolitical and non-judgmental of other people's convictions and beliefs, we cannot stand by idly in the face of 'unhuman' (meaning: no respect of human life, dignity and right to autonomy) events happening in the world. We know there is and has always been war, conflict, terror, famine, injustice in many places around us. All of these are worthy of consideration and protest. However, we chose, like many organisations devoted to science and medicine, to specifically let our voice be heard in the case of the Russian-Ukrainian conflict.

"*As scientists, devoted to human wellbeing, we abhor the use of military violence to resolve any conflict, be it political or economic, between free independent states. The Russian attack on Ukraine is a flagrant and utterly unacceptable act of violence and we urge all political and economic leaders worldwide to react strongly with any diplomatic or economic measures necessary to stop this senseless invasion. We are calling upon the Russian Federation and its leaders to end this aggression immediately, and express our undivided solidarity with the people of Ukraine*."

While we hope that by the time of publishing of this issue of *Diving and Hyperbaric Medicine* (DHM), state leaders from the Russian Federation have come to their senses and have stopped military actions to pursue a diplomatic, 'human' solution to the issues perceived, we will continue to have this statement on our website as long as this has not happened. At the time of writing this, the events show (once again) that resorting to the use of military force leads to deviation from moral behaviour and causes horrific suffering and wounds, both physical and psychological that will never heal.

EUBS Elections –Member-at-Large

Around the time of publication of this issue of DHM, the election process for the 2023 ExCom Member-at-Large of EUBS will have started.

We say goodbye to Dr Oscar Camacho (Porto, Portugal) as Member-at-Large 2020. ExCom would like to extend their thanks to Oscar for the work he did with ExCom, even though formal activities of EUBS have been hindered by the COVID pandemic, and, of course, we will still hear from him as he is the Secretary-General of our upcoming EUBS2023 meeting in Oporto.

Candidates for the position of Member-at-Large 2023 will be presenting themselves on the EUBS website with a picture and short CV, and you should by the time this journal issue is published, have received an internet ballot by email allowing you to cast your vote.

If you have not yet received an email regarding voting by the end of June, please notify us at secretary@eubs.org, and we will figure out the reasons why. As the system works via email, it is possible the message ended up in your SPAM folder. There may be other reasons however, usually we are able to solve them.

Professor Mike Bennett has passed away unexpectedly

Losing friends is never easy, and only recently we were again reminded of this sad fact of life.

On Friday, 28 April 2023 Professor Michael H Bennett (30 March 1956) passed away unexpectedly at his home in Australia.

Mike was one of the great personalities of hyperbaric and diving medicine, inspiring, witty, friendly yet rigorous in scientific matters. He was a perfect 'connector' and a fantastic personality. He only recently retired from his academic functions, but had no plans to retire from his advisory roles and engagement in SPUMS and the hyperbaric community in general.

A description of his life, achievements and personality was published in the previous issue of DHM, on the occasion of his official retirement. He will be missed so much.

Website and social media

As always, please visit the EUBS website (www.eubs.org) for the latest news and updates.

On the 'Research page' (http://www.eubs.org/?page_id=284) you will be able to find information on planned and recruiting clinical trials, including one on the use of HBOT for Traumatic Brain Injury in the acute setting.

While we value the membership contributions of all our members (after all, members are what constitutes our Society), EUBS ExCom would specifically like to thank our Corporate Members for their support of our society. You can find their names, logos and contact information on the Corporate Members page under menu item 'The Society'.

Please follow our Facebook, Twitter and Instagram account. While we will continue to use our 'EUBS website news' email messages as a way to communicate important information directly to our EUBS members, Twitter and Instagram is used to keep both members and non-members updated and interested in our society.
Here are the links to bookmark and follow:
Facebook: https://www.facebook.com/European-Underwater-and-Baromedical-Society-283981285037017/
Twitter: @eubsofficial
Instagram: @eubsofficial

ECHM-EUBS Position statement on the use of 'mild hyperbaric therapies' in humans

On 22 December 2022, the European Committee for Hyperbaric Medicine (ECHM) and the EUBS published a Joint Position Statement on the use of 'mild hyperbaric therapies' in humans. The following text, now translated in several European languages, can be downloaded free from the ECHM and EUBS website (http://www.eubs.org/?p=1650).

Introduction

Exposure of humans in hyperbaric treatment devices (hyperbaric chambers) up to 2.0 bar overpressure (equal to 20 meters depth of water) with the breathing of oxygen is known as hyperbaric oxygen therapy, HBO therapy or HBOT.

Due to tragic accidents with human fatalities in the past, different countries established safety regulations regarding technical and personnel standards for the performance of HBOT during the last decades. Within the European Union hyperbaric chambers are regarded as 'Class IIb medical device' according to the Medical Devices Regulation (MDR) and have to meet strict safety standards to prevent harm to patients, caregivers, and third parties.

In the last years, different manufacturers presented new hyperbaric chamber devices using relatively low-pressure e.g., up to 0.5 bar overpressure (equal to 5 meters depth in water). These devices are advertised e.g., as 'low-pressure hyperbaric chambers' for so-called 'mild hyperbaric oxygen therapies' or similar. The pressure exposures are claimed to be beneficial for a wide range of effects and wellness purposes.

With the arguments of different applications and low pressure compared to 'classical medical HBOT', some manufacturers claim to offer their chambers as wellness devices and not medical devices – with no need to meet the MDR standards mentioned above.

Looking at the physical principles, hyperbaric oxygen therapy depends a) on the breathing of enhanced oxygen concentrations and b) on the overpressure during the treatment. The combination of these two conditions is responsible for the treatment effects – for positive therapeutical effects as well as for possible side effects, and possible harm to exposed persons due to elevated oxygen pressure or effects of unplanned pressure changes.

In particular, the fire risk of enhanced oxygen concentrations and the barotrauma risk of unplanned pressure changes do not allow the definition of 'safe' thresholds regarding oxygen concentration or pressure in hyperbaric therapies.[4]

This is the basis for the European Committee for Hyperbaric Medicine (ECHM) and European Underwater Baromedical Society (EUBS) to publish this Joint Position Statement.

Statement 1

The administration of breathing gas in a pressurized chamber, regardless of the construction materials, the pressure used and the concentration of oxygen in the breathing gas, is a medical procedure which carries a certain risk for complications, side effects as well as patient and staff safety issues.

Statement 2

So-called 'mild HBOT chambers', whether they are claimed to be used for the treatment of certain conditions or diseases, or for general claims of enhancing well-being ('wellness', 'to increase energy', 'to rejuvenate', or similar claims), are medical products that must comply with the regulations according to Class IIb medical devices from the Medical Devices Regulation (Regulation EU 2017/745) of the European Parliament and Council (MDR).[1,2]

Statement 3

The operating of devices that can be classified as Class IIb medical devices, if those devices have not been presented for evaluation to the Medical Devices Coordination Group (MDCG)[2], may be punishable by Law, according to Art 113 of the MDR.
National Authorities have adopted appropriate legislation to implement this Art 113. ECHM and EUBS urge hyperbaric experts from those countries that have not yet done so, to call upon their respective governments to implement this as soon as possible.

Statement 4

All hyperbaric chambers (multiplace or monoplace) must comply with European Norms EN14931 (European Standard for Multiplace Hyperbaric Chambers)[3] and EN16081 (Hyperbaric Chambers – specific requirements for fire extinguishing systems)[4] or DIN 13256-4 (Pressure vessels for human occupancy – Part 4: One-human pressure vessels for hyperbaric therapy; Safety requirements and testing).[5] Furthermore, the operation of these chambers should comply with the European Code of Good Practice for Hyperbaric Oxygen Therapy[6] (published by ECHM). Staff should be trained according to the ECHM-EDTC Educational and Training Standards for Physicians in Diving and Hyperbaric Medicine,[7] and the EBAss-ECHM Resources Manual for hyperbaric technicians, nurses and operators.[8]

Statement 5

As a consequence of the requirement to perform a risk-benefit assessment and the identification of possible alternative treatments to achieve the same intended goal, the use of any hyperbaric chamber or therapy should only be proposed for reasonable evidence-based indications. Care providers should have a system in place to monitor possible side effects and assess the efficacy of the treatment (Such obligation is also imposed on the manufacturers of hyperbaric chambers by MDR Annex XIV Part A Section I).

Conclusions

The ECHM and EUBS strongly advise against the use of pressure chambers that do not comply with, or have not been presented for evaluation according to the Medical Devices Regulation of the European Parliament and Council). The use of pressure chambers by any professional medical care provider or in 'at-home' settings not compliant with ECHM – EDTC – EBAss guidelines is not compliant with the Medical Devices Regulation and may be punishable by Law in European member states, according to local legislation.
The ECHM and EUBS do not endorse the use of 'mild hyperbaric (oxygen) therapy' outside the conditions of safety and indications as set forth by the MDR, the ECHM and EBAss.

The ECHM and EUBS strongly advise against the promotion and use of these devices for unverified claims such as 'wellness', 'enhancing energy' or the treatment of diseases for which insufficient clinical, peer-reviewed, scientific evidence exists.

Joint position statement issued on 20 December 2022

Signed on behalf of the EUBS:
Jean-Eric Blatteau, President
Bengusu Mirasoglu, Vice-President
Peter Germonpre, Honorary Secretary

Signed on behalf of the ECHM:
Jacek Kot, President
Alessandro Marroni, Vice-President
Wilhelm Welslau, Secretary General

References

1 Medical Devices Regulation (MDR) [long name: Regulation (EU) 2017/745 of the European Parliament and of the Council of 5 April 2017 on medical devices], from April 5, 2017, valid

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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 'Father' of diving and hyperbaric medicine in Australia Professor Michael Bennett, AM passed away suddenly at the end of April, leaving a gaping chasm in our field and many colleagues both in Australasia and overseas devastated.

On behalf of SPUMS, I offer our sincere condolences to Mike's wife Sue and his family and friends, of which there were many.

A brief obituary appears earlier in this issue, complementing Mike's Bio which was published in the last (March) issue of *Diving and Hyperbaric Medicine.*

I have just returned from the 2023 SPUMS Annual Scientific Meeting (ASM) held at the Crystalbrook Riley Hotel in Cairns, which was a great success.

Conference theme

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

Many thanks to our Convenors Cathy Meehan and David Smart. The presentations from our invited Speakers Ove Hoegh-Guldberg and Professor Craig Johnson were of high quality and very thought provoking- awakening us all to the climate change emergency that is calamitous and happening now in our coastal waters as well as on the land.

There was also a highly productive workshop to develop a SPUMS Position Statement on paediatric diving, which we plan to publish in a forthcoming issue of our journal.

I thank all those that attended and presented.

At the ASM, several positions were up for election, including the Treasurer, Secretary and Education Officer (the most difficult and time-consuming ExCom positions) and five general members. On behalf of SPUMS, I would like to express our sincere appreciation for the hard work and diligence of our Treasurer Soon Teoh and Secretary Doug Falconer who have retired and Education Officer David Cooper who is continuing. Greg van der Hulst, who organised the 2022 SPUMS videoconference ASM has also retired, as has Jen Coleman and Sarah Lockley. I also thank them for their contributions to SPUMS.

Both Soon and Doug were re-elected as general members and will assist our new Treasurer Stephan Roehr and Secretary Ian Gawthrope. Cathy Meehan was re-elected, and we have two new ExCom members: Elizabeth (Lizzie) Elliott and Bridget Devaney. Congratulations to you both and welcome aboard!

Steve Goble formally retired from his duties as SPUMS Administrator at the ASM and his contributions over many years was acknowledged at the ASM Dinner. Steve's role included membership duties, answering or forwarding general queries and maintaining the SPUMS Diving Doctor List. Thanks Steve.

We can now all look forward to our 2024 ASM.

Conference theme

Recreational diving injuries – an update

Keynote Speaker: Dr Peter Wilmshurst
Convenors: David Smart, Scientific Convenor, Neil Banham
Venue: Pearl Resort, Pacific Harbour, Fiji
Dates: 12–18th May 2024

At the 2024 ASM we plan to hold a workshop to review and update the 2015 SPUMS/United Kingdom Sports Diving Medical Committee (UKSDMC) Joint Position Statement on persistent foramen ovale (PFO) and diving which was developed at our 2014 Bali ASM. We are also planning a workshop on immersion pulmonary oedema (IPO) with a view to developing a Position Statement on returning to diving (or not) following an episode of IPO.

More details will be posted on the home page of the SPUMS website as they become available.

The ANZHMG Introductory Course in Diving and Hyperbaric Medicine was again held in Fremantle from

19 February–1 March 2023 and was as always fully subscribed. This course is held yearly only and always fills early, so if you want to register for 2024, don't delay. The Unsworth-Bennett prize for the dux of the course was awarded to Dr Paddy Timms.

The next course dates are 11–22 March 2024, again in Fremantle.

More information available here: 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

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

HBO Evidence is seeking an interested person/group to continue the HBOEvidence site:

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

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

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

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SPUMS Facebook page

Like us at:
SPUMS on Facebook

The Australian and New Zealand Hyperbaric Medicine Group

Introductory course in diving and hyperbaric medicine

Dates: 19 February – 01 March 2024
Venue: Hougoumont Hotel, Fremantle, Western Australia
Cost: AUD$3,200.00 (inclusive of GST) for two weeks

Successful completion of this course will allow the doctor to perform Recreational and Occupational (as per AS/ NZS 2299.1) fitness for diving medicals and be listed for such on the SPUMS Diving Doctors List (provided that they continue to be a financial SPUMS member).

The course content includes:

History of diving medicine and hyperbaric oxygen treatment
Physics and physiology of diving and compressed gases
Presentation, diagnosis and management of diving injuries
Assessment of fitness to dive
Visit to RFDS base for flying and diving workshop
Accepted indications for hyperbaric oxygen treatment
Hyperbaric oxygen evidence based medicine
Wound management and transcutaneous oximetry
In water rescue and management of a seriously ill diver
Visit to HMAS Stirling
Practical workshops
Marine Envenomation

Contact for information:

Sam Swale, Course Administrator
Phone:+61-(0)8-6152-5222
Fax:+61-(0)8-6152-4943
Email: fsh.hyperbaric@health.wa.gov.au
Accommodation information can be provided on request.

The
SPUMS
South Pacific Underwater Medicine Society

website is at
https://spums.org.au/

Members are encouraged to login and check it out! Keep your personal details up-to-date.

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

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

Courses and meetings

DHM Journal Facebook

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

Historical Diving Society Australia - Pacific

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

The Science of Diving

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

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

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

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

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

Below the schedule of upcoming SHF-courses in 2023.

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

2023

9–10 June	In-depth course Nightmares for the Diving Doc (level 2d) The Netherlands
4–11 November	Medical Examiner of Divers part 1 (level 1) Manado, Indonesia
11–18 November	In-depth course Brain under pressure (level 2d) Manado, Indonesia
18–25 November	In-depth course Brain under pressure (level 2d) Manado, Indonesia
On request	Internship HBOt (level 2d certification) NL/Belgium

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

Diving and Hyperbaric Medicine: Instructions for Authors

(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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***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), **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.

Documents on DHM website https://www.dhmjournal.com/index.php/author-instructions

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

Instructions for Authors 2023 (this document)
DHM Keywords 2021
DHM Mandatory Submission Form 2020
Trial design analysis and presentation
English as a second language
Guideline to authorship in DHM 2015
Helsinki Declaration revised 2013
Is ethics approval needed?

DIVER EMERGENCY SERVICES PHONE NUMBERS

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ADSF
AUSTRALASIAN DIVING SAFETY FOUNDATION

Scholarships for Diving Medical Training for Doctors

The Australasian Diving Safety Foundation is proud to offer a series of annual Diving Medical Training scholarships. We are offering these scholarships to qualified medical doctors to increase their knowledge of diving medicine by participating in an approved diving medicine training programme. These scholarships are mainly available to doctors who reside in Australia. However, exceptions may be considered for regional overseas residents, especially in places frequented by Australian divers. The awarding of such a scholarship will be at the sole discretion of the ADSF. It will be based on a variety of criteria such as the location of the applicant, their working environment, financial need and the perception of where and how the training would likely be utilised to reduce diving morbidity and mortality. Each scholarship is to the value of AUD5,000.00.

There are two categories of scholarships:

1. ADSF scholarships for any approved diving medical training program such as the annual ANZHMG course at Fiona Stanley Hospital in Perth, Western Australia.
2. The Carl Edmonds Memorial Diving Medicine Scholarship specifically for training at the Royal Australian Navy Medical Officers' Underwater Medicine Course, HMAS Penguin, Sydney, Australia.

Interested persons should first enrol in the chosen course, then complete the relevant ADSF Scholarship application form available at: https://www.adsf.org.au/r/diving-medical-training-scholarships and send it by email to John Lippmann at johnl@adsf.org.au.
