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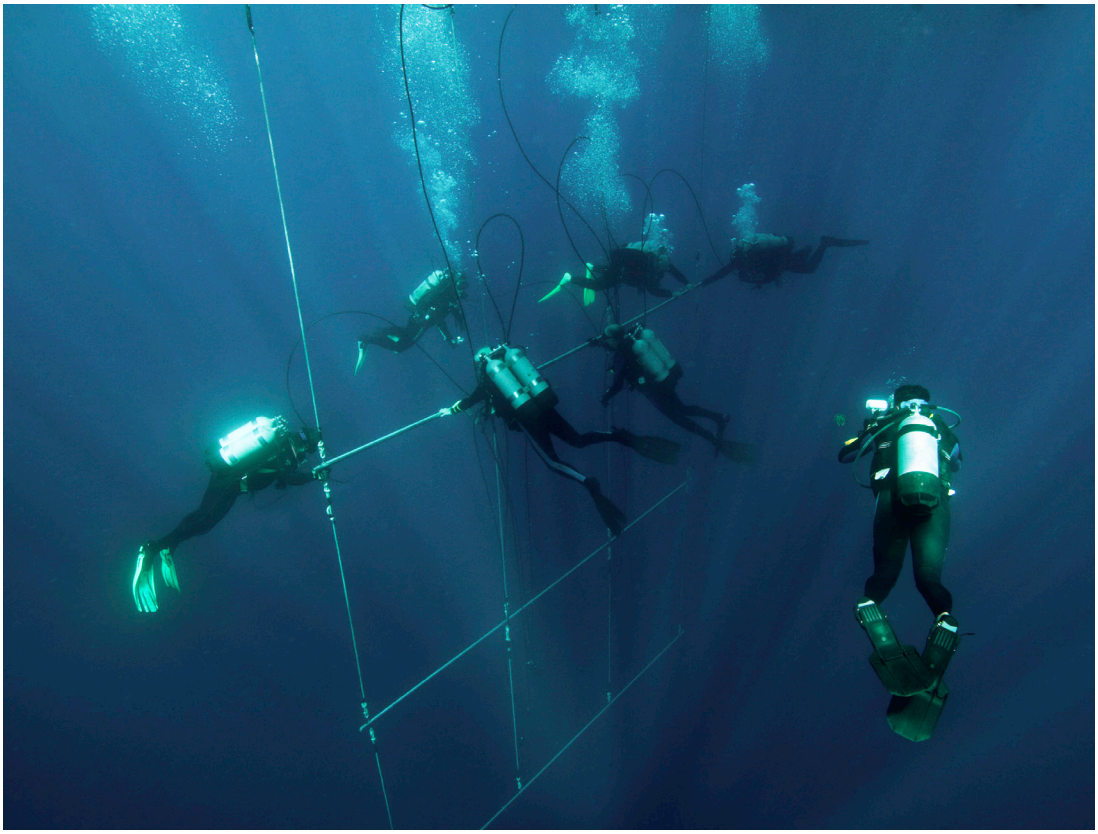

In-water recompression

Air breaks during HBOT in Australasian units

Maxillo-facial problems in diving

Does stored soda lime lose its absorbtive capacity?

Glass drug ampoules tolerate multiple recompressions

Bleak future for diving research in Norway

Variable performance of the Dräger Oxylog® under pressure

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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

Two issues that have been poorly addressed in the hyperbaric literature feature in this issue. Sherlock and her colleagues from Brisbane focus on the diversity of delivery in Australia and New Zealand hyperbaric units of the standard elective hyperbaric oxygen treatment (HBOT) at 243 kPa ('TT14') and the incorporated 'air breaks'.[1] Their data suggest these variations do little to influence the incidence of acute central nervous system oxygen (O_2) toxicity and argue that there is a strong need for standardisation for multicentre research. Another risk factor that needs consideration before the Antipodean hyperbaric community could agree on a standard protocol would be to look at whether the risks of decompression sickness (DCS) in chamber attendants are influenced by these variations which also include differences in the total time at depth and the rate of ascent. For a true epidemiological assessment of this issue, the database would need to be much larger, since the incidence of DCS in attendants appears to be much lower than that for O_2 toxicity. It would be interesting to know whether similar variations occur amongst European units.

In-water recompression (IWR) for DCS in recreational diving has been rubbished by many diving physicians for decades as too dangerous, delaying definitive treatment and potentially worsening outcome. Nevertheless, this technique is used, especially by indigenous commercial and recreational technical diving communities around the world, and there is a genuine need for its consideration for diving emergencies in remote areas. Therefore, the detailed review of IWR by Doolette and Mitchell is invaluable and timely. They present the evidence base for its efficacy and risks and of the historical experimental evidence for shorter, shallower HBOT protocols for DCS generally.[2] Some of the history relating to the evolution of shallow oxygen treatment tables half a century ago makes interesting reading. The evidence of the benefits of prompt, early recompression in DCS are clear from military and commercial experience. However, just what the place is of IWR in modern recreational diving has yet to be established.

DHM as an electronic journal

Traffic on the journal website has steadily increased (19,000 hits and counting) since the new site was launched. More and more members have accessed the March issue and now here is the June publication. Reading DHM is simple – just log onto your society website with your username and password and follow the instructions (slightly different for the two societies). Please also read the brief information text the first time you access the journal – this includes a few words on copyright and reminds you how to scroll around the text if you magnify it. DHM has been set up in as flexible manner as possible. Members of SPUMS and EUBS can:

- Read the journal on line on any smart devices/computers using a magazine-type user interface (including magnifying and scrolling);
- Download a pdf and save it to file in its entirety so it can also be read in pdf format instead of web-based format (you cannot extract pages from this pdf version, only from the on-line version);
- Select specific articles from the on-line version for either saving to file or printing;
- Print the saved-to-device version in its entirety for personal use);
- Select any web or email address and DOI, PMID and PMCID numbers listed in references – these are all now active hyperlinks.

Therefore, the need for members to have access to a separate print copy is superfluous as the reader has greater flexibility of use and many more features than there were with print. Potential options for the future are additional material associated with articles, including data and other appendices, photos and video links. We were tasked by the society ExComs (and by acclimation at an EUBS General Assembly a few years ago) to deliver an electronic journal – we have delivered what was asked for. It is time to retire DHM as a print journal and both I and my successor, Professor Simon Mitchell are disappointed that EUBS has not followed SPUMS in moving entirely to the electronic format. Please let us know what you think of the new format and ways that we might improve it for you.

Breaking news

I am delighted to announce that our application to have articles in DHM stored permanently on PubMed Central® has been successful. This is a huge bonus for authors to have their work stored permanently in the most important international public domain medical database.

References

1 Sherlock S, Way M, Tabah A. Audit of practice in Australian and New Zealand hyperbaric units on the incidence of (CNS OT). Diving Hyperbaric Med. 2018;48:73–8. doi: 10.28920/dhm48.2.73-78. PMID: 29888378.

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

Key words
Editorials; Oxygen; Toxicity; In-water ecompression (IWR); General interest

Michael Davis

Divers decompressing on fixed-depth frames and breathing surface-supplied nitrox after a deep wreck dive at Bikini Atoll. Such a physical set-up could be converted easily to in-water recompression.
Photo courtesy of Dr Martin Sayer.

Editorial

The future of diving research in Norway

Norway has a long tradition of quality research within the field of baromedicine. With the discovery of oil in the North Sea, it became important to establish scientific research facilities to overcome immediate challenges, but also to work towards long-term goals. For the diving community, an understanding of the pathophysiology of decompression sickness (DCS) has been one of the major forces to maintain focus on the importance of scientific research in this field. In addition to oil, the aquaculture and fish farming industries are increasing in size and are Norway's second biggest export industry today. It also requires underwater workers for the inspection and repair of underwater structures and fishnets.

The importance of health and safety for the underwater worker was identified early on by the offshore industry. The Norwegian Petroleum Safety Authority publishes a yearly report that identifies all offshore diving activity. The last reported incident of DCS was in 2002, whilst the last fatal saturation diving accident was in 1987.[1] In-shore diving operations in Norway are regulated through the Norwegian Labour Inspection Authority and here the track record is different; since 1979 there have been 28 fatalities, and they continue to occur.[2]

At the Norwegian University of Science and Technology (NTNU), there has been a research group investigating barophysiology since the early 1980s. Led by Professor Alf O Brubakk, this research has been recognized internationally and has provided ground-breaking insights into the pathophysiology of DCS. This has included the identification of vascular gas bubbles through the use of ultrasound and identifying the importance of both protecting the vascular endothelium to maintain fitness to dive and also regular physical activity to reduce the risk of the adverse effects of diving. The group has educated many students, physiologists, engineers, medical doctors and researchers, all in the spirit of Professor Brubakk who considered that education was at least as important as the research itself.

In 2008, Professor Brubakk was concerned about the future, as he was soon to retire. Great effort was put into perpetuating his position but this process ended when the University axed the only professorial position in environmental physiology in Norway. Today, there is only one non-permanent barophysiology research position at NTNU. This position and all research activity is dependent on external funding, so the education and research environment has changed drastically. Whilst there are clinicians in Norway working at different hyperbaric centres who participate in research related to barophysiology, this is not their primary task. With the lack of funding to include education and students in research, the rich history of barophysiological research at NTNU will be at an end. In Norway, the majority of grant-funded scientific programmes last only three years, so it has not been easy to recruit or to keep expertise between grants.

So, who is planning for long-term research efforts in Norway? Whilst there are obvious challenges left to study in barophysiology, there is a lack of understanding amongst those responsible for decision-making and funding of the importance of having an academic-based research centre for diving research. NTNU, one of the world's most advanced hyperbaric laboratories, built up at considerable capital expense to investigate the pathophysiology of diving and decompression, is about to be closed and dismantled.

At a time when the off-shore industry is putting greater focus on finding better solutions for safer underwater work environments, and in-shore diving is facing huge challenges due to a worrying level of serious accidents and increasing activity, there is no political drive in Norway to acknowledge the importance of maintaining the research facilities that support this industry. If the door does close on the NTNU facility, it will take many years and substantial funding to re-establish a modern research centre. Most importantly, it will be impossible to bring new students into the field of barophysiology in the foreseeable future.

Whilst the off-shore oil industry has a finite future, aquaculture and other in-shore activities requiring diving support continue to expand. Good barophysiological research in established centres will be essential to support these industries into the future. Alf Brubakk often quoted an old Chinese proverb: "*When planning for a month, sow rice, when planning for a year, plant trees, when planning for a decade, train and educate men*". In Norway, we are only planting trees.

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Key words
Decompression sickness; Diving research; History; Research; Editorials

Original articles

Audit of practice in Australasian hyperbaric units on the incidence of central nervous system oxygen toxicity

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Key words
Central nervous system; Clinical audit; Diving tables; Hyperbaric oxygen therapy; Toxicity

Abstract
(Sherlock S, Way M, Tabah A. Audit of practice in Australian and New Zealand hyperbaric units on the incidence of central nervous sytem oxygen toxicity. Diving and Hyperbaric Medicine. 2018 June;48(2):73–78. doi. 10.28920/dhm48.2.73-78. PMID: 29888378.)
Introduction: Central nervous system oxygen toxicity (CNS-OT) is an uncommon complication of hyperbaric oxygen treatment (HBOT). Different facilities have developed local protocols in an attempt to reduce the risk of CNS-OT. This audit was performed to elucidate which protocols might be of benefit in mitigating CNS-OT and to open discussion on adopting a common protocol for Treatment Table 14 (TT14) to enable future multicentre clinical trials.
Methods: Audit of CNS-OT events between units using different compression profiles for TT14, performed at 243 kPa with variable durations of oxygen breathing and 'air breaks', to assess whether there is a statistical diference between protocols. Data were collected retrospectively from public and private hyperbaric facilities in Australia and New Zealand between 01 January 2010 and 31 December 2014.
Results: Eight of 15 units approached participated. During the five-year period 5,193 patients received 96,670 treatments. There were a total of 38 seizures in 33 patients when all treatment pressures were examined. In the group of patients treated at 243 kPa there were a total of 26 seizures in 23 patients. The incidence of seizure per treatment was 0.024% (2.4 per 10,000 treatments) at 243 kPa and the risk per patient was 0.45% (4.5 in 1,000 patients). There were no statistically significant differences between the incidences of CNS-OT using different TT14 protocols in this analysis.
Conclusion: HBOT is safe and CNS-OT is uncommon. The risk of CNS-OT per patient at 243 kPa was 1 in 222 (0.45%; range 0–1%) and the overall risk irrespective of treatment table was 0.6% (range 0.31–1.8%). These figures are higher than previously reported as they represent individual patient risk as opposed to risk per treatment. The wide disparity of facility protocols for a 243 kPa table without discernible influence on the incidence of CNS-OT rates should facilitate a national approach to consensus.

Introduction

Hyperbaric oxygen treatment (HBOT) is defined as "*a treatment in which a patient breathes 100% oxygen whilst inside a treatment chamber at a pressure higher than sea level pressure*". For clinical purposes, the pressure must equal or exceed 142 kPa (1.4 ATA).[1] In Australia and New Zealand, most clinicians treating conditions published by the Undersea and Hyperbaric Medical Society (UHMS) or the Australian and New Zealand Hyperbaric Medicine Group (ANZHMG) use a treatment pressure with 100% oxygen at 243 kPa (2.4 ATA). This is equivalent to the pressure at 14 metres' sea water depth and commonly referred to as a Treatment Table 14 (TT14).

Complications of HBOT include barotrauma, pneumothorax, lung oxygen toxicity and central nervous system oxygen toxicity (CNS-OT). CNS-OT usually presents with prodromal symptoms such as sweating, twitching and tunnel vision, followed by a tonic-clonic seizure. It is most commonly brief and resolves spontaneously once the partial pressure of oxygen is reduced. However, patients are at risk of serious harm during a CNS-OT convulsion.

Air breaks are short periods of breathing air instead of oxygen that have been recommended traditionally to reduce the severity of pulmonary oxygen toxicity.[2] Extended air breaks or extra air breaks may be given with the physiological rationale that the length of exposure to higher oxygen pressures is one of the causes for CNS-OT. Recently

however, air breaks have been postulated as increasing the risk of seizure.[3] There are 10 public hospital and five private hyperbaric facilities in Australia and New Zealand that provide HBOT. Each facility uses a slightly different TT14, the main differences are in the provision of air breaks and total duration of therapy.

This retrospective analysis of data was undertaken to determine if different air-break practices significantly influenced the incidence of CNS-OT. Data concerning indications for treatment were also collected to ensure that similar demographics of patients and risk factors (known and hypothetical) were analysed to assess validity.

Methods

We contacted by phone and email the directors of the 10 public and five private facilities in Australia and New Zealand that provide HBOT inviting them to participate in this study. Nine facilities agreed to participate of which eight were able to contribute to this report. This analysis was deemed to be a quality assurance activity by the Royal Brisbane and Women's Hospital Human Research and Ethics Committee (HREC/15/QRBW/214) and all participating units applied for HREC approval prior to sharing de-identified data.

This is a retrospective cohort study of all consecutive patients who received treatments at eight hospitals in Australia and New Zealand over five years (01 January 2010 to 31 December 2014), examining the incidence of CNS-OT events. Data were collected using the Hyperbaric Technicians and Nurses Association (HTNA) data sets at each hospital. We collected data pertaining to the total number of treatments, treatment pressure, number of patients, conditions being treated and reported cases of CNS-OT. Cases with CNS-OT were analysed to obtain patient-level data. Since our aim was to analyse the effect of different air-break practices on the incidence of CNS-OT in patients treated at 243 kPa we prospectively decided to report events on all cases but restrict the analysis to events while on a 243 kPa TT14.

STATISTICS

The statistical analysis was performed using Stata version 13 (StataCorp, College Station, TX, USA) by an external biostatistician blinded to the hospitals. Means with standard deviation (SD) were used to describe patient characteristics. Poisson regression was used to model the rate of seizure events with hospital as the explanatory variable; Hospital 2 was defined as the reference as it had the most treatments and patients, to determine patient and treatment incidence rates for each hospital. An offset was introduced to account for difference in patient and treatment numbers between hospitals. A P-value of $< 0.05\%$ was considered significant. Hospital 5 and 7 were excluded from the statistical analysis as their incidence rate for CNS-OT was zero at 243 kPa. As this creates a numerator of zero when calculating the incidence rate, this cannot be accommodated in the calculation to compare facilities.

Results

Eight (seven public and one private) of the 15 facilities approached gained HREC approval and participated in the study. All hospitals used slightly different 243 kPa TT14 protocols. Differences included total duration of treatment, duration of 100% oxygen at 243 kPa, and the number, length and total duration of air breaks (see Table 2).

The range of treatment numbers between hospitals was 3,440–19,706, mean 12,083, with Hospital 5 having significantly fewer treatments compared to other facilities. One facility had a notably higher number of treatments per patient (31.5) compared to others, reflecting its chronic wound specialisation, whilst the overall mean number of treatments per patient was 19.

During the five-years, 5,193 patients received 96,670 treatments. There were a total of 38 seizures in 33 patients when all treatment pressures were included in the analysis (243 kPa, 284 kPa and Comex 30 – a helium-oxygen treatment with a maximum pressure of 405 kPa and oxygen partial pressure (PO_2) of 284 kPa). The overall incidence of seizures per patient was 0.039%. These data included emergent treatments of decompression illness (DCI) and toxic gas exposure; these groups of patients are thought to have a higher risk of CNS-OT due to the condition being treated and also a higher treatment pressure.

Table 1

Characteristics of 26 patients with CNS oxygen toxicity at a pressure of 243 kPa; data are shown as number (except age: mean ± SD); risk factors listed only for patients where the information was available; 'Any risk factor' includes those listed here and others, e.g., electrolyte disturbance or fever; ASA – American Society of Anesthesiologists risk grading

Characteristic	Number or mean (SD)
Female/Male	11/15
Age (years)	56.5 (± 2.9)
ASA 1	1
2	8
3	7
4	2
	No/Yes
Diabetes	22/4
Previous epilepsy	17/1
Previous O_2 toxicity seizure	22/4
>1 O_2 toxicity seizure	23/3
Steroids	24/2
Opioids	15/11
Any risk factor	14/12

Table 2

Characteristics of the 243 kPa hyperbaric oxygen treatment tables at the eight hospitals; treatments number and CNS oxygen toxicity (CNS-OT) incidence over a five-year period; some patients had more than one CNS-OT seizure. Total time is the total duration of the treatment from start of compression to end of decompression. Oxygen time is the time spent breathing 100% O_2 at 243 kPa; average treatment numbers rounded

Hospital	Treatments	Patients	Average no treatments	CNS-OT patients	CNS-OT events	Incidence by treatment (%)	Incidence by patient (%)	Time at 243 kPa	No. of air breaks	Duration of breaks	Total air break	Oxygen time	Total time
1	13,046	953	14	3	3	0.023	0.31	75	1	5	5	70	95
2	19,706	1225	16	3	4	0.020	0.24	90	1	5	5	85	110
3	5,701	275	21	3	4	0.070	1.09	90	1	10	10	80	120
4	13,304	927	14	8	8	0.060	0.86	95	1	5	5	90	119
5	3,440	228	15	0	0	0.000	0.00	90	3	5	15	75	120
6	18,788	597	32	4	4	0.021	0.67	90	1	5	5	85	104
7	13,989	515	27	0	0	0.000	0.00	90	2	5	10	80	110
8	8,696	473	18	2	3	0.035	0.42	90	3	5	15	75	110

Nearly half (15 of 33) the patients with CNS-OT had at least one of the commonly described risk factors for CNS-OT such as opiate use or CNS disease. When restricting the analysis to patients treated at 243 kPa there were a total of 26 seizures in 23 patients; three patients had more than one seizure event. The characteristics of these patients are summarised in Table 1.

The incidence of seizure was 0.024% (2.4 per 10,000 treatments, range 0–0.06%) at 243 kPa and risk per patient was 0.451% (4.5 in 1,000 patients, range 0–1.0%). There were no statistically significant differences in the incidences of CNS-OT amongst the different hospitals at 243 kPa TT14. Table 2 describes the variability in TT14 between the eight hospitals in treatment profiles and the incidence of CNS-OT. Table 3 shows the incidence rate ratios per treatment and per patient by hospital, (excluding those with an incidence rate of zero). Figure 1 describes the incidence of CNS-OT events versus the number of air breaks used at the eight hospitals.

Discussion

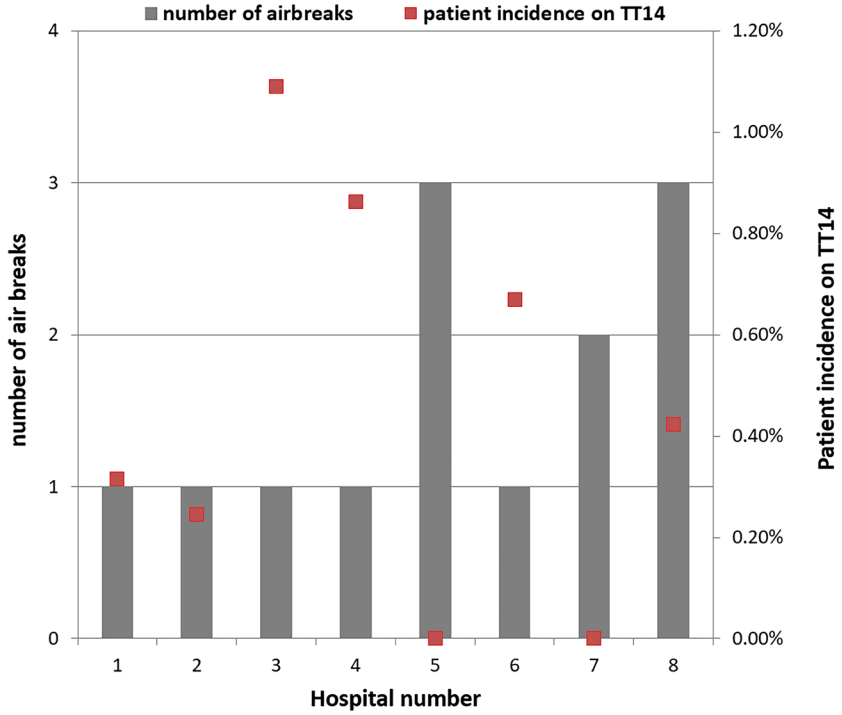
We report the incidence of CNS-OT events in 5,193 patients who have received over 96,000 hyperbaric oxygen treatments. The overall incidence of CNS-OT (irrespective of treatment pressure) when cited as 'risk per treatment' was 0.039% (33 events in 96,670 treatments). This is two-thirds of that reported previously in a single Australian facility (0.06%; 25 events in 41,273 treatments),[4] and 14% of what was reported in a cohort of children receiving HBOT (0.27%; 3 events in 1,099 treatments).[5] When restricting the analysis to treatments at 243 kPa, our cohort reports an incidence of 0.024% or 1 in 4,166 treatments which is 40% of that reported previously (0.06% or 1 in 1,719 treatments).[4]

Previously CNS-OT has been reported as the risk of seizure (numerator) divided by total number of facility treatments (denominator). This is likely to underestimate the risk to the patient in facilities treating chronic conditions with a large number of treatments (e.g., chronic wounds) when compared with emergent indications that receive a lower number of treatments (e.g., decompression illness (DCI)). We have chosen to present the incidence both as the risk per *patient* and the risk per *treatment*, as the former is a more appropriate patient-centred outcome. When receiving information on HBOT, the patient wants to know what is the risk to them. For CNS-OT it is the risk of seizure (numerator) in the population, which is calculated from the number of patients (denominator). This converts the risk in our audit to one per 222 patients (0.45%) as opposed to the higher figure of 0.06% as previously quoted in Australia per treatment (single unit data[4]). This is a relatively low risk when compared to other interventional medical and surgical therapies, e.g., the incidence of stroke after general surgery is reported as 2.9% (29 per 1,000).[6]

Table 3
Incidence rate ratios (IRR) for treatments and patients by hospital. Hospital 2 was the reference hospital as it had the most patients; as such, it does not appear in the table

	Treatments			Patients		
Hospital	**IRR**		***P*-value**	**IRR**		***P*-value**
1	1.13	(0.25–5.06)	0.25	0.96	(0.22–4.31)	0.24
3	3.46	(0.86–13.82)		4.45	(1.11–17.81)	
4	2.97	(0.89–9.84)		2.64	(0.80–8.78)	
6	1.05	(0.26–4.19)		2.05	(0.51–8.20)	
8	1.7	(0.38–7.59)		1.94	(0.43–8.68)	

Figure 1
Incidence of CNS oxygen toxicity and air breaks at the different hospitals (1–8) during a Treatment Table 14 (TT14)



Interestingly, the incidence reported recently for another large retrospective audit in Israel was considerably lower at seven in 62,614 treatments (0.01%). However, the majority (> 57%) of treatments in this report were at a pressure of 151 kPa; which is lower than the minimum therapeutic pressure used in Australia or New Zealand and likely to underestimate the incidence of seizures if applied to our population. In another recent report of a cohort of patients being treated for similar conditions but at the lower pressure of 203 kPa the incidence of seizure was 0.005 % events per treatment or 0.5% events per patient (1 in 200).[7] This is similar to our data at 243 kPa. The same group published a retrospective analysis of all adverse events (including CNS-OT) in the same population as event/treatment as opposed to event/patient in the same year. The overall adverse risk of any complication was published as 0.77% per treatment but would increase to 1.12% if corrected to only include patients treated at > 203 kPa and to give the risk as per patient.[8]

Retrospective audit data for risk of seizure at 243 kPa and 253 kPa reported a risk of 0.04% when results were presented as per treatment and considerably higher (1.07%), when recalculated as per patient.[2] A lower risk of CNS-OT per treatment was published over a decade ago (0.02%); however, this could not be converted to risk per patient as this study did not provide patient numbers.[9] The overall risk of seizure per patient at all pressures in the present audit (inclusive of Comex 30, and all types of 284 kPa profiles) was higher at 0.73% (range 0.19–1.8%) as expected since the risk of CNS-OT increases with increase in pressure.[3]

As consent forms usually list all common or serious side effects of therapy irrespective of treatment pressure, we

recommend that the overall risk of seizures per patient is reported, especially if therapy is at 283 kPa as is often the case for DCI. This risk is considerably higher than previously published. This equates to overall risk of 7.3 per 1,000 patients (almost 1 in 150) per individual.

We found a large variability in TT14 protocols at different facilities, both in terms of time at pressure and oxygen dose, and in relation to number (range 1–3), timing, duration and combined duration of air breaks (range 5–15 min). We did not find any correlation between number of air breaks and the risk of CNS-OT risk and there was no effect of centre on the risk of CNS-OT. This conflicts with previous studies.[3] The two facilities with zero incidences at 243 kPa reported three patients and one patient, respectively, who had a seizure at 283 kPa.

The exact mechanism of CNS-OT is poorly understood but increased cerebral blood flow via nitric oxide (NO) mediated responses is a critical factor and it has been hypothesised that reactive oxygen species may cause neuronal damage.[10] Phosphodiesterase inhibitors (which potentiate endogenous NO) have been implicated in opposing the protective vasoconstriction which is the initial response to hyperbaric hyperoxia.[11] Studies in rats have demonstrated a reduction in dopamine levels in the *substantia nigra pars compacta* which may be linked to seizure activity.[12] Dopamine is reduced in proportion to the increased PO_2. Recent studies in rats looking at striatal blood flow did not support the hypothesis of increased regional blood flow as the pathogenesis of CNS-OT.[13] A recent review suggests that an increased PO_2 saturates protective enzymes and causes neural network overstimulation.[14] Recent studies to develop drugs to reduce CNS-OT have focussed on the effects of pressure on astrocytes and adenosine metabolism which is thought to be crucial in the process of epilepsy.[15,16] Prevention of seizures has become increasingly important with the mounting evidence that seizures may cause cognitive dysfunction and apoptosis.[17,18]

We used seizure as our endpoint for CNS-OT as it is a clear, objective manifestation of toxicity. The experience in our own unit is that the more subjective prodromal symptoms which may precede a seizure are too difficult to confidently call oxygen toxicity. Known risk factors for CNS-OT include medical conditions or medications which are known to decrease seizure threshold.[19] These include electrolyte disturbances, epilepsy, hypercapnia, uraemia, narcotic use, fever and treatment with serotonin reuptake inhibitors. Treatment with corticosteroids has previously been hypothesised to be a risk factor for CNS-OT based on the results of a hypophysectomised rodent model demonstrating increased convulsion thresholds to HBOT.[20] There have been no trials to support the opposite effect (steroids reducing convulsion threshold) in humans. Our report did not find evidence of this association as only two of the patients with CNS-OT were receiving corticosteroids; too small a number for any useful conclusions to be drawn.

LIMITATIONS

Seven facilities were able to provide the condition being treated by broad category although there were discrepancies between total number of patients and patients by category, suggesting either 'off label' conditions being treated, poor data collection methodology or both (discrepancy in patient count ranged from 5 to 597). This highlights the need for an accurate national database, the adoption of which would allow trends in practice to be monitored for adoption of best evidence-based practices. The lack of accurate data in relation to indication for treatment is a flaw of this study; although CNS-OT is an uncommon enough event it is usually accurately recorded.

This audit also collected information regarding indications for HBOT. The facilities that were able to provide indications for treatment appeared to have a similar pool of conditions with the notable exception of idiopathic sudden sensorineural hearing loss. This condition has been on the UHMS indications list since 2014 and was recently endorsed by the European Committee for Hyperbaric Medicine as a valid indication for treatment with Level B evidence.[1,21] Despite this, between 2010 and 2015 two facilities did not treat any patients with this condition, whilst it accounted for 10% of the patient load in another facility. This may reflect a lag in the adoption of recommendations by Ear, Nose and Throat surgeons in different regions at the time of the survey and may not reflect the current situation.[22] A national database would also provide a better understanding of variations in practice.

Eight facilities were able to participate after their ethics committees agreed that this work constituted a quality assurance activity and was exempt from full ethics review. One public hospital facility was unable to participate as their ethics board deemed this to be low risk research and thus would require a lengthy full ethics review. It highlights the inconsistencies between hospital ethics committees when interpreting the National Statement on Ethical Conduct in Research in accordance with the National Health and Medical Research Council Act and institutional differences in governance.[23,24] No explanation was given for non-participation from other units invited to participate.

Conclusions

HBOT is safe and CNS-OT is uncommon. The risk of CNS-OT per patient at 243 kPa was 1 in 222 (0.45%; range of 0–1%) and the overall risk irrespective of treatment table was 1 in 137 (0.73%; range 0.31–1.8 %). These figures are higher than previously reported, as they represent individual patient risk as opposed to risk per treatment. The wide variation in facility protocols for a TT14 without discernible influence on the incidence rates of CNS-OT should facilitate an Australasian approach to consensus. Such consensus would simplify participation in multicentre trials and allow meta-analysis of smaller trials.

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Review articles

Rhinologic and oral-maxillofacial complications from scuba diving: a systematic review with recommendations

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Abstract

(Livingstone DM, Lange B. Rhinologic and oral-maxillofacial complications from scuba diving: a systematic review with recommendations. Diving and Hyperbaric Medicine. 2018 June;48(2):79–83. doi: 10.28920/dhm48.2.79-83. PMID: 29888379.) Rhinologic and oral maxillofacial complications from scuba diving are common, representing approximately 35% of head and neck pathology related to diving. We performed a systematic and comprehensive literature review on the pathophysiology, diagnosis, and treatment of rhinologic and oral maxillofacial pathology related to diving. This included complications due to sinus barotrauma, barodontalgia, odontocrexis, temporomandibular joint dysfunction, partially dentulous patients, and considerations for patients following major head and neck surgery. Of 113 papers accessed, 32 were included in the final synthesis. We created a succinct summary on each topic that should inform clinical decision making by otolaryngologists, dive medicine specialists and primary care providers when faced with pathology of these anatomic sub-sites.

Background

Scuba diving-related injuries in the head and neck are extremely common, and account for 80% of all diving injuries.[1] Approximately 35% of all dive-related head and neck complications occur in rhinologic and oral maxillofacial sub-sites.[2] Despite the prevalence and importance of injuries to these regions, it has been 25 years since the last comprehensive review of the topic.[3] Typically, dive injuries occur due to perturbations of normal physiology according to Boyle's Law, resulting in barotrauma, and Henry's Law, resulting in decompression sickness (DCS). The physics and implications of these laws are outlined in detail elsewhere.[4] The purpose of this report is to systematically review the published literature evaluating scuba diving physiology and complications related to rhinologic and oral maxillofacial sub-sites and provide a resource with evidence-based recommendations where possible.

Method

A systematic review of the literature was performed through a search of the following databases: Ovid/Medline, PubMed, EMBASE, UpToDate, Rubicon Repository, *Diving and Hyperbaric Medicine* publications, and the Cochrane Review Database up to September 2017. A screening literature search was used to identify all literature discussing scuba diving and any otolaryngology topics. Search terms included: "*SCUBA*" and/or "*diving*", and "*head and neck*", "*otolaryngolog**", "*otolog**" "*rhinolog**", "*sinus surgery*" or "*laryngolog**". Reference lists of identified publications were reviewed to ensure no relevant studies in this field were missed. 'Grey' literature, including the Diver's Alert Network online resources, was also queried for completeness. Inclusion criteria included any full text paper discussing scuba diving as it relates to rhinologic and oral maxillofacial anatomic subsites at any level of evidence (LOE). Exclusion criteria included papers that were not available in English or in an English translation.

The combined search resulted in a total of 398 abstracts to be reviewed according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.[5] Two-hundred-eighty-five abstracts were excluded due to duplications, leaving 113 abstracts to be reviewed. Nineteen abstracts were excluded, as they were not available full text or not available in English. Two were excluded because the topic did not include scuba diving. Sixty-nine were excluded as they solely discussed otology-related topics. This left a total of 23 articles that met the criteria of including both scuba diving and rhinology and oromaxillofacial topics. The works cited section of these articles were reviewed, in combination with discussion with experts in the field, and 12 additional studies were identified.

Results and discussion

A total of 32 articles were relevant to rhinologic and oromaxillofacial complications from diving, and were included in the study. There were no systematic reviews, metanalyses or randomised controlled trials found.

RHINOLOGIC PATHOLOGY AND COMPLICATIONS

Disorders of the nose and paranasal sinuses affect up to 18% of divers seeking outpatient ENT consultation.[6] Current evidence shows that chronic rhinosinusitis exists in greater frequency amongst divers, with one group finding an incidence of 11% among divers who presented for otolaryngological assessment.[6] However, study of 76 commercial divers did not show a correlation between radiographic sinus opacification and length of service,[7] albeit a finding of uncertain significance considering the poor sensitivity of plain film sinus radiography. Baseline paranasal sinus mucosa thickness may be greater among divers for uncertain reasons, even among those who have not experienced sinonasal barotrauma; for instance, mucosal hypertrophy was significantly more common among 79 recreational divers in comparison with case-matched controls (42% versus 23%).[8] This may represent thickening due to subclinical dysbaric stress placed on the sinus mucosa. Importantly, these divers were asymptomatic, despite the thickened mucosal lining, and the clinical significance of this finding is uncertain.[8]

Sinus barotrauma

Sinus barotrauma occurs in association with sinus outflow obstruction in the setting of rapidly changing ambient pressure. Sinus obstruction can occur due to many sinonasal pathologies but is most commonly related to acute or chronic rhinosinusitis. The first major series of sinus barotrauma from diving included 50 cases in an Australian Navy environment.[9] In this series of military divers, pain on descent was the predominant symptom in all cases, and in three-quarters of cases on ascent. Epistaxis was the second most common symptom occurring in over half the divers. There was also a strong association with middle ear barotrauma, which occurred in about half the cases.[9] Another series of 50 more severe cases in recreational divers, was self-selected owing to persistent symptoms after diving.[10] Four of these latter 50 divers experienced a dramatic popping sensation at depth, which may have represented haemorrhagic stripping of mucosa of the paranasal sinuses.[4]

The magnitude of barotrauma an individual will experience is related to the size of the sinus ostia, cavities and rate of ambient pressure change. When the sinus mucosal lining of a diver is subjected to a relative vacuum during descent, mucosal oedema, serosanguinous exudation and submucosal haematoma formation may occur.[4] Divers may experience pain, epistaxis and neuralgia within distribution of the maxillary division of the trigeminal nerve. Barotrauma may be limited to specific paranasal sinuses, resulting in a specific subset of symptoms. For example, isolated sphenoidal sinus barotrauma may present with retro-orbital/occipital pain with the absence of significant nasal secretions.[11] The optic nerve can also be affected; one diver experienced blindness due to compression of the optic nerve by a sphenoid sinus mucocoele.[12] Forced Valsalva at depth may also cause cranialisation of pus (and/or air) when diving with acute or chronic rhinosinusitis.[13] Barotraumatic orbital emphysema has been reported in breathhold and scuba divers, which likely tracked through an area of dehiscent *lamina papyracea*.[14,15] Thus, epidural abscess and empyema can occur, and these patients must be followed closely.

Sinus barotrauma may also occur on ascent. As gas expands within an obstructed sinus cavity, vascular compromise leading to mucosal necrosis, and sinus wall fracture leading to pneumocephalus, periorbital/orbital emphysema and meningitis may occur.[15,36] Ischaemic neuropraxia of the maxillary branch of the trigeminal nerve within the maxillary sinus[16] and of the posterior superior branch of the alveolar nerve may also occur, leading to numbness of the ipsilateral teeth, gums, and oral mucosa.[4] It is important to ask about any previous orbital or sinus trauma, in addition to symptoms of chronic rhinosinusitis, as any blocked sinus ostia could generate a closed cavity with potential for barotrauma.[17]

The most important method for prevention of paranasal sinus barotrauma in diving is abstention from diving during an upper respiratory infection, particularly sinusitis or rhinitis.[11] Topical or systemic vasoconstriction is typically contraindicated for 12 h before diving to prevent a rebound congestion and consequent barotrauma during the dive. Patients that present to an otolaryngologist for follow-up after suffering from sinus barotrauma should undergo a thorough endoscopic examination to rule out predisposing anatomic factors, such as septal deviation or nasal polyposis. Computer-aided tomography of the sinuses may be performed to establish persistent sinonasal disease after suffering barotrauma or to rule out pre-existing anatomic factors. Magnetic resonance imaging (MRI) using T1 and T2-weighted imaging can be useful to differentiate blood from mucosal thickening.[18] Endoscopic sinus surgery may be required in patients who have experienced recurrent sinus barotrauma or among those who remain symptomatic despite medical therapy.[19] Among the series of 50 Australian recreational divers published in 1994, six required operative intervention, "*such as sinus … and/or nasal surgery, often with excellent results*".[10]

Treatment of sinus barotrauma is based on the presenting symptoms. Medical therapy can include saline irrigations, use of decongestants, as well as topical and oral corticosteroids.[19] Intractable pain despite maximal medical therapy may require operative intervention, and should be guided by

imaging and a thorough endoscopic in-office examination. Divers may return to diving within six weeks provided imaging demonstrates resolution of sinus opacification and any underlying predisposing factors have resolved (i.e., sinonasal polyposis, coexistent infection or inflammation).[15] These divers should test for pressure-induced headache in a swimming or dive pool at a depth of at least 3 m before resumption of diving activities.[15] Comorbid chronic rhinosinusitis should be treated by an experienced otolaryngologist to ensure maximal patency of sinus ostia, in order to prevent further episodes. Prevention remains the most important clinical consideration, and patients should endeavor to avoid smoking and other nasal irritants, adopt a feet-first position on descent, and utilize frequent and appropriate equalization techniques.[4] Patients presenting with sinus barotrauma will benefit from a thorough otologic assessment to rule out concurrent otologic complications, such as middle or inner ear barotrauma, which can be guided by information contained in a separate review.[20]

ORAL AND MAXILLOFACIAL PATHOLOGY AND COMPLICATIONS

Barodontalgia

Barodontalgia refers to dental pain due to fluctuations in ambient pressure. It has been reported in divers at depths of 10 m (202 kPa) or less.[21] This condition is the most common dental symptom experienced during a dive, with maxillary teeth more frequently involved than mandibular teeth.[22] A 2016 online survey of recreational divers reported that 41% of respondents had experienced dental symptoms at some point during dives.[22] Dry mouth is also extremely common both during and after diving, which may exacerbate pain due to caries.[23] Treatment is aimed at the underlying source of odontalgia, including removal of dental caries, diseased tooth pulp and dental extractions when appropriate. As noted previously, compression of the second branch of the trigeminal nerve from maxillary sinus pathology can also cause odontalgia when diving and should be included in a clinician's differential diagnosis.

Odontocrexis and dysbaric osteonecrosis

Odontocrexis refers to fracture of teeth during ascent or descent. Most often this occurs in teeth that have undergone dental restorations. Air may become trapped at the porcelain metal interface among patients undergoing endodontic procedures. Certain dental cements may also contain microbubbles, specifically crowns luted with zinc phosphate and glass ionomer cements, leading to decreased retentive strength.[37] These cements may undergo volumetric contraction and microleakage, though hyperbaric environments do not seem to promote dental alloy corrosion *in vitro*.[38] Resin cements are relatively unaffected by pressure changes and are advocated among those who undergo exposure to rapid fluctuations in pressure, including divers.[16,39] Repetitive diving may also affect retentive strength of dental adhesives; fibre reinforced composite cements have been shown to have higher strength in comparison with titanium and Zirconia-based cements following simulated dives.[39] Dissolved zinc ions can also be released from low gold content dental alloys in hyperbaric environments, with potential toxicity. High gold content dental alloys are thus advocated among high activity divers.[38] A 2014 survey of 520 Swiss divers and caisson workers demonstrated a prevalence of odontocrexis of 6.3%.[24] Gas emboli due to DCS can theoretically infarct the end arteries within mandible or maxilla causing dysbaric osteonecrosis, though no such cases have yet been reported.[16]

Temporomandibular joint (TMJ) dysfunction (TMD)

Diving regulator mouthpieces are typically silicone rubber and are held in place by a bite platform between incisor and canine occlusion. Typically, the mandible must be positioned anteriorly to properly position the regulator, leading to uneven loading of the TMJ.[16] The lip flanges of the mouthpiece may also cause local gingival irritation, apthous ulceration and trauma. Symptoms of TMD related to diving include pain and fatigue in the TMJ and muscles of mastication, TMJ crepitus or clicking, headache and tinnitus.

Fatigue of the muscles of mastication is common during repetitive recreational diving due to the requirement for prolonged isometric contraction to retain the mouthpiece. MRI studies have demonstrated excessive retrodiscal stress within the TMJ using regulator mouthpieces, leading to worsened TMD.[25] Risk factors for TMD include female gender,[26] inexperience with diving,[27] whilst there is conflicting evidence as to whether cold-water or warm-water diving puts you at greater risk.[28,29] Bruxism also appears to be risk factor, and masticatory occlusal activity may be greater with softer mouthpieces.[26] Other risk factors include clenching, biting on the mouthpiece and a poor-quality mouthpiece.[29] Excessive occlusal pressure on a mouthpiece can also cause non-barotraumatic tooth fracture.[30] Exacerbation of pre-existing TMD is likely. Referred otalgia is common and should not be mistaken for otologic barotrauma.

Custom mouthpieces are recommended for divers with TMD to optimize underwater occlusal forces.[16] There seems to be no significant difference among currently available commercially produced mouthpieces among patients with TMD.[31] Mouthpiece design has been refined to the point that diving may simply be exacerbating pre-existing TMD, rather than causing new cases.[28] Current recommendations include a mouthpiece with an interdental bite platform with a thickness of less than 4 mm and a width less than 8 mm.[27] The interdental bite platform width also affects efficiency of air movement through the regulator, and should be considered in technical diving applications.[32] Cephalometric radiographs assessing jaw position is a

useful adjunct in custom mouthpiece design.[33] Conservative treatment measures include a soft diet, massage and moist heat application during surface intervals between dives. Consideration can also be given to non-sedating oral muscle relaxants and anti-inflammatory medications.[27]

Edentulous/partially dentulous patients

Complete or partial, removable dentures can be a hazard in diving with conventional mouthpieces, though removal of dentures prior to diving is not necessarily required. There have been documented cases of fatal aspiration from a dislodged dental prosthesis during dives.[34] Custom mouthpieces can be fabricated to be retained by edentulous arches. Alternatively, patients may opt for meticulously maintained fixed prostheses or implants. Osseointegrated implants are solid and not at risk for pressure related damage.[16]

Head and neck surgery

Three patients were reported to have successfully returned to diving after extensive head and neck reconstructive surgery. Individual case-by-case assessment involving dive medicine and surgical consultation is essential in such circumstances.[35]

Conclusion

Scuba diving holds significant potential for complications affecting rhinologic and oral-maxillofacial anatomic sub sites. Otolaryngologists and dive medicine specialists should have a thorough understanding of the pathophysiology, treatment and fitness to dive implications of disorders of the head and neck as they relate to diving. The recommendations within this review should be considered in the context of each individual patient.

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In-water recompression

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Abstract

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Divers suspected of suffering decompression illness (DCI) in locations remote from a recompression chamber are sometimes treated with in-water recompression (IWR). There are no data that establish the benefits of IWR compared to conventional first aid with surface oxygen and transport to the nearest chamber. However, the theoretical benefit of IWR is that it can be initiated with a very short delay to recompression after onset of manifestations of DCI. Retrospective analyses of the effect on outcome of increasing delay generally do not capture this very short delay achievable with IWR. However, in military training and experimental diving, delay to recompression is typically less than two hours and more than 90% of cases have complete resolution of manifestations during the first treatment, often within minutes of recompression. A major risk of IWR is that of an oxygen convulsion resulting in drowning. As a result, typical IWR oxygen-breathing protocols use shallower maximum depths (9 metres' sea water (msw), 191 kPa) and are shorter (1–3 hours) than standard recompression protocols for the initial treatment of DCI (e.g., US Navy Treatment Tables 5 and 6). There has been no experimentation with initial treatment of DCI at pressures less than 60 feet' sea water (fsw; 18 msw; 286 kPa; * see footnote) a since the original development of these treatment tables, when no differences in outcomes were seen between maximum pressures of 33 fsw (203 kPa; 10 msw) and 60 fsw or deeper. These data and case series suggest that recompression treatment comprising pressures and durations similar to IWR protocols can be effective. The risk of IWR is not justified for treatment of mild symptoms likely to resolve spontaneously or for divers so functionally compromised that they would not be safe in the water. However, IWR conducted by properly trained and equipped divers may be justified for manifestations that are life or limb threatening where timely recompression is unavailable.

Introduction

Recompression and hyperbaric oxygen (HBO) breathing is the definitive treatment for decompression sickness (DCS) and arterial gas embolism (collectively referred to as decompression illness (DCI)). Ideally, recompression of a diver should take place in the safety of a recompression chamber, but it is also possible to recompress a diver by returning them to depth in the water. The primary motivation for in-water recompression (IWR) is to rapidly treat DCI when a recompression chamber is not readily available. However, during IWR it is not possible to provide other medical care, the patient is exposed to environmental stresses, and a convulsion due to central nervous system oxygen toxicity (CNS–OT) can result in drowning. As a result, IWR is typically conducted at lower pressures, concomitantly lower inspired partial pressures of oxygen (PO_2), and for shorter durations than prescribed by recompression tables used in recompression chambers.

IWR has always been controversial; primarily because it is difficult to evaluate its potential benefits versus its recognized risks. Consequently, although IWR has been reviewed many times with input from the diving medicine community, prominent publications providing guidelines on treatment of DCI generally avoid the subject,[1,2] or are discouraging.[3] Some publications provide guidelines for IWR, generally as a last resort if there is no prospect of reaching a recompression chamber within a reasonable time frame.[4,5]

*** Footnote:** Consistent with the origin of much of the subject matter reviewed, this paper uses the US Navy convention that 33 fsw = 1 atm (101.3 kPa) (US Navy Diving Manual, Revision 7. Washington (DC): Naval Sea Systems Command; 2016. Chapter 2, Underwater Physics; paragraph 2-9.1.). Using this convention, the conversions for fsw to kPa are: 30 fsw = 193kPa; 33 fsw = 203 kPa; and 60 fsw = 286 kPa. Equivalent depths in msw are expressed to the nearest whole number. Where msw are the original unit, this paper uses the convention that 10 msw = 1 bar (100 kPa) (BRd 2806(2) UK Military Diving Manual. Fareham: Fleet Publications and Graphics Organisation. April 2014 Edition. Air Diving. Chapter 6, Decompression; paragraph 0603.g.). Using this convention, the conversions for msw to kPa are: 9 msw = 191 kPa; 10 msw = 201 kPa; and 18 msw = 281 kPa.

There are compelling reasons to revisit this issue. IWR continues to be promoted by some of the world's prominent diving medical experts for use by diving fisherman populations in locations remote from recompression chamber facilities.[5–7] Recreational diving is increasingly taking place in such locations. Moreover, with the increase in so-called "*technical diving*" there are more divers with the requisite equipment and skill mix that might be considered appropriate for conduct of IWR.[8] There is no documentation of how frequently technical divers use IWR, but one technical diving training organization has begun conducting training in IWR methods.[9] Divers suffering neurological DCI are often left with residual neurological problems despite evacuation for recompression.[3,10] There is a widely held belief that early recompression may be associated with better outcomes in such cases and IWR offers an obvious opportunity for very early recompression. It is thus possible to argue for consideration of IWR by appropriately trained divers for serious DCI cases in locations without ready access to a recompression chamber.

This paper begins with a brief review of previous experience with IWR, and a perspective on the relatively negative stance of the medical community over the years. We then address the pivotal issue of risk versus benefit. Most relevant studies do not address the potential benefit of the extremely early recompressions that can be achieved either with a recompression chamber on site or by using IWR methods. Therefore, we will focus on the sparse existing data pertaining to this issue and introduce new data not previously evaluated for this purpose. We will review the evidence that lower pressure and shorter recompressions can be effective in treating DCI when implemented early. The risks of IWR will be enumerated along with potential mitigations. Finally, we briefly discuss diver selection for IWR and potential approaches to its implementation.

Reports of in-water recompression

The fundamental problem bedevilling an objective evaluation of the utility of IWR (and, therefore, its wider acceptance) is a lack of data on cases and outcomes (both good and bad) where the clinical data can be considered reliable. There are a number of reports of apparently good results from systematic use of IWR by particular groups, but in most cases it is unclear how the data were gathered and to what extent there was any objective evaluation of the divers before and after IWR.

In a survey of their diving practices, Hawaiian diving fishermen self-reported 527 IWR events where air was used as the breathing gas, and in 78% of cases there was complete resolution of symptoms.[11] While this seems very positive, there was no independent verification of the severity of these cases, or of the alleged recoveries. Moreover, the apparent success of IWR in this survey approximates the rate for spontaneous recovery from cases of DCI reported in historical data before recompression became considered a standard of care.[12] Thus, while supportive, these data are of limited use in evaluating the efficacy of IWR.

Edmonds described a 1988–1991 study of log books maintained by pearl divers in Australia describing more than 11,000 dives.[13] The sample represented approximately 10% of divers working in the pearl diving industry operating out of Broome and Darwin over the period. There were 56 cases of DCI identified, all of which were treated by IWR on oxygen (O_2), typically at 9 metres' sea water (msw; 191 kPa), and instituted within 30 minutes (min) in most cases. Outcomes were apparently excellent with only one of the 56 requiring evacuation for further recompression in a chamber. It was notable that no cases of oxygen seizure were reported during any of these recompressions. Frequent use of IWR in the Australian pearl diving industry was corroborated by Wong who observed that in the Broome arm of the industry approximately 30–40 cases of mild DCI were treated every year (presumably in the years leading up to his 1996 publication) using IWR with oxygen.[14] However, as with the Hawaiian data, in neither of the Australian series was the severity of DCI or the recovery documented prospectively by competent observers.

Other populations of indigenous diving fishermen have been noted to "*routinely*" employ in-water recompression for DCI. In an observational study of their diving patterns it was reported that sponge divers of the Galapagos described frequent success with IWR on air.[15] However, the investigators only personally witnessed one case treated with IWR, which did not succeed in relieving the symptoms. One attempt has been made to measure outcomes in a small sample of the "*sea gypsies*" of Thailand who typically employ early IWR (within 60 min of symptom onset) using air at depths between 4 and 30 m and for durations between 5 and 120 min.[16] In 11 cases (of uncertain severity), seven had complete recovery, two had improvement at depth but return of symptoms back at the surface and two did not appear to benefit at all.

In 1997, a discussion paper described 16 moderately well documented cases of DCI treated with IWR (Table 1).[17] These cases have qualitative value in illustrating the spectrum of possible outcomes when the technique is employed. Importantly, unlike the poorly documented series involving sea harvesting divers, a large proportion of the cases were known to involve severe symptoms which would not usually be expected to resolve spontaneously. It seems clear from Table 1 that IWR using either air or O_2 appears to have positively modified the natural history of some severe cases. It is also germane that divers involved in two of these incidents (cases 2 and 13–15), but who chose not to be recompressed in-water, died during evacuation, whereas those who recompressed in-water survived. Equally, there were cases (both using recompression on air) where divers either worsened (case 11) or perished (cases 3 and 4) during IWR. Although the numbers are small and firm inferences are not justified, all the cases treated with O_2 could be

Table 1

Summary of data derived from 16 cases treated with IWR;[17] "*up*" implies a staged decompression regimen from the reported maximum depth; "*Severe*" implies potentially disabling neurological manifestations; "*Mild*" implies pain and/or subjective neurological manifestations; some latencies, durations and depth are approximated from the history provided

Case	Severity	Delay (min)	Depth (m)	Duration (min)	Gas	Outcome and notes
1	Severe	<15	18 up	50	Air	Initial relief with recurrence and evacuation for chamber treatment; incomplete recovery
2	Severe	< 30	12	< 120	Air	Complete recovery; buddy who elected not to have IWR died
3, 4	?	< 30	?	N/A	Air	Both divers died, failing to return to the surface
5	Severe	0	24 up	? (> 60)	Air	Complete recovery
6	Severe	< 5	24 up	?	Air	Complete recovery
7	Mild	< 30	12	?	Air	Complete recovery
8	No symptoms	< 5	6	60	100% O_2	Substantial omitted decompression, no symptoms developed
9	Severe	38 hrs	8	~200	100% O_2	Complete recovery
10	Severe	180	9	> 60	100% O_2	Complete recovery
11	Mild	?	?	?	Air	Symptoms worsened and paralysis ensued despite chamber treatment
12	Severe	< 30	15 up	?	Air	Complete recovery
13, 14, 15	Severe	?	12	?	100% O_2	Incomplete recovery in all three after chamber treatment; a fourth diver who elected not to have IWR died
16	Mild	< 90	30 up	90	50% O_2	Complete recovery

interpreted as having reaped some benefit (and no obvious harm) whereas all the poor outcomes (relapses, treatment failures, or fatalities) occurred when air alone was used.

Most recently, a programme designed to educate Vietnamese fishermen divers about safe diving practices and methods for IWR was described and 24 cases of DCI treated with IWR were reported.[7] Ten cases with pain-only symptoms were recompressed by IWR using air, and all had complete relief. There were 10 cases of neurological DCI of which four were treated by IWR using O_2 (9 msw depth for 60 min), all of whom recovered completely. In contrast, only two of the six cases undergoing IWR using air recovered immediately. Thus, like the 1997 series, this account also suggests that IWR using oxygen is more effective than using air.[7,17]

Principal controversies

There has been a long-standing reluctance by peak bodies in diving medicine to recognize IWR as a legitimate option for managing DCI. This reticence is explained by the risks of IWR, and the concomitant lack of medically supervised demonstration of its efficacy. There are a number of potential risks in using IWR (see below) but the use of O_2 as the treatment gas is a major concern since a convulsion due to CNS–OT whilst immersed at depth carries a significant risk of drowning. This concern is greatest if ill-equipped divers with inadequate training and experience attempt to apply the technique. However, with some diving groups being trained to use O_2 underwater it may be time to revise the medical community's attitude to use of IWR by those divers who are demonstrably better trained and equipped for its successful application.

Notwithstanding the case series above suggesting that IWR can be effective, there are no convincing data that it offers any advantage over the safer first-aid alternative of surface O_2. Specifically, what is missing from the above appraisal of the evidence for efficacy is an experimental comparison of outcomes achieved if a diver is simply treated with surface O_2 and evacuated to the nearest suitable hyperbaric chamber (even if this takes some time) versus earlier recompression to modest pressures using IWR. Such experiments are extremely unlikely to ever be undertaken. However, it is possible to make inferences on the efficacy of IWR based on the efficacy of early recompression to modest pressures (key features of IWR) achieved in other contexts. This is discussed in the following two sections.

Efficacy of treatment following short delay to recompression

Since it is theoretically possible to (eventually) evacuate anyone from anywhere to a recompression chamber, the key question is whether there is a threshold delay between onset of symptoms and signs of DCI and recompression (delay to recompression) beyond which prognosis for recovery worsens. There are no prospective studies on the effect of delay to recompression treatment for DCI. A number of retrospective studies report the effect of delay to treatment.[3,10,18,19] A chapter in the *Management of mild or marginal decompression illness in remote locations Workshop* proceedings articulated some of the challenges of conducting such studies, and reasons for variability in the effect of delay to treatment between studies.[19] These included difficulties associated with retrospective review, interaction of symptom severity and delay to treatment and the use of an imperfect outcome measure (full recovery versus presence of residual symptoms and signs).[19]

Analysis of Divers Alert Network data shows a small increase in the presence of residual symptoms after all recompression treatments with increasing delay to recompression for mild DCI. However, as would be expected with mild symptoms, this difference disappears at long-term follow-up.[19,20] These case series analyses were conducted in the context of retrieval of recreational divers, often from remote locations, and the median delays to treatment ranged from 16 to 29 h in different sets of data analyzed.[3,19,20]

Of greater relevance is the effect of delay to recompression in the presence of serious neurological symptoms. Analysis of Divers Alert Network data with divers stratified into a group designated "*serious neurological*" demonstrated a downward inflection (from approximately 60% to approx. 40%) in the proportion of divers making a full recovery after completion of all recompression therapy if the delay to recompression was > 6 h.[3] In another series of 279 divers with spinal DCS stratified according to delay to recompression latency (< 3, 3–6, > 6 h), the percentage of patients making a full recovery at one month follow-up in each group was 76%, 82%, and 63% respectively.[10] It is notable that delay to recompression was an independent predictor of outcome on univariate analysis, but not in a multivariate logistic model which included qualitative descriptions of symptoms and their progress at presentation.[10]

The above data pertain primarily to recreational diving scenarios where even the shorter delays to recompression are measured in hours rather than minutes. Evaluating any advantage of earlier recompression on the basis of such data may therefore underestimate the benefit of very early recompression. Indeed, it is widely believed that very early treatment, such as might occur in commercial or military settings where a recompression chamber is readily available, is likely to result in the best outcomes. Unfortunately, published data, all from the military, are relatively sparse.

In a dataset of military and civilian divers treated for DCS by the US Navy between 1946 and 1961, 885 cases had known delay to recompression.[21] Full recovery after all treatments was 98% or greater in all subgroups of two hours or less delay to recompression (< 15, 16–30, 31–60, 61–120 min) and 95% in divers treated within 3–6 h delay to recompression. Full recovery declined substantially for longer delays. It should be noted that these cases were treated with the US Navy Treatment Tables 1–4, before the development of the minimal-pressure O_2 breathing US Navy Treatment Tables 5 and 6 (USN TT5 nd USN TT6),[22] which have since become the standard of care. In a recently reported smaller case series of 59 military divers with neurological DCI who were treated a median of 35 min (range 2–350 min) after symptom onset, the odds of incomplete recovery at one month follow-up increased with delay to recompression. However, it is not possible to interpret the magnitude of this effect as it is not clear if delay was treated as a continuous variable in the logistic regression.[23]

A particularly high success rate for treatment of DCI is reported from US Navy diver training and experimental diving facilities, where, as a result of heightened vigilance among divers, close medical supervision and ready availability of recompression facilities, treatments are usually initiated within two hours of symptom onset. Fifty consecutive cases of DCS occurring at the Naval School of Diving and Salvage from 1975 to 1978 were treated with a single USN TT5 or 6 (eight Tables were extended), and 46 of these were recompressed within two hours of symptom onset.[24] Forty-nine patients reported complete relief of symptoms shortly after compression to 60 feet' sea water (fsw) (286 kPa, 18 msw) at the start of the treatment. One patient had residual arm soreness after a single recompression that resolved spontaneously over five days. In another series, 292 Type I DCS cases were treated at the Naval Diving and Salvage Training Center and Navy Experimental Diving Unit (NEDU) with a single USN TT5 or 6.[25] The delay to recompression is not given but is presumably short. Two hundred and eighty patients (96%) had complete relief after a single recompression. In a third series, 166 cases of DCS arising from experimental dives at NEDU and the Naval Medical Research Institute (NMRI) were recompressed from 1980–1989.[18] USN TT5 or 6 were used in all but four cases (two Treatment Table 4s, one Treatment Table 7 and one 60 fsw saturation treatment) and there was "*little or no delay between symptom occurrence and treatment*". One hundred and nineteen cases (72%) resolved during compression or within the first 10 minutes at depth during the first recompression treatment, 161 cases (97%) had complete resolution of DCS at the end of the first recompression treatment and all resolved eventually.

In addition to the above, we have collated reports of 140 cases of DCS arising from experimental dives at NEDU, NMRI, and the Naval Submarine Medical Research Laboratory from 1988 to 2006 in which delay to recompression and details of the clinical course are available.[26–33] Up to 16 of

Figure 1

Symptoms and signs of 140 cases of DCS arising from experimental dives at three US Navy research facilities from 1988 to 2006 (see text for Tier classification); "*Paralysis/Weakness*" includes motor weakness, whereas "*Weakness (with pain)*" is weakness associated with a painful joint; "*Girdle/Abdominal Pain*" includes bilateral hip pain; "*L.O.C.*" – loss of consciousness; "*S.O.B.*" – shortness of breath. "*Joint pain*" refers to classic musculoskeletal pain in the vicinity of a joint; "*Nausea*" is without vertigo and vomiting

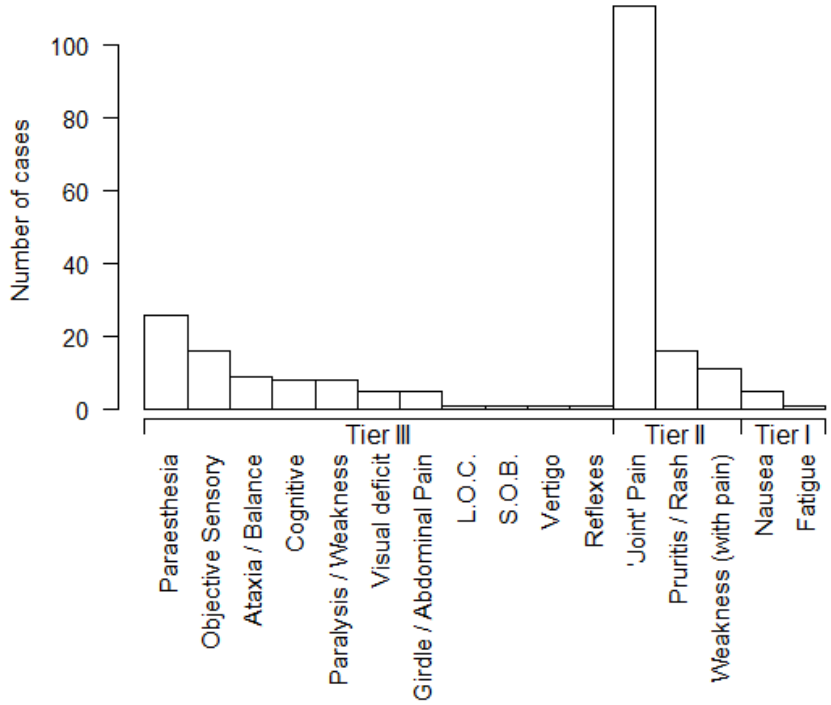


Figure 2

Delay to onset of symptoms and signs after surfacing from diving in 140 cases of DCS arising from experimental dives at three US Navy research facilities from 1988 to 2006; five cases with symptom onset before surfacing are included in the first bar

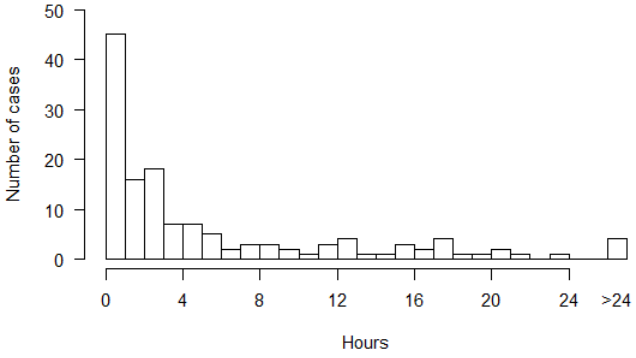
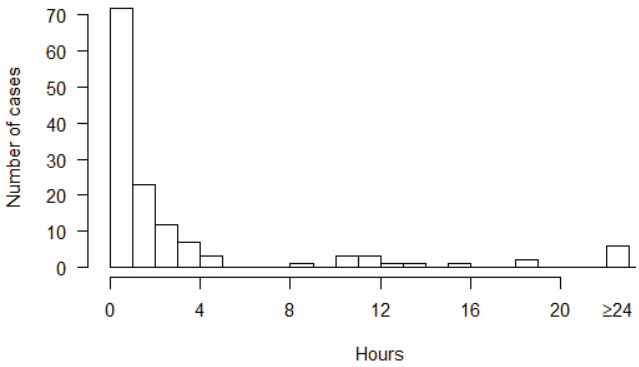


Figure 3

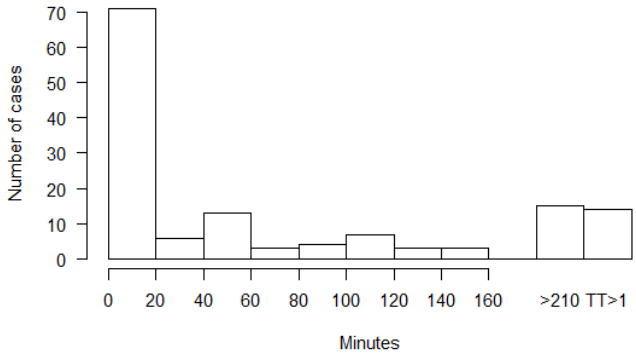
Delay to recompression after onset of symptoms and signs in 140 cases of DCS arising from experimental dives at three US Navy research facilities from 1988 to 2006



these cases may overlap with those previously reported.[18] Figure 1 shows the frequency of symptoms and signs in these 140 cases arranged into the 'tiers' that comprise a published diver selection algorithm for IWR (Table 2).[9] Figure 2 shows the delay to onset of symptoms and signs after surfacing. Figure 3 shows the distribution of delays to recompression.

The median delay to recompression was one hour. The majority of cases (87%) were treated within five hours of symptom onset. The initial recompression treatment was USN TT6 (with or without extensions) in 122 cases, USN TT5 (with or without extensions) in 16 cases, US Navy Treatment Table 7 and Comex 30 in one case each. Seventy-one cases (51%) resolved during compression or during the first 20-minute oxygen breathing period at 60 fsw, and 126 cases (90%) had complete resolution of DCS after the first recompression treatment. The distribution of times to resolution of symptoms or signs during recompression is shown in Figure 4. In 14 cases, complete resolution of DCS

Figure 4
Time to resolution of signs and symptoms during recompression in 140 cases of DCS arising from experimental dives at three US Navy research facilities from 1988 to 2006; times are generally from the beginning of oxygen breathing at treatment depth, but the first bar includes resolution during descent; the last bar indicates the number of cases that required more than one recompression treatment to achieve complete resolution of symptoms



required two to five recompression treatments (number of treatments/number of cases: 2/6; 3/3; 4/2; 5/1; 9/1; 14/1). There was nothing notable about the clinical presentation in the cases requiring multiple recompression treatments; the median delay to recompression was 0.44 h (range 0–94 h) and although nine of the 14 cases were severe DCS, twice as many severe DCS cases resolved with a single treatment.

There is insufficient variation in the times to treatment or outcomes in these US Navy training and experimental dives to identify an effect of time to treatment, but the efficacy of a single recompression in these data are in contrast to reported experience among mainly civilian divers.[10,34] For instance in a large contemporaneous case series of 520 mainly recreational divers, the median time from surfacing to treatment at a civilian recompression facility was two days and requirement for multiple recompression treatments was common (mean number of re-treatments = 1).[34] In this same case series, excluding those lost to follow-up, 438 (88%) divers had complete recovery after all treatments and 61 (12%) had incomplete recovery, a significantly lower proportion of complete recovery than in the 140 military experimental divers ($P < 0.0001$, two-sided Fisher's exact test). Collectively, the military data signal that delay to recompression of two hours or less is associated with a good prognosis for full recovery.

Shallow and short treatments

To manage the risk associated with IWR, recommended protocols typically involve recompression to a maximum depth of 30 fsw (193 kPa, 9 msw) breathing O_2 for 3 h or less (this is further discussed in the 'Risks and mitigation' and 'In-water recompression protocols' sections below). Figure 4 indicates that 88% of cases in our new series had complete resolution of signs and symptoms within 3 h after a short delay to recompression. This is an encouraging statistic for IWR, but since Figure 4 describes outcomes from principally USN TT5 and 6, it is possible that symptoms and signs may not resolve as quickly with HBO at shallower depths and may recur during decompression from shorter treatments.

Since the introduction of the USN TT5 and 6,[22] treatment tables which prescribe O_2 breathing at 60 fsw have become the standard of care, and there has been essentially no experimentation with treatment tables beginning at shallower depths or for shorter durations for the initial treatment of DCI. However, it is not widely appreciated that the development of these two tables included testing of treatment at both 33 fsw (203 kPa, 10 msw) and 60 fsw for relatively short durations.[22]

In these test programmes the "*provisional*" protocol was to compress divers breathing O_2 to 33 fsw and, if complete relief of symptoms occurred within 10 min, O_2 breathing was continued at this depth for 30 min after relief of symptoms and during decompression to the surface at 1 fsw·min^{-1}. If relief was not complete within 10 min at 33 fsw, divers were compressed to 60 fsw. If complete relief of symptoms occurred within 10 min at 60 fsw, O_2 breathing was similarly continued at this depth for 30 min and during 1 fsw·min^{-1} decompression to the surface. The test report tabulates 31 shallow recompression treatments that generally followed these rules:[22] 27 at 33 fsw, three at 30 fsw and one at 20 fsw. Seven treatments had longer time at maximum depth than specified above. Excluding one 26-h treatment, the total treatment times ranged from 35 to 180 min (mean 70 min). DCI signs and symptoms treated at 33 fsw or shallower (number of cases) included pain (26), special senses (6), rash (5), sensory (3), chokes (3), syncope (3), motor weakness/paralysis (3), loss of consciousness (1) and nausea and vomiting (1). Being largely treatments for experimental dives, the delay to recompression was relatively short, with a median of 37 min (range 0–270 min). It is perhaps pertinent that many of the inciting dives were non-trivial, including trimix bounce decompression dives to 200–400 fsw (61–122 msw), direct (no-stop) ascents from shallow 12-h sub-saturation exposures, and repetitive air decompression dives to a maximum of 255 fsw (78 msw). Twenty five of these 31 shallow treatments resulted in complete relief. Two treatments resulted in substantial relief; in one case the residuals are reported to have resolved spontaneously over three days. Four treatments were followed by recurrence of symptoms; in three cases complete relief was reported following a second treatment.

The report also tabulates 56 recompression treatments deeper than 33 fsw, mostly at 60 fsw.[22] There are three treatments which included compression to 165 fsw (50 msw) for relief of symptoms, two of these were followed by O_2 breathing at 60 fsw and one by O_2 breathing at 30 fsw. There are two treatments with an initial compression to 165 fsw that appear to be US Navy Treatment Table 6A (USN TT6A).

Collectively, these 56 deeper recompressions resulted in complete resolution of symptoms and signs in 53 cases. This just fails to reach statistical significance in comparison to outcomes of the shallow treatments ($P = 0.0653$, two-sided Fisher's exact test). Fewer than half of the 56 deep treatments represent failure to obtain complete relief within 10 min at 33 fsw in accord with the provisional protocol. Seven of the 56 deeper treatments had relief of symptoms shallower than 33 fsw but nonetheless continued to 60 fsw. The initial evaluation for relief of symptoms at 33 fsw was discontinued at an unspecified point in the test programme. Twenty-three of these treatments without the initial evaluation at 33 fsw can be identified with reasonable certainty: 13 treatments appear to follow the provisional 60 fsw treatment time, eight treatments appear to be the 'final format', i.e., USN TT6, and two appear to be the aforementioned USN TT6A.

Typical IWR protocols are of relatively short duration, comparable to the USN TT5 which lasts 135 min if not extended. As has been described earlier, USN TT5 is highly effective in the treatment of mild DCI when delay to recompression is short.[25] However, USN TT5 has a reported high rate of treatment failures for neurological DCI, albeit with significant delays to treatment in some cases.[35] Others report better success with short treatment tables for all manifestations of DCI, and with substantial delay to recompression (median 48 h): the 150-min, no-air-break Kindwall-Hart monoplace treatment table, which is similar to the progenitor of the USN TT5,[22] resulted in full recovery after all treatments in 98% of 110 cases of DCS.[36] In addition to providing evidence for the efficacy of relatively shallow recompression in cases recompressed relatively quickly, the US Navy treatment table development data presented here demonstrate the efficacy of the relatively short provisional protocol; the median total duration of their 87 treatments was 105 min, and 64 (74%) of the treatments were shorter than 135 min.[22]

Risks and mitigation

The potential benefit of earlier recompression using IWR must be balanced against its risks. These risks and their potential mitigations are relatively well understood and have been discussed extensively elsewhere.[9,17,37]

OXYGEN TOXICITY

A major risk of IWR is CNS–OT. This can manifest as a seizure, often without warning, and such an event underwater carries a significant risk of drowning. Seizure risk is a function of dose (inspired PO_2 and duration). The inspired PO_2 threshold below which seizures never occur irrespective of duration has not been defined but it is lower than exposures recommended for IWR (typically breathing 100% O_2 at 9 msw depth). Thus, while we are not aware of any reports of an oxygen toxicity event during IWR, seizures have certainly occurred in O_2 exposures of similar magnitude. In experimental O_2 dives (> 95% O_2 by rebreather), convulsions have not been observed at 20 or 25 fsw (163 or 178 kPa, 6 or 8 msw), but seven "*probable*" O_2 toxicity symptoms (nausea, dizziness, tingling, numbness, tinnitus, dysphoria) occurred in 148 man-dives to 20 fsw for 120 to 240 min duration while performing mild exercise (equivalent to 1.3 $L \cdot min^{-1}$ VO_2).[38] Under otherwise the same experimental conditions, no symptoms of CNS–OT occurred in 22 man-dives to 25 fsw for 240 min.[38] In other experiments, six divers developed symptoms of CNS–OT in 63 man-dives to 25 fsw of 120–240 min duration (reviewed in ref[39]). Convulsions have been observed during 30 fsw resting and exercising O_2 dives; in 92 man-dives of 90–120 min durations, three convulsions occurred at 43, 48, and 82 min, respectively.[39–41]

The actual risk of an O_2 seizure during IWR using O_2 cannot be usefully extrapolated from such studies because an individual's risk is so context sensitive. In this setting "*context*" refers to many factors such as individual susceptibility (which appears to vary widely), exercise (higher risk) versus rest (lower risk), and CO_2 retention (higher risk). It is also notable that divers undertaking IWR will have an immediately prior exposure to elevated inspired PO_2 which would increase risk. In the case of a technical diver this exposure may be substantial.

Mitigation of the risk of CNS-OT can focus either on preventing such an event, or lessening the risk of complications if one occurs. In relation to prevention, there is an obvious tension between the goal of safely increasing pressure to achieve bubble volume reduction, and the safety of the inspired PO_2. Arguably the most effective way of reducing the likelihood of a seizure is to reduce the inspired PO_2 into a range where seizures seem rare. While most IWR protocols recommend recompression breathing O_2 at 30 fsw (9 msw), for a limited time, a reduced risk of O_2 toxicity could be achieved by limiting oxygen breathing to lower pressures. For example, the protocol taught on the International Association of Nitrox and Technical Divers IWR course prescribes the vast majority of time to be spent at 25 fsw.[9]

Mitigating the risk of a seizure, if it occurs, centres primarily on protecting the airway. This can be achieved (though not guaranteed) through the use of a full-face mask, or a mouthpiece retaining strap.[42,43] Other key risk management strategies include tethering the diver to a decompression stage throughout the recompression so that they cannot sink in the event of loss of consciousness, and ensuring that the diver is accompanied at all times so they can be rescued immediately to the surface if a seizure occurs. Rescue of a seizing diver is discussed elsewhere.[44]

ENVIRONMENTAL FACTORS

Divers requiring IWR risk becoming cold or even hypothermic. In technical diving scenarios, they may already have completed long dives in cold water. On the plus side, the use of dry suits is common among these

divers, and so is the application of active heating systems in dry suit undergarments. It is beyond the scope of this review to discuss thermal considerations in detail, but this is a factor that must be taken into account in deciding whether to undertake IWR. IWR requires a stable platform that can remain in one place for three hours. Changes in environmental factors like weather, current and light can all potentially cause disruption to an IWR process, and projections of these factors should be taken into account in deciding whether to undertake IWR.

PATIENT DETERIORATION

It is well recognized that divers with DCI can deteriorate clinically despite (and indeed during) recompression. Such deterioration during IWR, particularly in respect of consciousness, could represent a very real threat to safety. This threat can be mitigated by careful selection of patients for IWR (see below and Table 2), and ensuring that a patient is accompanied at all times, so that the procedure can be safely abandoned and the patient assisted to the surface in the event of deterioration. Limiting the depth of recompression and use of equipment that helps to protect the airway, such as a full-face mask or mouthpiece retaining device, are also useful mitigations in this context.

A related question is whether IWR itself can be the cause of a worse DCI outcome. This is an issue sometimes raised in respect of using air for IWR. Although recompression on air will produce an initial compression of bubbles, and possibly a related clinical improvement, bubbles will dissolve more slowly and more inert gas will be taken up into some tissues. Persistent bubbles will re-expand and possible take up more gas during decompression, with a possible recurrence or worsening of symptoms. Such mechanisms may help explain outcomes such as those in cases 1 and 11 in Table 1. This argument along with the corroborating observational evidence of weaker efficacy if IWR is conducted on air[7,17] probably constitutes adequate justification for recommending that O_2 is always used and air be avoided.

DIVER SELECTION

One of the most vexing challenges of IWR is the selection of DCI-afflicted divers whose condition justifies the risks of IWR and whose clinical state does not contraindicate it. There is no agreed formula for such determinations. The risk of IWR may not be justified for those cases where the natural history of the symptoms is for spontaneous recovery irrespective of whether the diver is recompressed or not. The findings of the UHMS 2004 remote DCI Workshop provide some guidance on how a "*mild DCI*" presentation that might not justify the risks of IWR could be defined.[45] The symptom constellation comprising the mild syndrome was one or more of musculoskeletal pain, rash, subjective sensory change in a non-dermatomal distribution, and constitutional symptoms such as fatigue. The workshop concluded that divers with presentations limited to these symptoms could be adequately managed with surface oxygen and careful observation after discussio with a diving physician. It could therefore be argued that exposing divers with static mild symptoms to the risks of IWR might not be justified. At the other extreme of severity, IWR should not be undertaken if the diver is so compromised that they would not be safe in the water. In between these extremes, there will be many potential presentations, and decisions may not be straightforward. Decisions about which cases to recompress in water are likely to be nuanced and difficult to codify in rules.

In an attempt to bring some structure to the decision-making process around IWR for divers in the field, (and following a consensus meeting with expert diving medical input) the International Association of Nitrox and Technical Divers recently categorized potential DCI symptoms into "*tiers*" (Table 2).[9] These lists are intended to be sufficiently descriptive as to allow application by divers without medical training, and their application relies on history or gross observation alone, as opposed to a more detailed neurological examination as might be conducted by someone

Table 2

Symptom severity 'tiers' for triage of DCI for IWR adapted from the International Association of Nitrox and Technical Divers in-water recompression course for technical divers[9]

Tier I: Non-specific symptoms that may not be DCI and do not represent a significant threat:
Lethargy
Nausea
Headache

Tier II: Symptoms and signs likely to be DCI but unlikely to result in permanent injury or death irrespective of treatment:
Lymphatic obstruction (subcutaneous swelling)
Musculoskeletal pain (excluding symmetrical "*girdle pain*" presentations)
Rash
Paraesthesias (subjective sensory changes such as "*tingling*")

Tier III: Symptoms and signs likely to be DCI and which pose a risk of permanent injury or death:
Changes in consciousness or obvious confusion
Difficulty with speech
Visual changes
Walking or balance disturbance
Sensory loss (such as numbness) that is obvious to the diver or examiner
Weakness or paralysis of limbs that is obvious to the diver or examiner
Bladder dysfunction (inability to pass water)
Sphincter (bowel) dysfunction
Loss of coordination or control in the limbs
Shortness of Breath
Girdle pain syndromes (such as both hips, abdomen, or back)

with medical training. The tiers conform approximately to both perceived severity and levels of justification for IWR to provide a guide to the appropriateness of the intervention. Thus, divers with only Tier I symptoms would not justify IWR. That is not to say that the symptoms should be ignored. The diver should be carefully monitored and perhaps discussed with a diving medicine authority, but they would not justify IWR unless the symptoms progressed beyond Tier I. At the opposite end of the spectrum, divers with Tier III symptoms or signs do justify expeditious IWR provided the logistic requirements for IWR are met and there are no contraindications. Divers with Tier II symptoms present the greatest challenge. Where a diver reports Tier II symptoms some hours after surfacing and where those symptoms are not progressive, the risk of IWR is probably not justified. On the other hand, where Tier II symptoms occur early after a dive and appear progressive, prompt IWR could be justified on the basis that it may prevent the development of more serious symptoms.

Contraindications for in-water recompression

There are several signs of DCI which pose a risk of permanent injury, but which are contraindications for IWR and are therefore not included in the Tier III list. Hearing loss and vertigo are both potential symptoms of DCI that can lead to permanent injury. However, when they occur in isolation, that is, with no other symptoms of DCI from any of the other tiers, it is possible that they have been caused by inner ear barotrauma rather than DCI. Inner ear barotrauma is generally considered a contraindication for recompression. Moreover, even when caused by DCI, vertigo is a debilitating symptom which is usually accompanied by nausea and vomiting, and which would make IWR hazardous. Change in consciousness is included in the Tier III group, where it is meant to indicate transient episodes. A diver with a deteriorating level of consciousness or with a persisting reduced level of consciousness should not be recompressed in-water. Other contraindications for IWR include an unwilling or reticent patient, O_2 toxicity as part of the course of the preceding events and any physical injury or incapacitation to the point where the diver may not be able to safely return to the water.

In-water recompression protocols

The requirements for conducting IWR have been detailed elsewhere,[9,17,46] and include: a patient willing and capable of undergoing IWR; adequate thermal protection; a means of supplying 100% O_2 (or close to it) underwater for the duration of the anticipated protocol; a stable platform for maintaining depth, such as the bottom or a decompression line or stage under a boat; a method for tethering the patient; and a competent experienced buddy to accompany the patient. All divers involved (a minimum of a surface supervisor, dive buddy and patient) must be competent in IWR methods, achieved through specific training in IWR methods or in O_2 decompression procedures. A full-face mask or mouthpiece-retaining device is strongly recommended.

Figure 5
Australian IWR schedule; the patient breathes oxygen at 9 msw (30 fsw) for 30 min for mild cases, 60 min for serious cases, and for a maximum of 90 min if there is no improvement in symptoms. The patient continues to breathe O_2 during the 120-min ascent; the ascent rate was originally specified as 1 fsw (0.3048 msw) every 4 min; dashed line shows ascent from maximum 90 min bottom time; O_2 breathing continues on the surface (indicated by the arrow) for six 1-h O_2 periods each followed by a 1-h air break

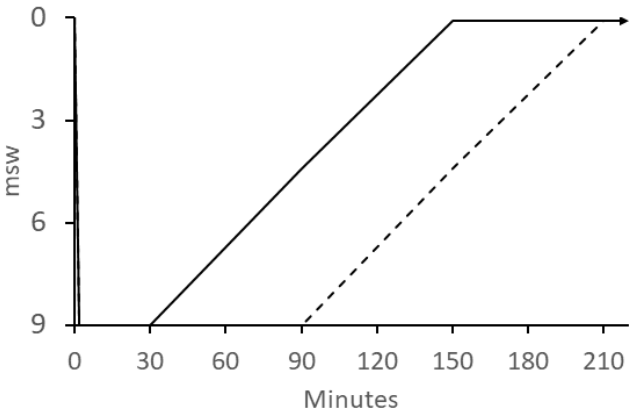
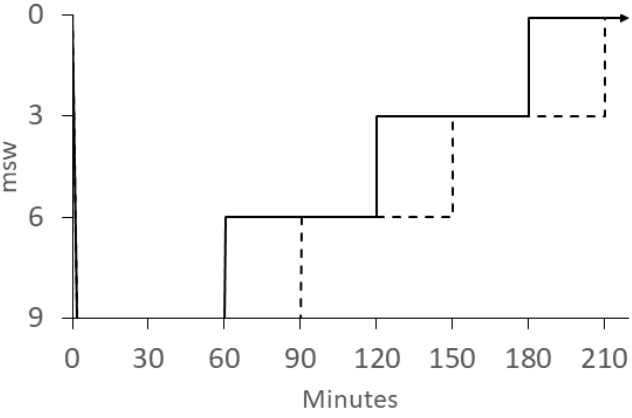
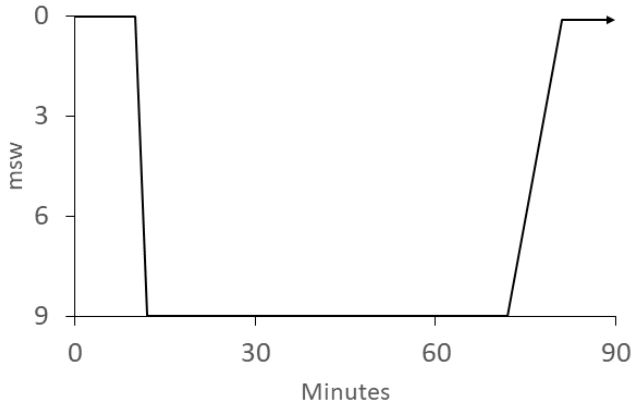


Figure 6
US Navy Diving Manual IWR schedule; the patient breathes O_2 at 9 msw (30 fsw) for 60 min for mild DCS (solid line ascent) or 90 min (dashed line ascent) for neurological DCS; the patient continues to breathe O_2 during 60-min stops at 6 msw (20 fsw) and 3 msw (10 fsw); O_2 breathing continues on the surface (indicated by the arrow) for 3 h



Most published schedules for IWR involve recompression to 30 fsw (9 msw) while breathing pure oxygen. The best known of these is the "*Australian*" method (Figure 5). The US Navy IWR schedule (Figure 6) is adapted

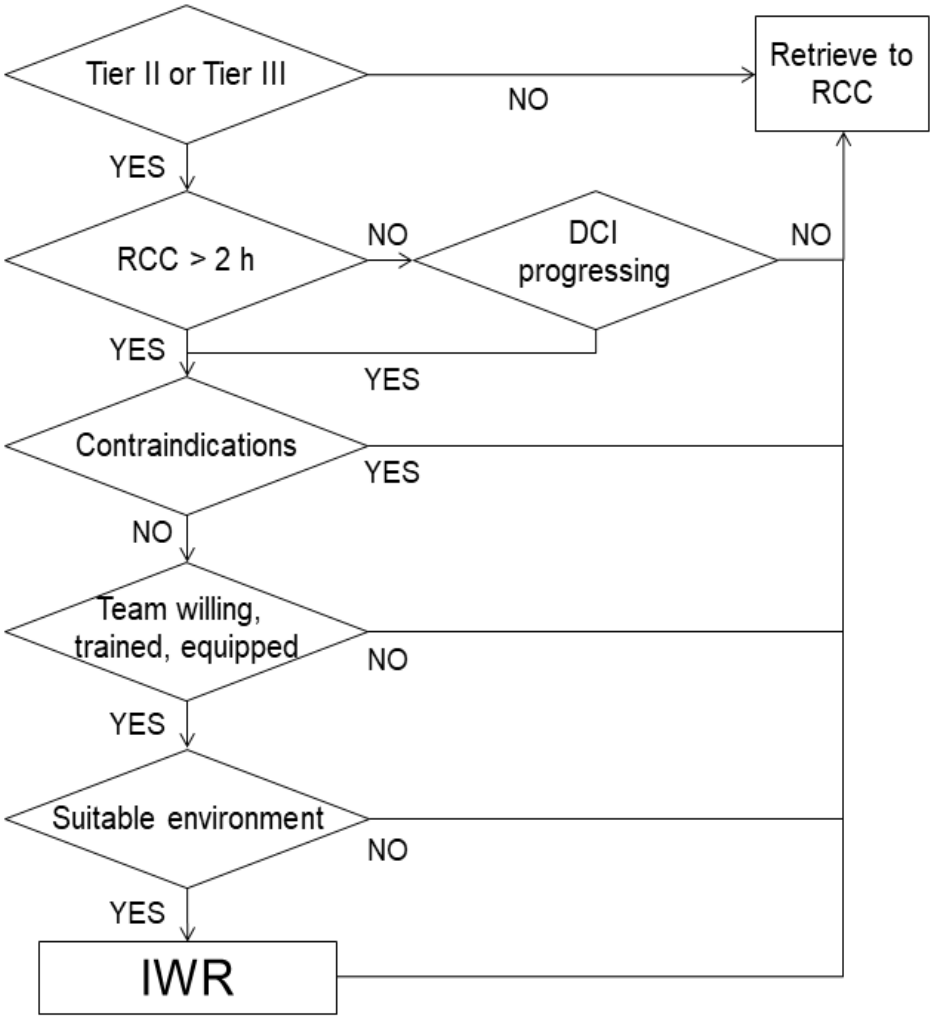
Figure 7
Clipperton IWR schedule; the patient breathes O_2 at the surface for 10 min and, if symptoms do not resolve; descends to 9 msw and continues breathing O_2 for 60 min; the patient continues to breathe O_2 during the 1 msw·min^{-1} ascent; O_2 breathing continues on the surface (indicated by the arrow) for 6 h



from the Australian method but instead of ascending at 4 min·fsw^{-1}, it prescribes a 120-min decompression with 60-min stops at 20 and 10 fsw (6 and 3 msw).[4] It was noted that divers often continued to improve during ascent using the Australian procedure and this was attributed to faster dissolution of bubbles than their Boyle's-law expansion.[6] We support the more gradual ascent prescribed in the Australian procedure. The Clipperton procedure (Figure 7) was proposed as a shorter alternative to other procedures to mitigate dehydration and risk of O_2 toxicity.[7]

Although recompression with a short delay after symptom onset can effectively treat DCI, it does not guarantee there will not be residual or recurring signs and symptoms. Therefore, IWR conducted without medical supervision should be considered a first-aid measure. The patient should be reviewed by (or at least discussed with) a diving medicine authority at the earliest possible time for a possible evacuation for definitive recompression therapy after IWR is completed. The key elements of a potential decision-making approach to IWR are summarised in Figure 8.

Figure 8
A flow chart depicting the key steps in decision-making for in-water recompression (IWR)



Conclusions

Despite lack of widespread support within the medical community, divers are being treated with IWR in locations remote from recompression chambers, particularly by groups of 'technical divers'. No data exist to definitively establish the benefits of IWR compared to the more widely supported first-aid treatment of surface O_2 and transport to the nearest recompression chamber. Moreover, there are very real risks of IWR that require mitigation. Nonetheless, strikingly good outcomes are achieved with very early recompression, using relatively shallow and short hyperbaric oxygen treatments, such as can be achieved with IWR. These considerations recently led a panel of diving medicine experts reviewing the field management of DCI to state that "*in locations without ready access to a suitable hyperbaric chamber facility, and if symptoms are significant or progressing, in-water recompression using oxygen is an option*".[47]

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Technical reports

Storage of partly used closed-circuit rebreather carbon dioxide absorbent canisters

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Key words
Hypercapnia; Technical diving; Soda lime; Respiratory; Equipment

Abstract
(Pollock NW, Gant N, Harvey D, Mesley P, Hart, J, Mitchell SJ. Storage of partly used closed-circuit rebreather carbon dioxide absorbent canisters. Diving and Hyperbaric Medicine. 2018 June;48(2):96–101. doi: 10.28920/dhm48.2.96-101. PMID: 29888381.)
Introduction: Diving rebreathers use "*scrubber*" canisters containing soda lime to remove carbon dioxide (CO_2) from the expired gas. Soda lime has a finite ability to absorb CO_2. We undertook an experiment to determine whether the manner of storage of a partly used scrubber affected subsequent CO_2 absorption.
Methods: An Evolution Plus™ rebreather was mechanically ventilated in a benchtop circuit. Respiratory minute volume was 45 L·min^{-1} and CO_2 was introduced to the expiratory limb at 2 L·min^{-1}. The scrubber canister was packed with 2.64 kg of Sofnolime 797™. Scrubbers were run in this circuit for 90 minutes then removed from the rebreather and stored in packed form under one of three conditions: "*open*" (unsealed) for 28 days ($n = 4$); vacuum "*sealed*" in an airtight plastic bag for 28 days ($n = 5$); or open overnight ($n = 5$). Following storage the scrubber canisters were placed back in the rebreather and run as above until the PCO_2 in the inspired gas exceeded 1 kPa. The total duration of operation to reach this end-point in each storage condition was compared.
Results: The mean run times to reach an inspired CO_2 of 1 kPa were 188, 241, and 239 minutes in the open-28-day, the sealed-28-day and the open-overnight storage conditions, respectively.
Conclusion: Rebreather divers should consider placing partially used soda lime scrubber canisters in vacuum-sealed plastic bags if storing them for longer periods than overnight. If a partially used scrubber canister is to be used again the next day then the storage modality is unlikely to influence scrubber efficacy.

Introduction

Rebreather devices have dramatically enhanced the exploration capabilities of recreational technical divers and scientific divers.[1] Configurations vary, but fundamentally, rebreathers incorporate a circle circuit in which expired gas passes into a counterlung, and is then re-inhaled from the counterlung. Since the diver is metabolising oxygen (O_2) and producing carbon dioxide (CO_2) the O_2 must be replaced, and the CO_2 removed from the circuit. Thus, there is a system (which varies between rebreather designs) of gas addition designed to maintain a safe level of inspired oxygen partial pressure (PO_2) at all times, and the expired gas is passed through a canister containing a CO_2 absorbent material. There are several CO_2 absorbents that may be used in rebreathers, but the most common is soda lime.

Soda lime is a compound substance containing sodium hydroxide, calcium hydroxide and water. It absorbs CO_2 in a three-step chemical reaction in which the sodium hydroxide is recycled and the calcium hydroxide is irreversibly converted to calcium carbonate.[2] Once all the calcium hydroxide is consumed the compound can no longer absorb CO_2 and the canister assembly (typically referred to as a "*scrubber*") is exhausted. This is clearly an important limitation on the duration for which a rebreather can be safely used underwater. If the scrubber is exhausted (or near to it) during a dive, then CO_2 will 'break through' to be re-inspired, and the diver may develop dangerous levels of hypercapnia as a result.[2]

The safe duration of a CO_2 scrubber is determined by many factors including the scrubber design, ambient

temperature, the depth of use, the mass of soda lime it contains, and the physical activity level (and, therefore, the CO_2 production and respiratory minute volume) of the diver. Rebreather manufacturers typically promulgate maximum recommended durations based on 'worst case scenario' testing in which the rebreather is operated mechanically at moderate depth in cold water with ventilation parameters chosen to simulate moderate to heavy exercise. These limits are recognised as being conservative, and their interpretation is further complicated by other factors which might affect duration.

One such factor is the manner in which a partially used scrubber canister is stored between dives. It is common for a rebreather dive to be of substantially shorter duration than the manufacturer's recommended scrubber life. Under these circumstances divers will frequently keep a record of the duration of use, then store the scrubber canister for further use on the next dive without changing the soda lime material. This practice has given rise to a debate on the best practice for storage of a partly used scrubber canister. In particular, it is not known whether sealing a partially used scrubber canister from the environment will confer any advantage in terms of its subsequent CO_2 absorbing performance in comparison to simply storing it unprotected.

To our knowledge there has been only one other relevant study. The Canadian Navy investigated the effect of storing new or partially used soda lime in a rebreather for seven days and found no difference in total duration of effective CO_2 removal compared to soda lime that was not removed from its usual storage container until just prior to use.[3] However, all of these storage modes were effectively sealed, and this study therefore did not address the issue of whether a partly used scrubber canister needs to be sealed for storage. Resolution of this question was identified as a research priority at the recent *Rebreathers in scientific diving Workshop*.[4] Moreover, although this issue could be viewed as 'technological' rather than 'medical', the performance expectations of CO_2 scrubbers are of direct relevance to the prevention of an important gas toxicity (hypercapnia), and any significant effect of scrubber storage conditions could be of relevance to forensic investigations of rebreather accidents where hypercapnia appeared to be a plausible cause.

We undertook a study to determine whether airtight sealing of a partially used CO_2 scrubber canister for storage purposes improved subsequent CO_2 absorbing performance. The null hypothesis was that the manner in which a partially used CO_2 scrubber is stored (sealed vs. open) makes no difference to its CO_2 absorbing capacity during subsequent use.

Methods

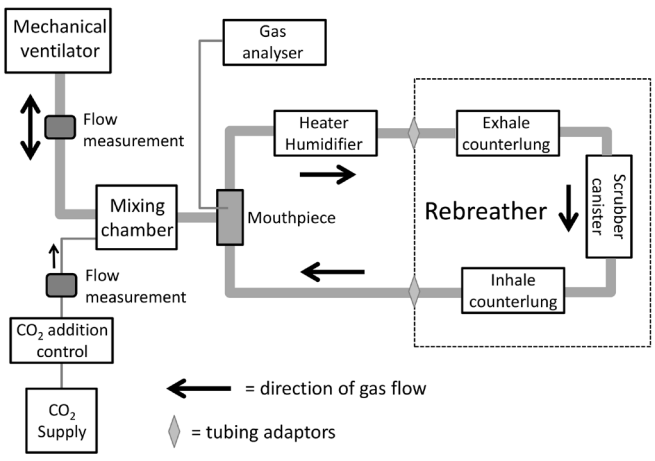
Those aspects of the protocol requiring human participation were approved by the University of Auckland Human Participation Ethics Committee (Reference 015280).

This was a bench-test laboratory study in which an Evolution Plus™ rebreather (Ambient Pressure Diving, Helston, Cornwall) was operated in a test circuit designed to emulate use by an exercising diver. Thus, in a preliminary phase of this study which is described in more detail elsewhere,[5] we established indicative values for respiratory minute (min) ventilation (V_E), tidal volume (T_V), respiratory rate (RR), oxygen consumption (VO_2), and CO_2 production (VCO_2) in a working subject at our chosen exercise intensity. A recent consensus on functional capacity for diving activity identified continuous exercise at 6 MET (one MET equals 3.5 $mL \cdot kg^{-1} \cdot min^{-1}$, the assumed oxygen consumption of an individual at rest) as a desirable and plausible target for sustained exercise output in a diver.[6] Therefore, our human participant exercised at 6 MET on an electronically braked cycle ergometer whilst breathing on the Evolution Plus rebreather in dry conditions. At steady state V_E was 44 $L \cdot min^{-1}$ (T_V = 2.0 L, RR = 22 $breaths \cdot min^{-1}$) and VCO_2 was 2.0 $L \cdot min^{-1}$.

BENCH TEST CIRCUIT DESIGN AND OPERATION

For the subsequent bench test study, the inspiratory and expiratory hoses of the Evolution Plus rebreather were attached to a test circuit (Figure 1) using tubing adaptors (MLA304, AD Instruments, Dunedin, New Zealand). The test circuit was composed of 35 mm (internal diameter) smooth bore respiratory tubing (MLA1015, AD Instruments, Dunedin, New Zealand) connected to a one-way respiratory valve (5710, Hans Rudolph, Shawnee, KS, USA) which simulated the rebreather mouthpiece. This valve was ported to allow continuous sampling of the inspired and expired gas for infrared analysis of inspired and end-tidal PCO_2 (ML206 Gas Analyser, AD Instruments, Dunedin, New Zealand). A clinical heater-humidifier (Fisher and Paykell Medical, Auckland, New Zealand) was incorporated into the exhale limb of the circuit to reproduce the heating and

Figure 1
Schematic layout of the test circuit and monitoring equipment; see text for explanation



humidification of expired gas that would occur with a human breathing on the loop. The heating function was set to 34ºC for all experiments.

Breathing was simulated using a sinusoidal mechanical ventilator (17050-2 Lung Simulator, VacuMed, Ventura, CA, USA) with an inspiratory-expiratory ratio of 1:1. The T_V was set at 1.5 L and the RR at 30 breaths·min^{-1} for all experiments. These parameters differed slightly from the derived human values described above (T_V 2.0 L, RR 22 breaths·min^{-1}) because we found that the ventilator struggled with the work of moving gas around this circuit with a T_V of 2.0 L. Accurate ventilation was ensured through independent monitoring with a pneumotachograph (800 L, Hans Rudolph, Shawnee, KS, USA). The ventilator was connected to the circuit one-way valve via a 4 L mixing chamber where the inspired and expired gas mixed with instrument grade CO_2 introduced at 2 L·min^{-1} using a precision flow pump (R-2 Flow Controller, AEI Technologies, Pittsburgh PA, USA) drawing from a Douglas bag reservoir. The CO_2 flow was also independently monitored to ensure accuracy using a flow transducer (MLT10L, AD Instruments, Dunedin, New Zealand). Operated in this mode with a functional CO_2 scrubber canister in the rebreather, the circuit consistently produced a physiologically authentic inspired CO_2 partial pressure (PCO_2) of close to zero and an end-tidal PCO_2 of 5–6 kPa at the simulated mouthpiece.

CO_2 SCRUBBER CANISTER PACKING

Sofnolime 797™ (Molecular Products, Essex, UK) is the recommended CO_2 absorbent for the Evolution Plus rebreather and was used for all experiments. All Sofnolime was newly purchased from the same batch, in date, and stored before use in the manufacturer-supplied sealed containers. The initial packing of the scrubber canister was supervised by an experienced instructor (PM) on this rebreather. Emphasis was placed on ensuring an evenly distributed tight pack to eliminate the possibility of settling of absorbent material and channelling of gas flow which might cause inaccurate results. After the first supervised pack the Sofnolime was precisely weighed (2.64 kg) before exposure to CO_2 using a laboratory balance (GM-11, Wedderburn Scales, Auckland, New Zealand), and exactly the same weight of material was used for all subsequent trial repetitions. Each new scrubber canister was packed approximately 15 min before the start of an experiment.

TRIAL PROTOCOL

After scrubber canister installation, the rebreather was incorporated into the circuit as described above. The circuit was tested for leaks by holding a positive pressure. The rebreather was switched on and its default surface PO_2 set point of 0.7 atm was chosen. The diluent gas was air for all experiments. Ventilation of the circuit was initiated and, after appropriate operation was confirmed, a timed trial started with the continuous addition of CO_2 at 2.0 L min^{-1}. Every 30 min the ventilation and CO_2 addition were briefly paused to recheck the CO_2 flow sensor calibration and to remove any excess moisture from the circuit hoses. The addition of this step to the protocol reflected the criticality of consistently accurate CO_2 addition to the circuit.

Previous experiments had shown that when packed and operated in the test circuit as described above, it took approximately 200 min for the scrubber to fail (defined as a rise in the inspired PCO_2 to 1 kPa (7.5 mmHg).[5] To evaluate the effect of different storage modalities after partial use, we operated each new scrubber in the rebreather for exactly 90 min after which the scrubber canister was removed intact from the rebreather and immediately stored; either unprotected ("*open*") on a shelf in the laboratory, or in a vacuum-sealed plastic bag ("*sealed*") on the same shelf. The airtight bags were commercially available 0.8 m x 0.8 m household double zip vacuum seal clothing storage bags made from polyethylene (wall thickness 70 micron) with a polyamide valve (All Set Brand, China). Residual air was evacuated through the one-way screw cap valve using a household vacuum cleaner.

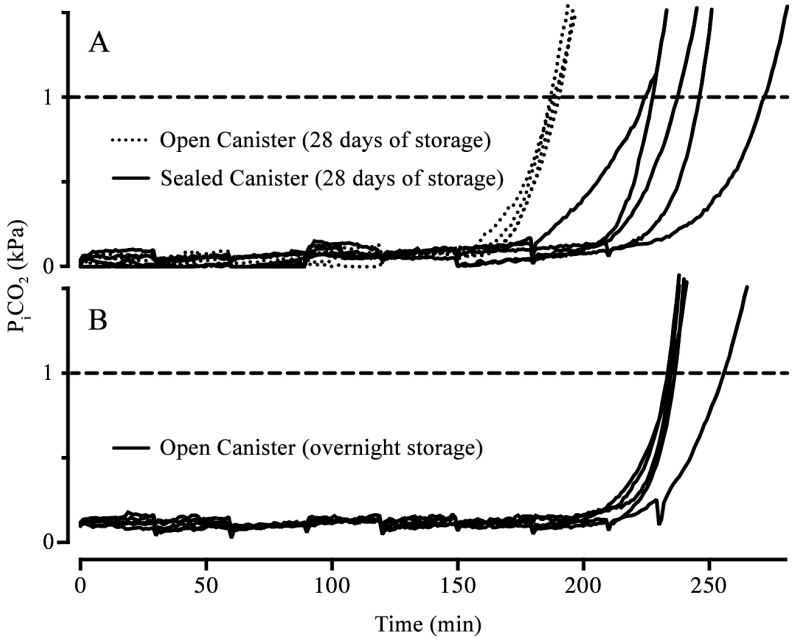
We investigated two periods of storage. The principle set of experiments evaluated one month of storage (exactly 28 days in all cases) which was considered to represent a typical interval between dives for recreational divers. We subsequently added another series involving overnight storage because this is a relevant storage interval for divers on live-aboard or scientific diving trips. The laboratory conditions were kept constant throughout the period of storage with a mean (± SD) temperature of 19.7 ± 3.1˚C and a relative humidity of 53 ± 9%. After the storage period, the scrubber was re-installed in the rebreather and operated under the same conditions until the scrubber failed; that is, until an inspired PCO_2 of 1 kPa was recorded. We had four scrubber canisters available for the study. For each complete cycle of the 28-day storage study two canisters were allocated to open and two to sealed storage so that in any storage period two canisters were stored open and two were stored sealed. For the next cycle of the study each canister would be stored in the opposite condition.

We aimed to investigate five scrubbers in each of three storage conditions: 28-day-sealed; 28-day-open; and overnight-open. The primary outcome was a comparison of the mean total scrubber duration (the sum of pre- and post-storage operating time before failure) in each of the three conditions. Where necessary, statistical comparison between two conditions was made using a two-tailed *t*-test. A *P*-value less than 0.05 was taken to indicate statistical significance.

Results

We completed five trials in each of the 28-day-sealed and overnight-open conditions, and four experiments in the 28-day-open condition. Unfortunately, we exhausted our

Figure 2
Breakthrough curves for the individual scrubber trials in three storage conditions subdivided into the 28-day storage duration (Panel A) and the overnight storage duration (Panel B). The small periodic downward spikes in the curves correspond to the short half-hourly pauses for recalibration of the CO_2 flow sensor and removal of moisture from the circuit hoses (see Methods)



same-batch supply of Sofnolime 797 with one trial in the 28-day-open condition remaining to be done. We attempted to run the trial with Sofnolime from another batch and obtained an aberrant result. Given the confluence of the results obtained in the trials performed using Sofnolime from the common batch (see below) we considered it reasonable to stop the study one trial short in the 28-day-open condition, rather than repeat the entire study with a new batch of Sofnolime.

The elapsed times to reach the failure end-point in each scrubber trial arranged by storage condition, and the mean times to failure for each condition are shown in Table 1. The breakthrough curves for each scrubber trial are shown in Figure 2. There was a substantial (> 50 min) difference in mean duration to failure between scrubbers stored for 28 days in the sealed condition (longer) compared to the open condition (shorter; $P = 0.003$). Scrubbers stored 'open' for the much shorter overnight period showed no difference in mean duration to failure when compared to the canisters sealed for 28 days.

The volumes of CO_2 introduced prior to reaching the failure end point in each scrubber trial arranged by storage condition and the mean volumes for each condition are shown in Table 2. Since some CO_2 was accumulating in the circuit (as opposed from being removed by the scrubber) prior to reaching the 1 kPa inspired CO_2 endpoint, it is not strictly correct to view these data as representing the volume of CO_2 absorbed. Nevertheless, it is a good approximation for the latter. On that basis, on average, the scrubber canisters stored

Table 1
Elapsed time (min) to reach the predefined failure point (an inspired PCO_2 of 1 kPa) in trials in which scrubber canisters were ventilated at 45 L·min^{-1} with introduction of CO_2 at 2 L·min^{-1} to simulate 6 MET exercise. All scrubber canisters were stored in the condition indicated after an initial 90 minutes of operation, and then run to failure after storage

Trial	Storage Condition		
	28 days open	28 days sealed	Overnight open
1	188	229	234
2	188	224	235
3	187	237	237
4	190	246	255
5	–	271	235
Mean (SD)	188 (1)	241 (19)	239 (9)

Table 2
Estimated volume (L) of introduced CO_2 required to elicit an inspired PCO_2 of 1 kPa in trials in which scrubber canisters were ventilated at 45 L·min^{-1} with introduction of CO_2 at 2 L·min^{-1} to simulate 6 MET exercise

Trial	Storage Condition		
	28 days open	28 days sealed	Overnight open
1	378.1	458.9	484.3
2	377.5	451.5	484.2
3	375.3	476.8	498.1
4	381.5	493.6	480.7
5	–	544.8	502.3
Mean (SD)	378.1 (2.6)	485.1 (37.1)	489.9 (9.6)

sealed for 28 days were capable of absorbing approximately 100 L more CO_2 than those stored open for the same period. Although these latter data are largely reflective of the durations reported in Table 1, presentation of the outcome as a function of CO_2 absorption has implications for interpretation of the results (see discussion below).

Discussion

We have shown that storage of a partially used CO_2 scrubber for 28 days in a vacuum-sealed bag substantially preserves its ability to absorb CO_2 during subsequent use when compared to a scrubber that has been stored in an open (unprotected) condition. Our null hypothesis was therefore rejected. This result is consistent with the Canadian Navy finding of no apparent degradation in absorbing function when a partially used scrubber was stored in a sealed environment for seven days,[3] but our study is the first to compare sealed versus open conditions. The results support the view of those in the rebreather community who advocate a sealed condition when a scrubber is stored for a protracted period.

However, our results also indicate that open storage for 24 hours or less does not appear to result in significant degradation of scrubber function. Therefore, it is unlikely to be disadvantageous to store a partly used scrubber in an open condition if it is going to be reused the next day. In this regard we acknowledge that we did not test overnight storage in a sealed condition as a direct comparator. We considered it unlikely that this would reveal significant benefit, not least because the scrubbers stored overnight in the open condition were performing as well or even slightly better than scrubbers operated under identical conditions without a storage period in a previous study.[5]

An obvious question that arises is the cause of the degradation in scrubber function during storage in the open condition. An intuitively obvious explanation is the absorption of CO_2 from the surrounding air. However, the data in Table 2 suggest that for this to be the explanation the scrubber would have absorbed over 100 L of CO_2 during storage. This represents the content of approximately 250,000 L of air (the CO_2 content of air = 0.04%). It is clearly implausible that this degree of bulk flow occurred through the scrubber during its storage, but the extent to which an equivalent amount of CO_2 absorption could have occurred by diffusion of CO_2 into the scrubber canister is unknown.

Another possible answer lies in the dependence of soda lime on the presence of water for the reaction with CO_2 to proceed efficiently.[7] Unfortunately much of the relevant literature is old and published in foreign language journals.[8] Nevertheless, it is part of the wisdom of anaesthesia that dry soda lime is inefficient, and one anaesthesia education website states "*dry granules become exhausted quicker than granules with correct water percentage*".[9] It seems plausible that in an air-conditioned environment at a relative humidity of 54% the canisters stored open may have desiccated to some extent, and this may have resulted in reduced absorptive capacity in subsequent use.

It is relevant to briefly discuss our choice of an inspired PCO_2 of 1 kPa as an end point for our experiments. There has been some debate over safe limits for inspired CO_2 during diving, but recent evidence suggests that limits should be low.[10] Indeed, a widely accepted breakthrough end point for the testing of CO_2 scrubber duration is 0.5 kPa. We chose 1 kPa as a level of inspired CO_2 that few (if any) would regard as clinically insignificant in the diving context. However, we provide the breakthrough curves (Figure 2) partly as evidence that our conclusions would not have materially changed whether we chose 0.5, 1.0, or 2.0 kPa of inspired CO_2 as the end point. We also chose to use a simulated workload (VE = 45 $L \cdot min^{-1}$ and VCO_2 = 2 $L \cdot min^{-1}$) that has a published physiological provenance of relevance to diving,[6] rather than the European standard that is often used for scrubber endurance testing (VE = 40 $L \cdot min^{-1}$ and VCO_2 = 1.6 $L \cdot min^{-1}$).[11] These parameters are similar, and in a study comparing the effect of different storage conditions (as opposed to generating guidelines on scrubber durations) this choice is also of no material significance.

Another methodological matter that deserves comment is our use of a benchtop circuit with the rebreather operated in dry conditions at one atmosphere pressure rather than immersed at elevated ambient pressure. It is known, for example, that immersion in cold water negatively affects the efficiency of CO_2 scrubbers, and operation at greater pressure also shortens duration. However, it must be clearly understood that the primary goal of this study was to investigate any effect of storage conditions on subsequent efficacy of a partly used scrubber. For that purpose, provided methodologic consistency was maintained, the mode of use of the rebreather was essentially irrelevant. We can think of no plausible reason why running the experiment at atmospheric pressure would either mask or exaggerate any deterioration in scrubbing capacity arising from non-optimal storage. As a corollary to these comments it must also be clearly understood that we were not attempting to generate data that might be used to guide the duration of use of scrubbers in real world diving, and our data must not be used in this way. For the sake of comparability, duration testing would best be conducted to a more widely used protocol such as the European standard.[11]

There are several observations that we have not elaborated on in detail here. First, we found greater variability in the time to the endpoint in the canisters that were stored sealed for one month. We do not have an explanation for this observation. Though interesting, it does not materially alter our conclusions. Second, as alluded to earlier, we have noted that canisters stored either sealed for 28 days or open overnight actually appear to have a longer total duration

than those used from new through to the endpoint without interruption in another study.[5] We have not detailed this observation in this paper because it will be the subject of further work designed to more formally investigate and document the phenomenon. Finally, the aberrant result seen with a change in scrubber batch requires further evaluation.

LIMITATIONS

Firstly, this is a small study, and was one trial smaller than intended because we exhausted our supply of soda lime from the same batch. Nevertheless, we believe the results establish a clear signal that a sealed condition is likely to be the optimal approach for prolonged storage without the need for a larger study. Sealing the scrubber inside the rebreather may confer a similar advantage,[3] but we did not specifically test that.

Secondly, in relation to the above, we have not accurately defined "*prolonged*" in relation to scrubber storage. That is to say, we have not established a threshold storage period beyond which soda lime absorptive efficacy declines. Although it would be possible to undertake such work it would be a substantial effort. Moreover, it may be confounded by factors other than time which affect storage (see below), and we are satisfied that simply identifying an advantage for sealed storage under a limited set of conditions is a valuable observation in itself.

Thirdly, it is possible that different conditions of storage may affect the outcome, particularly in relation to the open storage condition. For example, if desiccation is the explanation for degradation of efficacy in the open condition, then the effect may be less dramatic in a more humid non-air-conditioned environment. Similarly, it is possible that the effect may be more dramatic (and possibly apparent over a shorter storage period, including overnight) in a much drier environment. Our results may not be generalizable to all environments.

Finally, we cannot definitively rule out an advantage from sealing for overnight storage because we did not perform trials in this condition. Nevertheless, any such advantage is likely to be small. Despite being stored open overnight, the scrubbers performed in a virtually identical manner to the scrubbers that were sealed for 28 days and slightly better than scrubbers that were run from new to the end point without storage in an earlier trial.[5]

Conclusions

Rebreather divers should consider placing partially used soda lime scrubber canisters in vacuum-sealed plastic bags if storing them for longer periods than overnight. If a partially used scrubber canister is to be used again the next day then the storage modality is unlikely to influence scrubber efficacy.

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Conflicts of interest: nil

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Performance of the Oxylog® 1000 portable ventilator in a hyperbaric environment

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Key words
Equipment; Performance; Pressure; Hyperbaric research; Safety

Abstract
(Lie SA, Loy ST, Lee CC, Kim SJ, Soh CR. Performance of the Oxylog® 1000 portable ventilator in a hyperbaric environment. Diving and Hyperbaric Medicine. 2018 June;48(2):102–106. doi. 10.28920/dhm48.2.102-106. PMID: 29888382.)
Introduction: The management of mechanically ventilated patients in the hyperbaric environment requires knowledge of how the physical properties of gases change under pressure and how this affects the operation of the ventilator. The primary objective of this study was to test the performance of the Dräger Oxylog 1000® ventilator in a hyperbaric environment.
Methods: Each of two ventilators was connected to a mechanical test lung system with an in-built pressure gauge. We used a Wright's respirometer to measure the tidal volumes. The same ventilator settings were tested under varying environmental pressures from ambient (101.3 kPa) to 18 meters' sea water (284 kPa) in a multiplace hyperbaric chamber.
Results: A decrease was found in tidal volume, decrease in airway pressure and increase in respiratory rate delivered by the Dräger Oxylog 1000 portable ventilator with increasing pressures to 284 kPa.
Discussion: These findings can be explained by the operating principles of the Oxylog 1000, which is a time-controlled, constant-volume ventilator that functions as a flow chopper. Even between the two Oxylog 1000 ventilators tested there were different absolute changes in tidal volume, airway pressures and respiratory rates at various depths. Hence, the trend of changes in these variables is probably more important than absolute values.
Conclusion: In summary, understanding the trend of changes in ventilator variables will allow clinicians to make appropriate corrections in ventilator settings and carefully monitor adequacy of ventilation to prevent adverse ventilator-associated events. The Dräger Oxylog 1000 portable ventilator is an adequate back-up ventilator for use with straight-forward, ventilator-dependent patients undergoing hyperbaric treatment.

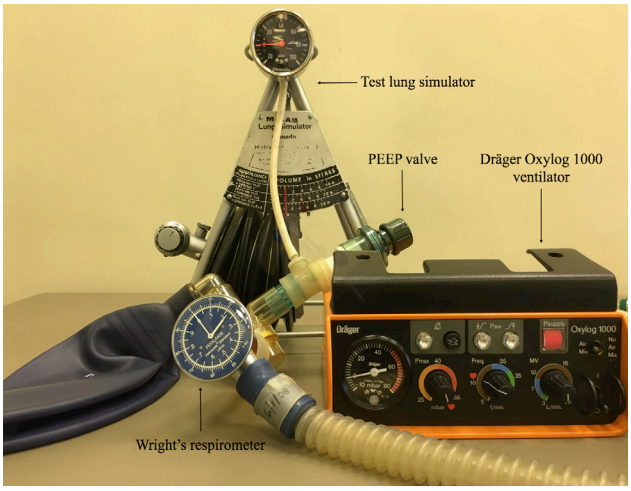
Introduction

Treatment of mechanically ventilated, critically ill patients in the hyperbaric chamber presents unique challenges to the clinician. It requires knowledge of how the physical properties of gases change under pressure, and how this affects the operation of the ventilator before appropriate technical modification or change of settings can be undertaken.[1] Engineering challenges include lack of access to standard high voltage alternating current power supply and risks of fire in a high-pressure, high-oxygen (O_2) environment from sparks generated by motor parts and combustibility of standard lubricants. Hence, electrical equipment for use in the hyperbaric chamber should be *"CE marked"* and validated safe for use.[2] Of equal safety concern for patients is ensuring consistent performance of monitoring devices, infusion pumps and mechanical ventilators under changing ambient pressures.

The performance of all pneumatic devices in a hyperbaric environment is altered by the increase in ambient pressure and gas density. The flow resistance of airways also increases under hyperbaric conditions.[3] As a result, flow of gas in and out of the lungs is slowed, reducing the flow delivered by the ventilator.[4] Such performance characteristics of ventilators under hyperbaric conditions are hard to predict as they differ widely depending on underlying operating mechanics. Most studies done on various ventilator models demonstrate a lower tidal volume delivered compared to the actual set volume, which may lead to hypoventilation if unrecognized.[5–8] As a result, only a few ventilators have been "*CE marked*" for hyperbaric use. Many other simpler transport ventilators are generally capable of functioning in such non-standard conditions, but with recognition of their limitations and modification of settings.[1,9]

The hyperbaric chamber in our institution has a pneumatic powered Dräger Oxylog® 1000 portable patient ventilator and a Siemens Servo 900C ventilator, which is CE-approved. The Dräger Oxylog 1000 is a time controlled, constant volume ventilator that functions as a flow chopper for which there is a paucity of data describing its performance under hyperbaric conditions.[1] Extrapolating from studies that demonstrate a fall in respiratory rate and rise in tidal

Figure 1
Experimental setup in the hyperbaric chamber; the Oxylog 1000® ventilator is connected to a mechanical test lung system with an in-built pressure gauge; positive end expiratory pressure is provided by an Ambu© PEEP valve; a Wright's respirometer measured delivered tidal volumes



volume and minute volume in a hypobaric environment with the Dräger Oxylog,[10,11] it was hypothesized that the converse would happen in a hyperbaric environment – an increase in respiratory rate and fall in tidal volume. Hence, the primary objective of this study was to test the performance of the Oxylog 1000 ventilator in a hyperbaric environment. Clinicians looking after critically ill patients in the hyperbaric environment need to be cognizant of such differences and tailor their monitoring and ventilator strategies accordingly.

Materials and methods

This study was conducted in a multiplace hyperbaric chamber in a tertiary referral centre. Two Oxylog 1000 ventilators were tested. We connected each ventilator to a mechanical test lung system with an in-built pressure gauge (Ohmeda©) that can simulate low lung compliance (dynamic compliance of 20 ml·cmH_2O^{-1}) and high airway resistance, mimicking what would likely be observed in a patient with acute respiratory distress syndrome, with an obstructive airway pattern. Delivered tidal volume (TV) was measured with a Wright's spirometer and airway pressure (P_{aw}) with the in-built pressure gauge of the test lung, rather than the displayed inspiratory airway pressure on the ventilator, for more accurate representation of airway pressure in the test lung. Positive end expiratory pressure (PEEP) was provided using an external PEEP valve (Ambu©). The respiratory rate on the ventilator was set at 15 breaths per minute (min) and actual respiratory rate (RR) was checked manually by counting respiratory movements of the test lung for 1 min. We performed the measurements at two different PEEP levels (0 and 5 cmH_2O) and two different settings for fractional inspired concentration of oxygen (FiO_2) by switching between the "*Air Mix*" mode and "*No Air Mix*" mode on the ventilator. "*Air Mix*" corresponds to a FiO_2 of 60% O_2 by volume (± 10% for minute ventilation (MV) greater than 7 L·min^{-1}) whereas the "*No Air Mix*" mode corresponds to a FiO_2 of 100% by volume under standard manufacturer conditions. MV was set at 20 L·min^{-1} and the upper alarm limit for airway pressure (P_{max}) at 55 cmH_2O. RR, TV and P_{aw} were measured under various pressures ranging from ambient pressure (101.3 kilopascal, kPa) to 284 kPa (equivalent to a depth of 18.4 metres' sea water). Both ventilators were tested under identical conditions, as shown in Figure 1. Internal review board approval was not required as this is an equipment performance experimental study with no research subjects.

Results

The measured RR, TV and P_{aw} obtained at various depths for the two ventilators are detailed in Tables 1 and 2 ("*Air Mix*", FiO_2 of 60%) and Tables 3 and 4 ("*No Air Mix*", FiO_2 of 100%). With increasing pressure from ambient to 284 kPa, tidal volumes delivered in both O_2 modes decreased by up to 64%. This decrease in delivered tidal volume was less at a PEEP of 5 cmH_2O compared to PEEP of 0 cmH_2O. P_{aw} decreased consistently by up to 50% compared to that at 101.3 kPa, whilst the increase in RR with increasing pressure was substantial (up to 180%). Trends in the changes in TV, RR and P_{aw} appeared to be independent of FiO_2 and PEEP levels. The change in MV was inconsistent, with a trend towards achieving a greater than set MV on the ventilator with increasing depth. This is contributed to by the greatly increased RR. Whilst the trends of decreased TV and P_{aw} and increased RR were similar in the two ventilators tested under identical conditions, the actual values observed differed between the two.

Discussion

Our findings of a decreased TV and P_{aw} and increased RR delivered by the Dräger Oxylog 1000 portable ventilator with increasing pressure up to 284 kPa are consistent with the hypothesis posed. The opposite trends were reported in a study of the Oxylog 1000 ventilator under hypobaric conditions from 17 to 3,048 metres altitude.[10] These findings can be explained by the operating principles of the ventilator. The Oxylog 1000 is a time-controlled, constant-volume ventilator that functions as a flow chopper.[12] It has no electronic parts, allowing its safe use in a hyperbaric chamber. Cycling is triggered by a change in pressure in the capacitance chamber caused by a fixed mass of gas. This mass of gas entering the capacitance chamber is controlled by a rotating needle valve linked to respiratory rate. Although mass flow across the valve is increased under hyperbaric conditions, the smaller expansion due to Boyle's law combined with the effect of the shorter inspiratory time means that a smaller TV is delivered at the same ventilator setting at pressure.[10]

Table 1

Oxylog® 1000 ventilator 1, "*Air Mix*" (FiO_2 of ~60%), effect of change in pressure on TV, Paw and RR at PEEP 0 and 5 cmH_2O; ventilator settings – MV 15 $L{\cdot}min^{-1}$, RR 15 per min, "*Air Mix*"; PEEP – positive end expiratory pressure; TV – tidal volume; Paw – airway pressure; RR – respiratory rate; MV – achieved minute ventilation based on TV x RR; * – percentage change in measurement from 101.3 to 284 kPa

Pressure	**PEEP 0** (cm H_2O)				**PEEP 5** (cm H_2O)			
(kPa)	TV (ml)	Paw (cmH_2O)	RR (min^{-1})	MV ($L{\cdot}min^{-1}$)	TV (ml)	Paw (cmH_2O)	RR (min^{-1})	MV ($L{\cdot}min^{-1}$)
101.3	900	50	15	13.5	650	45	15	9.75
162	800	45	22	17.6	650	45	26	16.9
223	550	35	30	16.5	550	40	30	16.5
284	400	30	38	15.2	400	35	39	15.6
% change*	-56	-40	+153		-39	-22	160	

Table 2

Oxylog® 1000 ventilator 2, "*Air Mix*" (FiO2 of ~60%), effect of change in pressure on TV, Paw and RR at PEEP 0 and 5 cmH_2O; ventilator settings – MV 15 $L{\cdot}min^{-1}$, RR 15 per min, "*Air Mix*"; PEEP – positive end expiratory pressure; TV – tidal volume; Paw – airway pressure; RR – respiratory rate; MV – achieved minute ventilation based on TV x RR; * – percentage change in measurement from 101.3 to 284 kPa

Pressure	**PEEP 0** (cm H_2O)				**PEEP 5** (cm H_2O)			
(kPa)	TV (ml)	Paw (cmH_2O)	RR (min^{-1})	MV ($L{\cdot}min^{-1}$)	TV (ml)	Paw (cmH_2O)	RR (min^{-1})	MV ($L{\cdot}min^{-1}$)
101.30	800	50	15	12	750	55	15	11.3
162	750	40	23	17.3	700	55	25	17.5
223	500	30	33	16.5	550	40	33	18.2
284	400	25	42	16.8	400	30	42	16.8
% change*	-50	-50	+180		-47	-45	+180	

Table 3

Oxylog® 1000 ventilator 1, "*No Air Mix*" (FiO2 of 100%); effect of change in pressure on TV, Paw and RR at PEEP 0 and 5 cmH_2O; ventilator settings – MV 15 $L{\cdot}min^{-1}$, RR 15 per min, "*No Air Mix*"; PEEP – positive end expiratory pressure; TV – tidal volume; Paw – airway pressure; RR – respiratory rate; MV – achieved minute ventilation based on TV x RR; * – percentage change in measurement from 101.3 to 284 kPa

Pressure	**PEEP 0** (cm H_2O)				**PEEP 5** (cm H_2O)			
(kPa)	TV (ml)	P_{aw} (cmH_2O)	RR (/min)	MV ($L{\cdot}min^{-1}$)	TV (ml)	Paw (cmH_2O)	RR (min^{-1})	MV ($L{\cdot}min^{-1}$)
101.3	900	50	15	13.5	600	45	15	9
162	800	45	20	16	600	45	19	11.4
223	600	35	28	16.8	580	40	28	16.2
284	500	30	35	17.5	450	35	35	15.8
*% change	-44	-40	+133		-25	-22	+133	

Table 4

Oxylog® 1000 ventilator 2, "*No Air Mix*" (FiO2 of 100%); effect of change in pressure on TV, Paw and RR at PEEP 0 and 5 cmH2O; ventilator settings – MV 15 $L{\cdot}min^{-1}$, RR 15 per min, "*No Air Mix*"; PEEP – positive end expiratory pressure; TV – tidal volume; Paw – airway pressure; RR – respiratory rate; MV – achieved minute ventilation based on TV x RR; *% change – percentage change in measurement from 101.3 to 284 kPa

Pressure	**PEEP 0** (cm H_2O)				**PEEP 5** (cm H_2O)			
(kPa)	TV (ml)	Paw (cmH_2O)	RR (min)	MV ($L{\cdot}min^{-1}$)	TV (ml)	Paw (cmH_2O)	RR (min^{-1})	MV ($L{\cdot}min^{-1}$)
101.3	1100	50	15	16.5	800	55	15	12
162	800	45	21	16.8	750	55	21	15.8
223	500	30	30	15	550	40	30	16.5
284	400	25	38	15.2	450	30	38	17.1
% change*	-64	-50	+153		-44	-46	+153	

The effect of smaller TV leads to lower P_{aw} as depth increases. Overall, despite the decrease in TV, the actual MV achieved was greater than the programmed MV on the ventilator, although this effect was varied and inconsistent. This can be explained by the fact that TV is likely pressure-limited at shallower depths. As TV falls with increasing pressure, it is no longer pressure-limited and the MV subsequently increases. Notably, the two Oxylog 1000 ventilators (even though they were of the same model) performed differently under identical simulated test conditions, producing different absolute changes in TV, RR and P_{aw} at various depths. Therefore, one must bear in mind always that the trend of changes in these variables is probably more important than absolute values since these will be largely unpredictable.

LIMITATIONS

Firstly, we simulated a depth of up to only 284 kPa. Beyond this, there would be further changes in delivered ventilator variables or device malfunction that were not seen.

Secondly, we did not measure the actual delivered FiO_2. The Oxylog 1000 delivers 100% O_2 when there is "*No Air Mix*", or approximately 60% O_2 through a venturi injector when there is "*Air Mix*". In the "*Air Mix*" mode, the O_2 concentration may be increased in situations where there is a high P_{aw} and applied TV is reduced. This is due to the physical characteristics of the injector used for air mixing where the suction effect of injectors decrease with increasing back pressure so less air will be mixed.[12] This has implications in patients who require tight control of FiO_2 and increases the risk of O_2 toxicity.

Thirdly, we assumed that the PEEP generated by the attached AMBU PEEP valve used in the experimental setup was affected mainly by the setting on the valve and not by changes in the ambient pressure in the chamber. In our experiment, the exhaust was dumped into the chamber environment rather than an independent exhaust so the pressure on the exit side of the AMBU PEEP valve will be equal to the chamber pressure. As such, the PEEP setting on the AMBU PEEP valve should approximate the generated PEEP at surface pressure. If on the other hand, for example, under clinical conditions, the exhaust from the ventilator is connected directly to the dumping system of the chamber so as to prevent dumping oxygen into the chamber environment, the opening pressure of the exhaust valve of the dumping system may present a pressure to the exhaust of the ventilator circuit which may then result in a PEEP which differs from the setting on the AMBU PEEP valve. This may then result in difficulties in setting the PEEP accurately and merits further investigation.

Lastly, the Wright respirometer (functioning on the rotating vane principle) used in our study has been shown to overestimate volumes in a hyperbaric environment where gas has greater density than that used for calibration.[4,13] This overestimation can be as much as 18% at 284 kPa.[14] This means that tidal volumes achieved in our study may in fact be an overestimate of actual tidal volumes delivered. As such, volume calibration with a syringe is often used to accurately measure tidal volume in the hyperbaric chamber. In a comparison between the Wright respirometer and Dräger Volumeter 3000, the Volumeter showed a high degree of precision, but accuracy of the Wright respirometer varied with both gas flow and pressure.[4] In another study, good correlation at modest volumes and pressures (up to 254 kPa) between volumes measured by the Wright respirometer and a calibrated displacement lung ventilation performance tester were reported, supporting "*the use of the Wright respirometer alone for monitoring ventilation in clinical practice*".[15] We felt that trends in the changes in achieved ventilation variables would be more important than absolute values (which may differ even between two ventilators of the same model), hence, the Wright's respirometer was sufficient for our research intent.

Conclusions

The functioning of the Dräger Oxylog 1000 portable ventilator is altered under hyperbaric conditions. There is a trend towards decrease in delivered TV and Paw and an increase in RR, while maintaining (or even increasing) achieved MV. Understanding this, the Dräger Oxylog 1000 portable ventilator is an adequate back-up ventilator for use with straight-forward, ventilator-dependent patients undergoing hyperbaric treatment. Clinicians should be cognizant of the differences and appropriate corrections in ventilator settings (where possible) and constant monitoring of the adequacy of ventilation should be performed to prevent adverse ventilator-associated events.

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Repeated hyperbaric exposure and glass ampoule safety

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Key words
Pharmacology; Equipment; Safety; Risk assessment; Pressure

Abstract
(Teoh SY, Vangaveti VN. Repeated hyperbaric exposure and glass ampoule safety. Diving and Hyperbaric Medicine. 2018 June;48(2):107–109. doi: 10.28920/dhm48.2.107-109. PMID: 29888383.)
Introduction: It has been our institution's policy to not place glass medication ampoules inside our hyperbaric chamber for fear of rupture. There is only a small and conflicting amount of data as to whether lass ampoules are safe for use under hyperbaric conditions.
Objectives: The primary objective of this study was to test the safety and usability of glass medication ampoules inside a hyperbaric chamber.
Methods: Repetitive, rapidly staged compressions and decompressions were performed on multiple different glass medication ampoules inside the medical lock of a medical hyperbaric chamber. Medication ampoules of varying sizes (1 ml to 10 ml) of medications that may be required in a hyperbaric emergency were assessed. The ampoules were rapidly compressed 100 times to pressures of 142 kPa, 183 kPa, 300 kPa, 405 kPa and 507 kPa. They were then dropped from a height of 30 cm while compressed at 507 kPa and then half the ampoules were opened while pressurized at 507 kPa.
Results: No ampoules were broken during compression or decompression. No ampoules broke when dropped from 30 cm onto the chamber floor. All ampoules opened at a pressure of 507 kPa functioned normally. No lids/ampoules shattered upon opening.
Conclusion: This study suggests that glass medication ampoules appear to be safe for use inside a medical hyperbaric chamber at routine treatment pressures.

Introduction

Glass ampoules are common ways of storing sterile medications, especially those medications that are for intravenous use. Many of the medications used in our hospital's emergency trolleys are contained in glass ampoules. This poses particular challenges in a hyperbaric environment as these ampoules are a fixed rigid container and contain a gas, usually carbon dioxide or nitrogen, together with the medication either as a liquid or solid. According to Boyle's law, as the ambient pressure increases during compression of the chamber the pressure is transferred onto the glass and as the gas is compressible no support of the glass is provided. If the pressure difference is too great the strain on the glass will cause the ampoule to shatter. For this reason, it is our local policy not to allow glass ampoules into our hyperbaric chambers. It is, however, well known among the diving community, especially the wreck diving community, that there are many ampoules which remain intact on sunken ships at depths exceeding our equivalent normal hyperbaric treatment pressures, e.g., *HMS Pandora*, which lies in 30+ metres' sea water.[1]

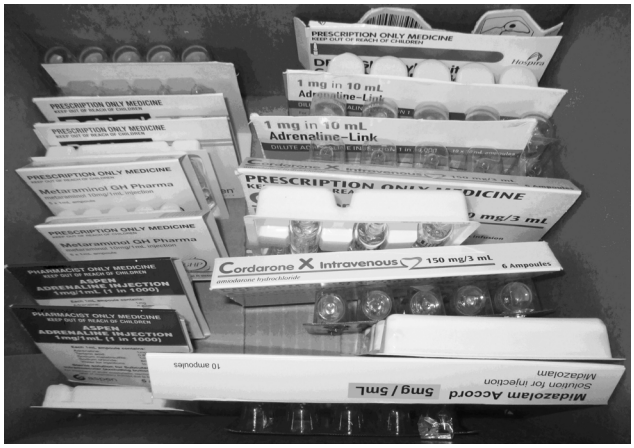
A literature review of Medline, the South Pacific Underwater Medicine Society (SPUMS) journal archives, The *Diving and Hyperbaric Medicine* Journal, the Rubicon Foundation database and Google Scholar revealed no articles on the breaking pressure and safety of glass medication ampoules. One article in 1964 commented that glass ampoules "*appear to withstand 5 to 6 atmospheres of pressure*".[2] However, recent literature, such as *Miller's textbook of anaesthesia*[3] and *Anaesthesia: a core review*[4], caution against the use of glass ampoules in a hyperbaric environment due to the risk of "*explosive rupture*". Neither Aspen Pharmaceuticals nor Pfizer Pharmaceuticals, the two largest suppliers of medications in glass ampoules to our institution, were able to advise whether there was a safe pressure to which such ampoules could be subjected, as they had no testing data for this (Lee K (Pfizer) and Thai M (Aspen), personal communications, 2017).

The current practice at our facility is to have medications outside the chamber and then if needed the outside attendant would draw up the medication into a syringe, making sure to expel any air, and then send it in through the medical lock. This method does delay the time taken for medications to be delivered to patients, especially in an emergency. It also utilises the outside attendant who is then unable to assist in other ways. There has also been a trend in high risk scenarios to pre-draw the medications into syringes and take

Table 1
Medication ampoules used in testing

Medication	**Dose** (mg)	**Volume** (ml)	**Batch/lot**	**Expiry date**	**Manufacturer**
Adrenaline 1:1000	1	1	AS711A1	07/2019	Aspen
Adrenaline 1:10,000	1	10	0085815	07/2018	Link Pharma
Metaraminol	10	1	7J0011C29	09/2020	Global Harvest
Noradrenaline	4	4	203931	09/2017	Mayne Pharma
Glycopyrrolate	0.2	1	AS704R1	04/2019	Aspen
Midazolam	5	5	W08873	05/2021	Accord Health
Amiodarone	150	3	7A032	04/2019	Sanofi
Glyceryl Trinitrate	50	10	709063 and 544058	03/2019 10/2017	Hospira

Figure 1
Drug ampoules in an open cardboard box ready to be placed in the medical lock of a hyperbaric chamber



them in with the patients; however, if unused these are then discarded. This has contributed to wastage of medications. Given the lack of evidence for ampoule pressure rating, a staged protocol was designed to test the strength of glass ampoules.

Methods

After appropriate ethics approval through the Townsville Hospital and Health Service Human Research Ethics Committee (HREC/17/QTHS/162), medications which were held in glass ampoules and used for predictable emergencies that might occur during a hyperbaric treatment were supplied by the hospital pharmacy (Table 1). Ten ampoules of each medication were tested to assess for any inter-ampoule differences. The ampoules had any plastic/foil covers removed and were kept in their plastic holders with cardboard dividers to simulate how they would be stored in the chamber. This also ensured that any ampoule breaking would not affect any other ampoule. All ampoules were placed inside an open cardboard container. The container was then placed into the medical lock of an empty chamber. The chamber was then pressurized to 142 kPa (the routine treatment pressure at our institution) and the medical lock containing the medications was repeatedly pressurized and depressurized 100 times. This was repeated at pressures of 183 kPa and 304 kPa. The ampoules were then compressed to 405 kPa and 507 kPa to assess the breaking strain of the ampoules at the maximum pressure of our hyperbaric chamber. The average rate of compression varied from 16 kPa·sec^{-1} at 142 kPa to 26 kPa·sec^{-1} at 507 kPa; the maximum rate exceeding 70 kPa·sec^{-1}. Ampoules were held at pressure for a minimum of five seconds for each compression.

Ampoules were checked regularly for breakage and data recorded. The protocol for any particular type of ampoule was to stop once 50% of that type had broken. If 50% of any type of ampoule had not broken at 507 kPa, the ampoules were removed from the lock into the chamber and dropped from a height of 30 cm to check for increased fragility.

Prior to the study, an ampoule of each type was dropped in 10 cm increments in height, at room pressure (approx. 101.3 kPa). The 10 ml glyceryl trinitrate ampoule broke when dropped from a height of 40 cm. Therefore, a height of 30 cm was used at which to drop these ampoules. None of the ampoules used in the drop test were utilised clinically. Half the remaining ampoules were then opened at depth to check usability and the remainder opened at the surface to again check usability. Broken ampoules were discarded into a sharps container and disposed of as per hospital policy. All testing was carried out by one researcher (SYT).

Results

No ampoules broke during any of the multiple recompression/decompression cycles.

The containers holding the ampoules were dropped from 30 cm without any ampoules breaking.

Ampoules were opened at 507 kPa pressure without the tops shattering.

Discussion

This experiment far exceeded the expected number, pressures and rates of compression and decompression in clinical hyperbaric practice to assess the safety and breaking strain of various glass ampoules inside a hyperbaric chamber. The usability of medications contained in glass ampoules in a hyperbaric environment was confirmed by opening half the ampoules at pressure. Despite the rapid compression/decompression pressure profiles there were no ampoule breakages and certainly no "*explosive ruptures*". That no ampoules broke when dropped in their storage box from a height of 30 cm onto the chamber floor at 507 kPa suggests that no increase in fragility of the ampoules occurred.

It appears that despite previous concerns regarding the safety of glass ampoules inside a hyperbaric chamber, the strength of currently manufactured ampoules appears to be quite robust and will tolerate repeated routine compression and decompression in a medical hyperbaric chamber at normal treatment pressures, remaining usable at pressure and can be opened as normal without incident.

LIMITATIONS

More testing would need to be carried out to test breaking strain at greater pressures which may be experienced in the commercial or military industries. Neither an exhaustive list of ampoules from different manufacturers nor multiple batches of ampoules were tested. Further testing of ampoules for microscopic fractures as well as for the stability and bioavailability of the contained medication was considered, especially given the temperature fluctuations that occur with rapid compression and decompression. After discussion with colleagues, it was felt that the first step should be to simply test whether ampoules were able to be repeatedly subjected to pressure and utilised in a high pressure environment. Since this necessitated opening the ampoules at depth, any further testing was void.

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Case reports

Problems with an intrathecal pump in a paraplegic scuba diver

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Recreational diving; Medications; Implantable devices; Pressure; Spinal cord injury; Case reports

Abstract

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Scuba diving with an intrathecal baclofen pump is encouraged for people with spinal cord injury who are suffering from spasticity. However, the diving depth is limited to 10 metres in this context. Proper physician and patient education in this respect is mandatory since non-compliance can lead to an irreversible loss of drug reservoir capacity due to collapse of the bottom shield. We report such an incident in a paraplegic diver diving to depths down to 30 metres' water.

Introduction

For more than three decades, scuba diving has been identified as a positive neuro-rehabilitative strategy.[1] People with spinal cord injury (SCI) often suffer from severe spasticity impairing quality of life and can benefit greatly from aquatic therapy. Aquatherapy, performed under the right circumstances, can alleviate spasticity and provides many paraplegic patients with a unique feeling of freedom, resulting from buoyancy.[2] In this report, we describe a paraplegic scuba diver who had a collapsed intrathecal baclofen (ITB) pump after diving to depths down to 30 metres' water (mw).

Case report

A 41-year-old male patient was seen at the outpatient clinic for a refill of his recently implanted ITB pump (Medtronic SynchroMed II®) which had been implanted for the treatment of spasticity after sustaining a traumatic high thoracic spinal cord injury (T4) secondary to a road traffic accident in 2003 (level T4, American Spinal Injury Association (ASIA) Impairment Scale A[3]). Spasticity was well controlled and the patient had no complaints at that time. However, after aspiration of the residual volume, only 13 ml could be injected into the pump. At a refill three years earlier, an identical problem was encountered after the patient went scuba diving to 30 mw when only 27 cc could be injected into a 40 cc ITB pump.[4] At that time, the patient was unaware of Medtronic's advisory of a 10-metre diving depth restriction.[5] At the battery end-of-life, the collapsed pump was replaced by a smaller (20 cc) ITB pump for aesthetic reasons.

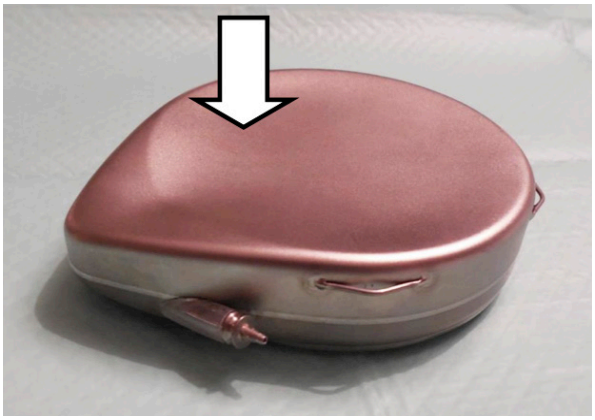
At his visit three years later, he admitted repeated non-compliance, still diving to 30 mw. He reported absolutely no side-effects during diving and stated that diving has a very positive effect on his spasticity. He described a huge positive impact of scuba diving on his quality of life as, whilst underwater, he experienced no handicap or impairment. His diving history revealed that diving to 30 mw had only occurred on two occasions. In between times, he dived two to three days per month, limiting his diving depth to approximately 20 mw. A certified diving buddy always accompanied him.

Figure 1 shows the ITB pump after removal. Although the collapsed pump was still functional, the patient requested its removal so he could continue deep diving. Ceassing the baclofen infusion was associated with the recurrence of debilitating spasticity. To control this, botulinum toxin injections and eventually selective peripheral neurotomies were performed. Two years after removal of the ITB pump and one-and-half years after his last neurotomy, the patient remains satisfied and continues to dive.

Discussion

Intrathecal baclofen (ITB) is an established treatment option for severe spasticity that is insufficiently controlled by oral medication and physical therapy. Since scuba diving offers mobility-impaired people the unique opportunity for three-dimensional movement through a gravity-free environment,[1] the possibility of diving with an ITB pump needs to be discussed with the individual patient.

Figure 1
Collapsed bottom shield of 20 ml intrathecal baclofen pump upon surgical removal



Research on cardiac pacemakers and scuba diving indicates that recommendations vary from country to country.[6] For example, the United Kingdom Sport Diving Medical Committee (UKSDMC) advises the use of resin-filled pacemakers rather than a gas-filled model. The maximum diving depth recommended by the UKSDMC is 10 mw, shallower than the depth rating on the pacemaker model. Other official diving associations are less conservative and allow diving in accordance with the manufacturer's recommendations.[7]

Searching PubMed, only one case report (that of the same patient) could be retrieved concerning ITB pump dysfunction after scuba diving.[4] As mentioned above, Medtronic's guidelines prescribe a maximum diving depth of 10 mw (201.3 kPa)[5] and warn that the bottom shield of the ITB pump may collapse during a single exposure to a pressure greater than his, especially when the pump is not entirely full or after repeated exposure to increased pressures even less than 201.3 kPa.

Mechanical deformation results in a diminished drug reservoir capacity and thus a need for more frequent refills. Besides deformation, a temporary effect on pump flow rate exists due to the increased pressure since the flow rate accuracy decreases by approximately 3% for a pressure of up to 251.3 kPa (15 metres' sea water). Further testing indicated that, when pressure continues to increase and although the pump head continues to rotate, the pump is not able to generate sufficient pressure to dispense, causing the flow to stop. This phenomenon occurs at a pressure of 304 kPa. The pump regains its normal function at normal ambient pressure. Our patient never experienced an increase in the level of spasticity during or after diving, although pump flow would have been temporarily reduced during dives to 20 mw or deeper.

Although the collapsed ITB pump was still functional and spasticity was well controlled, the patient himself insisted on removal with the aim of continuing deep water diving. Given the increasing popularity of scuba diving amongst people with paraplegia, it is important for their carers and diving physicians assessing fitness to dive to be aware of the potential technical problems associated with infusion pumps and other implantable devices.

Conclusion

This report describes a diver with SCI whose physical and mental health benefitted greatly from scuba diving. However, scuba diving with an ITB pump should be restricted to the diving depth recommended by the manufacturers. In this case, non-compliance led to an irreversible loss of reservoir capacity from collapse of the bottom shield of a Medtronic Synchromed II® and likely decreased flow rates during diving to depths of 20 mw or deeper. Proper physician and patient education on scuba diving and ITB therapy is essential to guarantee patient safety whilst enjoying the benefits of scuba diving.

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Acute ophthalmic artery occlusion in decompression illness with underlying anterior cerebral artery A1 segment hypoplasia

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Key words
Decompression sickness; Cerebral arterial gas embolism; Blindness, sudden; Cerebral blood flow; Ophthalmology; Risk factors; Case reports

Abstract

(Omar AR, Ibrahim M, Hussein A. Acute ophthalmic artery occlusion in decompression illness with underlying anterior cerebral artery A1 segment hypoplasia. Diving and Hyperbaric Medicine. 2018 June;48(2):112–113. doi: 10.28920/dhm48.2.112-113. PMID: 29888385.)

A diver presented with total loss of vision in the left eye and right hemiparesis following a routine no-stop scuba dive to 20 metres' depth. A diagnosis of decompression illness (DCI) with acute ophthalmic artery air embolism and left carotid artery insult causing acute anterior circulatory ischaemia was made. He underwent seven hyperbaric treatments leading to a full recovery. Magnetic resonance angiography revealed an underlying left anterior cerebral artery A1 segment hypoplasia. Making a prompt diagnosis and early hyperbaric oxygen treatment are crucial to halt further tissue damage from ischaemia in central nervous system DCI. In this case, the finding of a left A1 anterior cerebral artery segment hypoplasia variant may have increased the severity of DCI due to deficient collateral circulation.

Introduction

Breathing compressed air under pressure results in increased dissolved inert gases in tissues and reduction in ambient pressure may lead to dysbaric diseases, commonly referred as decompression illness (DCI).[1] A common feature of DCI is the embolization of gas bubbles in the venous and/or arterial circulation. This causes ischaemia in surrounding tissues, which is responsible for the large variation of presenting symptoms.

The congenital variation of hypoplastic A1 segment anterior cerebral artery (ACA) of the circle of Willis is reported in 1–10% of the population, based on angiographic and autopsy studies.[2] This preexisting variation may impair collateral blood flow through the circle of Willis and is a recognised risk factor for ischaemic stroke.[2,3] We report a diver with DCI in whom this congenital defect may have contributed to an ipsilateral ophthalmic artery occlusion and carotid artery insult causing anterior circulatory ischaemia.

Case report

A 28-year-old man presented four hours after surfacing from scuba diving with painless, acute total loss of vision in the left eye. He was a smoker with a five-year-pack history with no other significant medical history. He had three years of diving experience, completing 68 uneventful dives. This particular dive was a single dive to 20 metres' depth and a 30 minutes bottom time. He had good buoyancy control, maintained a safe ascent rate and completed a safety stop. He developed sudden blurring of vision and floaters in his left eye a few minutes after surfacing and within four hours his left eye vision was totally lost with no light perception (NLP). This was associated with bilateral fronto-orbital headache and right-sided hemiparesis. He immediately sought treatment at a nearby hospital. Upon arrival, his right visual acuity was 20/20 and the left eye was NLP in all quadrants. The relative afferent pupillary defect sign (RAPD) was positive, indicating absence of retinal signal passing through the optic nerve from the left side. Anterior segment examinations were unremarkable. Posterior segment finding and disc appearance of both eyes were normal. There was no 'cherry red spot' sign, no visible venous pulsation or focal arterial narrowing. Neurological assessment revealed a right-sided sensorimotor deficit.

The diagnosis of left ophthalmic artery occlusion secondary to arterial gas embolism (AGE) was made in view of total visual loss and positive RAPD as well as absence of a 'cherry red spot' (due to lack of blood flow to both the retinal and the choroidal circulation).The contralateral motor-sensory deficit was the result of a vascular insult to the left carotid artery, causing acute anterior circulatory ischaemia with lacunar syndrome (from probable occlusion of one of the penetrating arteries providing blood to deep brain structures).

He was admitted and hyperbaric oxygen therapy (HBOT) was commenced according to the Royal Navy Treatment Table 62.[4] He received a total of seven HBOT sessions at

the end of which left visual acuity was 20/20 with no RAPD and the right hemiparesis had recovered fully. Magnetic resonance angiography (MRA) showed hypoplasia/absence of the A1 segment of the left anterior cerebral artery (ACA). Transthoracic echocardiography (TTE) was performed and there was no evidence of a persistent foramen ovale (PFO).

The patient was referred to a diving medicine specialist who certified him fit to return to diving. However, he was advised to dive within the no-decompression limits, to optimise his fitness and to dive in areas where HBOT was readily accessible.

Discussion

Arterial gas embolism (AGE) is often due to the pulmonary over-inflation syndrome, caused by the expansion of trapped gas in the lung during ascent.[5] It is especially associated with rapid or uncontrolled ascent. Alveolar air may enter the pulmonary venous circulation, thence to the systemic arterial circulation blocking the lumen and damaging the endothelium of small distal arteries, causing ischaemic tissue damage. The brain is especially vulnerable because it obtains a high proportion of cardiac output.[6,7] In this diver's case, neither a rapid ascent nor exceeding recommended dive times appear to have been contributing factors.

The incidence of ocular symptoms in patients with DCI has been reported to be 7% and 12% in two large study series.[8,9] Ocular features described include nystagmus, diplopia, visual field defects, cortical blindness, periocular pain, convergence insufficiency, optic neuropathy and central retinal artery occlusion.[8,9] Delay in therapeutic recompression will cause tissue hypoxia, subsequently leading to permanent ischaemic injury and poor recovery.[9]

The diver was shown to have ACA A1 segment hypoplasia on MRA imaging. The A1 segment of the ACA is a principal supplier of anterior collateral blood flow. Symptoms of neurological deficit in this patient were consistent with a study in which the majority of patients with A1 hypoplasia-related stroke had lacunar infarcts with a contralateral hemiparesis, resulting from occlusion of the penetrating arteries which supply the deep structures of the brain.[3]

TTE was within normal parameters, with no evidence of a PFO. The presence of a PFO is reported to increase the risk of AGE in divers. High-resolution computed tomography (HRCT) of the lungs was not done because the patient defaulted his appointment. Due to our limited resources, we were unable to proceed with pulmonary angiography. HRCT and pulmonary angiography are important investigations in order to look for known risks of AGE such as small airway disease and pulmonary bullae.

Issuing a diving certification or permitting hyperbaric exposure in workers with known ACA A1 segment hypoplasia must take into consideration the absence of collateral circulation in that brain area and the risk must be evaluated thoroughly because the condition has both medical and safety implications. If a fit-to-dive certification is issued, the hyperbaric hazards and risks of a higher morbidity in case of DCI should be fully explained to he diver who should be cautioned to avoid diving in locations lacking rapid accessibility to HBOT.

Conclusion

The developmental variant of ACA A1 segment hypoplasia in the circle of Willis was a major contributing factor in the severity of the ophthalmic artery and anterior circulatory arterial occlusion in a diver with DCI. Early recognition and prompt therapeutic recompression with well-established protocols are crucial to prevent ischaemic tissue injury and permanent disability. Any decision regarding fitness to dive or other hyperbaric exposure for people with this variation should be made taking into account medical safety, economics and medico-legal concerns.

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Letter to the Editor

Do skin rash and *cutis marmorata* stem from lamellar bodies within the skin?

Cutis marmorata (CM) manifests as bluish-red spots on the skin following decompression. These are often itchy or painful to touch, and appear half to one hour after surfacing. The pathogenesis of skin lesions in decompression illness (DCI) remains unresolved. The common belief has been that bubbles that shunted to the arterial circulation reached the skin and clogged blood vessels. An alternative explanation from studies in which air was injected into the internal carotid artery of swine is that arterial bubbles at the brain stem disturb the control of skin blood flow, causing CM.[1] Other brain syndromes have also been seen to cause CM. It was suggested that bubbles affecting the brain stem result in the release of neuropeptides in the skin which control vasodilatation and vasoconstriction. However, this does not explain the inflammation in the skin lesions, with red blood cells, haemorrhage and neutrophil infiltrates. The percentage of right-to-left circulatory shunts in divers who suffered CM was 77% compared with 28% in divers with no record of CM, a finding which supports either of these explanations.[2]

Another study in swine concluded that there was "*strong evidence to support autochthonous bubbles as the etiology of skin lesions*".[3] Lesions appeared without right-to-left shunting. Skin thickness from the squamous keratin to the dermis increased by 10% in the affected areas. The lesions showed congestion, haemorrhage and neutrophil infiltrates. Superficial counter-diffusion as a cause of CM, the increased risk of CM in a dry as opposed to a wet dive and the prevalence of CM in proximity to subcutaneous fat (which acts as a nitrogen reservoir), all support an autochthonous origin.

Decompression bubbles can develop and expand only from pre-existing gas micronuclei. It is known that nanobubbles form spontaneously when a smooth hydrophobic surface is submerged in water containing dissolved gas. We have shown that these nanobubbles are the gas micronuclei underlying decompression bubbles and DCI.[4] After decompression, bubbles evolved at definite hydrophobic sites composed of the lung surfactant dipalmitoylphosphatidylcholine. Nanobubbles are formed on the surface of these lamellar layers of phospholipids, and on decompression expand into venous and arterial bubbles.

Lamellar bodies of phospholipids produced in the granular layer of the skin are used for the formation of a hydrophobic barrier at the cornified layer.[5] We suggest that the hydrophobic layers in the skin may be the site at which bubbles develop from nanobubbles and cause CM, just as occurs at the active hydrophobic spots on the luminal aspect of a blood vessel. This is the reason no bubbles were observed in the skin microcirculation. Unlike bubbles on the inner wall of venous blood vessels, which are supplied with high quantities of nitrogen from the incoming venous blood, the expansion of skin bubbles will be limited due to a low supply of nitrogen (possibly from the nearby subcutaneous fat). Therefore, skin bubbles should be small and have a short life span, which may be why they have hitherto remained undetected. The sensitivity of some divers to CM and its localization to specific skin areas may be related to individual variability in the lamellar bodies and phospholipid skin barriers.

Support for the present hypothesis may be found in the observation in some cases (though not all) of the movement of gas under the skin by means of echography (Balestra C, personal communication, 2018). CM is more frequent in female divers, and more so in subtropical than in cold European waters (van Ooij P-JAM, personal communication, 2018). This may be explained by women having more subcutaneous fat than men, coupled with the higher skin perfusion (and nitrogen loading) in warm water. This suggestion of possible autochthonous bubble formation in the skin does not exclude other causes, but may open a window for further investigation.

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Key words
Skin; Decompression illness; Bubbles; Hypothesis; Letters (to the Editor)

Comment

Evidence brief: hyperbaric oxygen therapy (HBOT) for traumatic brain injury and/or post-traumatic stress disorder

Peterson K, Bourne D, Anderson J, Boundy E, Helfand M. Evidence brief: hyperbaric oxygen therapy (HBOT) for traumatic brain injury and/or post-traumatic stress disorder. VA ESP Project #09-199; 2018. [cited 018 May 14]. Available from: https://www.hsrd.research.va.gov/publications/esp/hbot.pdf.

This report is a product of the VA Evidence-based Synthesis Program. The purpose is to provide "*timely and accurate syntheses of targeted healthcare topics …. to improve the health and healthcare of Veterans"*. The authors have made a comprehensive search and analysis of the literature and make recommendations to assist clinicians in dealing with veterans suffering from either traumatic brain injury (TBI) or post-traumatic stress disorder (PTSD). The report is timely and of great potential impact given the vigorous and lengthy debate among hyperbaric physicians and lay people determined to find an answer for the large numbers of veterans deeply affected with some combination of PTSD and post-concussion dysfunction.

The authors lament the evidence on using hyperbaric oxygen treatment (HBOT) for TBI/PTSD has been "*controversial, widely debated, and potentially confusing*." Unfortunately, this report will not improve that situation. The report is as much a political document as it is evidence-based. That politics are involved is apparent from the outset with the statement "*The ESP Coordinating Center is responding to a request from the Center for Compassionate Innovation (CCI)…"* The report fails to further illuminate the situation than the many thousands of words already spent on summarising the evidence.

Let me save you some time and get to the quick of this report. The authors (rightly) highlight the fact that uncontrolled case series and a randomised, controlled trial (RCT) without blinding or a sham control all suggest HBOT may be of benefit for these Veterans. Somewhat disappointingly, well-controlled, blinded RCTs using a sham exposure to 1.2 or 1.3 ATA (121 or 131 kPa) breathing air fail to confirm any such benefit. While the conventional interpretation of these data is that there is no reliable evidence of an effect of HBOT, proponents have responded by postulating these control exposures are not 'sham' because they are clinically active. Any putative mechanism remains unknown and unproven outside the context of this clinical area. These exposures just happen to be about equipotent with true HBOT. With this accurate summary, the authors conclude that any effect of HBOT is as yet unclear. They suggest that in Veterans who have not responded to other therapeutic options, the use of HBOT is "*reasonable*".

This conclusion allows for a similar recommendation for any unproven therapeutic option where there is no clearly effective treatment available and is, to this reviewer, unacceptable. While any putative mechanism for low-pressure air exposure owes more to magical thinking than physics, physiology or therapeutics, this is an argument the authors of this report seem to have accepted at some level. The proponents of HBOT have an obligation to both show the greater effectiveness of HBOT than a functional sham and to demonstrate a plausible mechanism. Until then, the strongest recommendation that should be made is that the 'sham' therapy can be used until the case is proven. It is not clear why the proponents of HBOT do not advocate this, given the 'efficacy' seems roughly equal with HBOT.

Logic determines one cannot prove a negative. This reviewer agrees it is not possible to definitively prove trivial pressure exposures breathing air may have a comparable effectiveness in treating TBI/PTSD as true HBOT. Using the principle of Occam's razor it seems far more likely any apparent effect is the result of a 'participation effect' in both groups.

In my view, the authors of this report have taken an easy option in allowing that HBOT use is reasonable. The tragedy is potentially the waste of time, money and hope this may bring to the very Veterans the authors are charged to serve. I have discussed this issue in more detail previously in the pages of this journal.[1,2]

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Key words
Hyperbaric research; Central nervous system; Medical condition and problems; Outcome

Reprinted from the literature

Hyperbaric oxygenation for tumour sensitisation to radiotherapy

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Abstract
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Background: Cancer is a common disease and radiotherapy is one well-established treatment for some solid tumours. Hyperbaric oxygenation therapy (HBOT) may improve the ability of radiotherapy to kill hypoxic cancer cells, so the administration of radiotherapy while breathing hyperbaric oxygen may result in a reduction in mortality and recurrence. **Objectives:** To assess the benefits and harms of administering radiotherapy for the treatment of malignant tumours while breathing HBO.

Search methods: In September 2017 we searched the Cochrane Central Register of Controlled Trials (CENTRAL), the Cochrane Library Issue 8, 2017, MEDLINE, Embase, and the Database of Randomised Trials in Hyperbaric Medicine using the same strategies used in 2011 and 2015, and examined the reference lists of included articles. Selection criteria: Randomised and quasi-randomised studies comparing the outcome of malignant tumours following radiation therapy while breathing HBO versus air or an alternative sensitising agent. Data collection and analysis: Three review authors independently evaluated the quality of and extracted data from the included trials.

Main results: We included 19 trials in this review (2,286 participants: 1,103 allocated to HBOT and 1,153 to control). For head and neck cancer, there was an overall reduction in the risk of dying at both one year and five years after therapy (risk ratio (RR) 0.83, 95% confidence interval (CI) 0.70 to 0.98, number needed to treat for an additional beneficial outcome (NNTB) = 11 and RR 0.82, 95% CI 0.69 to 0.98, high-quality evidence), and some evidence of improved local tumour control immediately following irradiation (RR with HBOT 0.58, 95% CI 0.39 to 0.85, moderate-quality evidence due to imprecision). There was a lower incidence of local recurrence of tumour when using HBOT at both one and five years (RR at one year 0.66, 95% CI 0.56 to 0.78, high-quality evidence; RR at five years 0.77, 95% CI 0.62 to 0.95, moderate-quality evidence due to inconsistency between trials). There was also some evidence with regard to the chance of metastasis at five years (RR with HBOT 0.45 95% CI 0.09 to 2.30, single trial moderate quality evidence imprecision). No trials reported a quality of life assessment. Any benefits come at the cost of an increased risk of severe local radiation reactions with HBOT (severe radiation reaction RR 2.64, 95% CI 1.65 to 4.23, high-quality evidence). However, the available evidence failed to clearly demonstrate an increased risk of seizures from acute oxygen toxicity (RR 4.3, 95% CI 0.47 to 39.6, moderate-quality evidence).

For carcinoma of the uterine cervix, there was no clear benefit in terms of mortality at either one year or five years (RR with HBOT at one year 0.88, 95% CI 0.69 to 1.11, high-quality evidence; RR at five years 0.95, 95% CI 0.80 to 1.14, moderate-quality evidence due to inconsistency between trials). Similarly, there was no clear evidence of a benefit of HBOT in the reported rate of local recurrence (RR with HBOT at one year 0.82, 95% CI 0.63 to 1.06, high-quality evidence; RR at five years 0.85, 95% CI 0.65 to 1.13, moderate-quality evidence due to inconsistency between trials). We also found no clear evidence for any effect of HBOT on the rate of development of metastases at both two years and five years (two years RR with HBOT 1.05, 95% CI 0.84 to 1.31, high quality evidence; five years RR 0.79, 95% CI 0.50 to 1.26, moderate-quality evidence due to inconsistency). There were, however, increased adverse effects with HBOT. The risk of a severe radiation injury at the time of treatment with HBOT was 2.05, 95% CI 1.22 to 3.46, high-quality evidence. No trials reported any failure of local tumour control, quality of life assessments, or the risk of seizures during treatment.

With regard to the treatment of urinary bladder cancer, there was no clear evidence of a benefit in terms of mortality from HBOT at one year (RR 0.97, 95% CI 0.74 to 1.27, high-quality evidence), nor any benefit in the risk of developing metastases at two years (RR 2.0, 95% CI 0.58 to 6.91, moderate-quality evidence due to imprecision). No trial

reported on failure of local control, local recurrence, quality of life, or adverse effects.

When all cancer types were combined, there was evidence for an increased risk of severe radiation tissue injury during the course of radiotherapy with HBOT (RR 2.35, 95% CI 1.66 to 3.33, high-quality evidence) and of oxygen toxic seizures during treatment (RR with HBOT 6.76, 96% CI 1.16 to 39.31, moderate-quality evidence due to imprecision).

Authors' conclusions: We found evidence that HBOT improves local tumour control, mortality, and local tumour recurrence for cancers of the head and neck. These benefits may only occur with unusual fractionation schemes. Hyperbaric oxygenation therapy is associated with severe tissue radiation injury. Given the methodological and reporting inadequacies of the included studies, our results demand a cautious interpretation. More research is needed for head and neck cancer, but is probably not justified for uterine cervical or bladder cancer. There is little evidence available concerning malignancies at other anatomical sites.

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Key words
Evidence; Medicine; Cancer; Abstract; Reprinted from

An evidence-based system for health surveillance of occupational divers

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Abstract
(Sames S, Gorman DF, Mitchell SJ, Sandiford P. An evidence-based system for health surveillance of occupational divers: Occupational diver health surveillance. Internal Medicine Journal. 2016 August;46(10):1146-52. doi: 10.1111/imj.13204.)

Background: The value of the commonly required routine annual medical examination of occupational divers has been questioned, and there is a need for a robust, evidence-based system of health surveillance for this group of workers.

Aims: To determine whether the medical examination and investigation component of occupational divers' routine comprehensive health surveillance adds significantly to the information gained from the questionnaire component in determining fitness for diving.

Methods: An occupational diver database was interrogated to identify divers issued with a 'limited' medical clearance or considered 'unfit' for diving over a five-year period. Reasons for the 'unfit' or 'limited' designation and the source of the critical information, whether the annual health questionnaire or the medical examination or questionnaire component (or both) of the initial or five-yearly comprehensive medical evaluation, was recorded. For divers completing the five-yearly repeat comprehensive medical evaluation, the sensitivity and specificity of the questionnaire alone for determining unfitness for diving was compared with that of a nominal 'gold standard'.

Results: Of 5,178 certificates issued to 2,187 divers over a five-year period, 158 (3%) were provisionally designated as either 'limited' or 'unfit'. Of nine divers identified by the examination component of the five-yearly comprehensive medical evaluation, four were eventually designated 'fit', two 'limited', and three were lost to follow up. None who had completed subsequent investigations remained 'unfit'. The sensitivity and specificity of the questionnaire to detect unfit divers compared with the gold standard were 84.6 and 99.3%, respectively, and its accuracy was 98.9%.

Conclusion: The current New Zealand occupational diver medical certification process, comprising annual health questionnaires and five-yearly full examinations, detects all health issues critical to the determination of fitness to dive.

Republished with kind permission from: Sames S, Gorman DF, Mitchell SJ, Sandiford P. An evidence-based system for health surveillance of occupational divers: Occupational diver health surveillance. Internal Medicine Journal. 2016 August;46(10):1146-52. doi: 10.1111/imj.13204.

Key words
Fitness to dive; Occupational health; Diving industry; Abstract; Reprinted from

Immersion pulmonary edema and comorbidities: case series and updated review

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Abstract
(Peacher DF, Martina SD, Otteni CE, Wester TE, Potter JF, Moon RE. Immersion pulmonary edema and comorbidities: case series and updated review. Med Sci Sports Exerc. 2015;47:1128–34. doi: 10.1249/MSS.0000000000000524. PMID: 25222821.)

Purpose: Immersion pulmonary edema (IPE) occurs in swimmers (especially triathletes) and scuba divers. Its pathophysiology and risk factors are incompletely understood. This study was designed to establish the prevalence of preexisting comorbidities in individuals who experience IPE.

Methods: From 2008 to May 2010, individuals who had experienced IPE were identified via recruitment for a physiological study. Past medical history and subject characteristics were compared with those available in the current body of literature.

Results: At Duke University Medical Center, Durham, NC, 36 subjects were identified (mean age = 50.11 ± 10.8 yr), of whom 72.2% had one or more significant medical conditions at the time of IPE incident (e.g., hypertension, cardiac dysrhythmias or structural abnormality or dysfunction, asthma, diabetes mellitus, overweight or obesity, obstructive sleep apnea, hypothyroidism). Forty-five articles were included, containing 292 cases of IPE, of which 24.0% had identifiable cardiopulmonary risk factors. Within the recreational population, cases with identifiable risk factors comprised 44.9%. Mean age was 47.8 ± 11.3 yr in recreational divers/swimmers and 23.3 ± 6.4 yr in military divers/swimmers.

Conclusions: Cardiopulmonary disease may be a common predisposing factor in IPE in the recreational swimming/diving population, whereas pulmonary hypertension due to extreme exertion may be more important in military cases. Individuals with past history of IPE in our case series had a greater proportion of comorbidities compared to published cases. The role of underlying cardiopulmonary dysfunction may be underestimated, especially in older swimmers and divers. We conclude that an episode of IPE should prompt the evaluation of cardiac and pulmonary function.

Reprinted with kind pemission from Peacher DF, Martina SD, Otteni CE, Wester TE, Potter JF, Moon RE. Immersion pulmonary edema and comorbidities: case series and updated review. Med Sci Sports Exerc. 2015 Jun;47(6):1128-34. doi: 10.1249/MSS.0000000000000524. PMID: 25222821.

Key words
Medical conditions and problems; Cardiovascular; Pulmonary function; Abstract; Reprinted from

Professional development meeting summary

Hull DCI study day – optimising outcomes

A professional development day entitled "*DCI study day – optimising outcomes*" took place at Hull Royal Infirmary, Hull, UK on 14 April 2018. The day was organised by Gerard Laden and the medical team from the Hull Hyperbaric Unit in cooperation with Mimir Marine Ltd., a company with a global responsibility for hyperbaric rescue of saturation divers following vessel abandonment.

Approximately 120 delegates were present and there were live telecast links to doctors in Malta, Gozo and Croatia. Speakers and chairpersons were present from Norway, the Netherlands, Germany and the UK. Fifteen presentations were given to an enthusiastic audience of medical practitioners, researchers and off-shore diving operations managers.

A highlight was a fascinating case from Sebastian Klapa (University of Kiel, Germany) who presented a case of decompression illness (DCI)-induced hypovolemic shock with a slow but life-threating evolution, which included a (scary!) dive profile that gave much cause for discussion. Other talks included submarine mass casualty escape medical management, followed by individual case reports, including cerebral DCI and severe spinal cord injury. These emphasised that there is much we cannot see or still do not know about DCI, and recalled the celebrated Palmer histology case report.[1]

Steve Sheppard, saturation diving operations manager for Helix WellOps, showed that having good communications, with policies, plans and procedures in place, does not always guarantee a good outcome and that provision of a 'Plan B' is always necessary.

The possibility of 'optimising outcomes' was reviewed with presentations on lidocaine by Robert Weenink (AMC, the Netherlands) and steroids by Vincent Hong (Hull, UK) whilst Bruce Mathew (Hull, UK), consultant neurosurgeon, considered the use of lumbar cerebrospinal fluid drains. This discussion stemmed from the view that US Navy Treatment Table 6 is no panacea and we all have a responsibility to remain open and thoughtful when trying to provide the best patient outcomes.

The usefulness of dive computers to aid with the investigation of diving accidents was discussed by Martin Sayer (Tritonia Scientific, UK) and James Francis (UK) looked at the pitfalls of not recording treatments in great enough detail should a legal case be pursued after a poor outcome.

The consensus on the meeting from the audience was positive and appreciative of the efforts made by the Hull team to organise the day's events.

Reference

1 Palmer AC, Calder IM, McCallum RI, Mastaglia FL. Spinal cord degeneration in a case of "*recovered*" spinal decompression sickness. Br Med J (Clin Res Ed). 1981;283(6296):888.

Lesley Blogg, SLB Consulting Ltd, UK
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Key words
Decompression illness; Treatment; Outcome; Meetings

Diving and Hyperbaric Medicine Journal is now on Facebook

NEW Facebook page to conincide with our electronic launch - Like us at:

https://www.facebook.com/divingandhyperbaricmedicine/

SPUMS Facebook page

Keep up with SPUMS .. remember to 'like' us at:

http://www.facebook.com/pages/SPUMS-South-Pacific-Underwater-Medicine-Society/221855494509119

Second Tricontinental Scientific Conference on Diving and Hyperbaric Medicine

http://www.tricon2018.org

Dates: 23−29 September 2018
Venue: Durban, South Africa

Tricon2018 replaces the usual EUBS and SPUMS meetings for 2018.

We are once again organising a full week with scientific days interspersed between diving workshops and social events. The academic programme will include oral and poster presentations, workshops, discussion sessions and special topic conferences.

The closing date for Abstracts and Submissions for the Zetterström or Musimu Awards has been extended, please send your abstract in time!

A joint organising committee from EUBS, SPUMS, SAUHMA and the Scott Haldane Foundation are working with local Durban Hyperbaric Centre staff and a South Africa Event Management Bureau to make sure everything runs smoothly.

An excellent social calendar as been planned, including opportunities to dive the nearby Aliwal Shoal, visit wildlife game parks, take in a local rugby match and explore Zulu culture. A combination of easy access, friendly people, rich culture, nature at its most spectacular and affordable prices makes this an opportunity not to be missed. The weather in September is ideal with temperatures in the low 20s on land and in the sea and little chance of rain. Why not plan an extra week before or after the conference to travel the area and experience more of South Africa's amazing diversity, hospitality and wildlife. Bring your family too – there are lots of child-friendly activities nearby!

The dedicated website: http://www.tricon2018.org is ready and waiting for your registration and accommodation booking.

Notices and news

SPUMS society information and news is to be found mainly on the society website: www.spums.org.au

ANZ Hyperbaric Medicine Group Introductory Course in Diving and Hyperbaric Medicine 2019

Dates: 18 February–01 March
Venue: Esplanade Hotel, Fremantle, Western Australia
Cost: AUD2,600 (inclusive of GST)

Course Conveners: Ian Gawthrope and Neil Banham
The Course content includes:
- History of diving medicine and hyperbaric oxygen
- 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:
Sue Conlon, Course Administrator
Phone: +61-(0)8-6152-5222
E-mail: fsh.hyperbaric@health.wa.gov.au

SPUMS 48th Annual Scientific Meeting 2019 Preliminary notice

Dates: 20–26 May 2019
Venue: Heritage Park Hotel, Honiara, Solomon Islands
Theme: "*Old divers and bold divers but not old, bold divers. Cardiovascular health risk assessment and diving.*"

Convener: Catherine Meehan
Scientific Conveners: David Smart and Michael Bennett
More information soon on the SPUMS website.

Australian and New Zealand College of Anaesthetists Diving and Hyperbaric Medicine Special Interest Group

The new Diploma of Advanced Diving and Hyperbaric Medicine was launched on 31 July 2017. Those interested in training are directed to the ANZCA website http://www.anzca.edu.au/training/diving-and-hyperbaric-medicine. Documents to be found at this site are:
Regulation 36, which provides for the conduct of training leading to the ANZCA Dip Adv DHM, and the continuing professional development requirements for diplomats and holders of the ANZCA Certificate of DHM;
ANZCA Advanced DHM Curriculum which defines the required learning, teaching and assessment of the diploma training programme; and
ANZCA Handbook for Advanced DHM Training which sets out in detail the requirements expected of trainees and accredited units for training.

At this stage, the **ANZCA Handbook for Advanced DHM Accreditation** is not finalised and cannot be accessed on the website. All training units which were accredited under the previous Certificate programme have been granted temporary accreditation for the new Dip Adv DHM, but will need to apply for formal accreditation once the Handbook is released.

Holders of the Certificate of DHM and highly experienced practitioners of DHM are eligible for recognition of prior experience towards the ANZCA Dip Adv DHM, as outlined in the guidelines for the transitional award of diploma in Regulation 36. Applications for credit must be made in writing to the ANZCA TA unit and must be submitted prior to 31 January 2019.

All enquiries should be submitted to dhm@anzca.edu.au.

Suzy Szekely, Chairperson, ANZCA DHM SIG
Suzy.Szekely@health.sa.gov.au

The SPUMS website is at **www.spums.org.au**

Members are encouraged to log in

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 www.spums.org.au or at 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: www.nhmrc.gov.au/_files_nhmrc/publications/attachments/r39.pdf, or the equivalent requirement of the country in which the research is conducted. All research involving humans, 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 January 2016, the SPUMS Academic Board consists of:

Dr David Wilkinson, Education Officer, Adelaide;
Professor Simon Mitchell, Auckand;
Dr Denise Blake, Townsville.

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

Key words
Qualifications; Underwater medicine; Hyperbaric oxygen; Research; Medical society

The SPUMS President's message

David Smart, President SPUMS

Corporate governance of *Diving and Hyperbaric Medicine*

The Australian Government defines corporate governance as "*the process by which agencies are directed and controlled. It is generally understood to encompass authority, accountability, stewardship, leadership, direction and control.*"[1]

Diving and Hyperbaric Medicine (DHM) is a scientific journal jointly published by the South Pacific Underwater Medicine Society and the European Underwater and Baromedical Society. As such, both societies are responsible for the corporate governance of DHM. The journal has come a long way under the editorship of Mike Davis. After assuming the role of Editor for the September 2002 edition of the *SPUMS Journal*, he has methodically developed the Journal into a world-class publication, with a steadily rising Impact Factor. In March 2008, our European Colleagues from EUBS joined with SPUMS as joint publishers of DHM.

The March 2018 edition celebrated a 10-year union of EUBS with SPUMS, and was for the first time produced as an e-journal. The quality of the March 2018 edition was testimony to the attention to detail that we now know to be a Mike Davis hallmark. He has been ably supported by Nicky Telles who has exceptional talents that have been applied to the task of transitioning DHM. The e-journal production was no mean feat, requiring extensive behind the scenes work, and the SPUMS ExCom is delighted with the result.

At present, despite 10 years of joint publication, very little has been documented about the governance of the journal. The e-journal will permit greater exposure to the world and potential for even greater global impact from DHM. This has highlighted a number of governance issues requiring attention from both societies. With the appointment of the new DHM Editor for 2019, Professor Simon Mitchell, and his transition into the role in late 2018, I have prepared a summary of my understanding of the current governance of DHM, and areas that need to be addressed. These are my personal observations, and the list is by no means complete.

(1) Editing and production of DHM. This is entirely the responsibility of the Editor, free from publisher influence, in accordance with a contract (defining roles, two-way responsibilities and an honorarium – not based on market rates), which is renewed on a three- to five-year basis. The contract is administered by the Societies' Presidents. Currently there is no written contract for the European (Deputy) Editor. Once Simon Mitchell is established as Editor, an appropriate governance structure and contract will need to be set up for the European (Deputy) Editor, with a new call for expressions of interest.

(2) Academic – via the Editorial Board (EB). The process of academic review has been functioning successfully for a number of years. The EB has an academic and ethical focus and has not been involved in the corporate or financial governance of DHM. Appointment to the EB is made by the Editor-in-Chief, usually with consultation of current EB members; there is no specific term of office. The roles and responsibilities of EB members are not clearly defined but are set out in various documents generated by such organisations as the European Association of Science Editors.

(3) Financial management of DHM – via the Journal Governance Committee (JGC). The JGC became operational four years ago. After some initial fact-finding, the two appointed members from each Society, together with the SPUMS Treasurer, have played a significant role in assessing the income and expenses for DHM, and then they forward plan the following year's budget, to advise the publishing societies, for their acceptance. This allows calculation of the unit cost per issue, which greatly assists budgeting.

Assisted by the Editor in identifying costs and future trends, the key role of the JGC cannot be underestimated, given the prominence of the cost of DHM in members' subscriptions. The JGC also has roles to review other financial issues such as advertising, fees for immediate release articles and to facilitate communication between the Editor and the publishers on matters related to publication of DHM. It is my opinion that the JGC functions and terms of reference will need review once the new Editor has settled into the role. Given the use of the term *governance* for the committee, an additional role could be to assist corporate policy development for the journal.

(4) Journal Copyright – DHM is registered in Australia with the National Library of Australia and the Copyright Agency Australia (CAA), reflecting its origins as the *SPUMS Journal* and SPUMS as a registered Australian Company. This does not at all affect the joint academic and publisher status of EUBS and SPUMS,as the funders of DHM. When copyright payments are made to ACA (and then forwarded to the DHM Journal account), they have three defined origins: for *SPUMS Journal* with SPUMS as the publisher (up to December 2005); for DHM with SPUMS as the publisher (March 2006 to December 2007) and for DHM with SPUMS and EUBS as publishers from March 2008. These payments for use of copyright are usually small, around $300–$400 per annum from all sources.

Currently authors sign a copyright and release form which states that the authors retain copyright to their work, allows limited rights to DHM to publish the paper in electronic form and other media and signs over all copyright income less than $1000 to DHM. Following my inquiry in April, I received advice from the CAA that the publishers (SPUMS and EUBS) hold the copyright, irrespective of the format of the journal. Following e-journal production, the Editor

has raised with the Presidents his concerns about copyright, particularly relating to individual author copyright and the terms and conditions under which their material is published. Given this conflicted situation and the importance of copyright, CAA referred me to the Australian Copyright Council for further advice. I will report further on copyright in due course.

(5) Publisher Memorandum of Understanding (MOU). The MOU is signed by the Society Presidents, following tabling and discussion at Executive Committee meetings. The MOU expires 30 June 2018, and must be updated to ensure that it encompasses all the changes that have taken place over the last three years. Before the next MOU is signed, a number of common policies need to be developed by the Societies in relation to DHM. It is essential that both Societies are in agreement as to how DHM is governed, and its strategic direction. This needs to include clear definition of the Editor's and the Societies' relationships with various committees, how the society websites link to the DHM website, how payments are made for subscriptions to DHM on behalf of each Society's members and dispute resolution. This will also include clear definition of the issues that are publisher responsibility, in facilitating the viability and prosperity of DHM.

(6) Journal operations manual (a work continuously in progress). This manual currently resides with the Editor, and it is the Societies' expectation that this operations manual will be handed over in the Editorial transition process.

(7) Relationship agreements with key individuals who provide services to support the journal. These are essential so that there is mutual understanding of each other's roles and responsibilities.

Other areas of governance will likely be identified, which I have not listed and that will require adding to the list. I am fully committed as current President of SPUMS to join my EUBS colleagues in creating an effective but uncomplicated structure of governance so DHM can thrive into the future. To the best of my ability I also plan to pass on a written corporate record of what I have learned in the role, so that future office bearers in both Societies will not need to reinvent the wheel and pass on verbal history to the next generation.

The last decade has indeed spread the wings of change across DHM – for the better. There is yet more to achieve but I perceive a spirit of cooperation between the Societies and excitement to take our relationship to the next level. I look forward to enhancing our links with EUBS and SAUHMA colleagues when we meet in Durban in September.

Reference

1 Corporate Governance Handbook for Company Directors and Committee Members. A reference guide to understanding the serious commitment of being a company director or committee member. 2nd Edition. 2010. [cited 2018 May 04]. Available from: https://www.dss.gov.au/sites/default/files/documents/05_2012/gov_handbook_2010.pdf

Key words
Medical society; Policy; Copyright; General interest

Royal Adelaide Hospital Medical Officers' Course, Diving and Hyperbaric Medicine 2018

Dates: 05–16 November
Venue: The Royal Adelaide Hospital, Adelaide
Cost: AUD2,500.00 (inclusive of GST)

Course Conveners: David Wilkinson and Suzy Szekely
Invited faculty includes: Professors Michael Bennett and Simon Mitchell
The course content includes:

- Physics and physiology of diving
- Recreational fitness-to-dive
- Occupational fitness-to-dive
- Decompression illness and non-dysbaric injuries
- Medical management and return to diving
- Technical and professional diving
- Marine envenomation
- Introduction to hyperbaric medicine

Contact for information:
Ms Lorna Mirabelli, Course Administrator
Phone: +61-(0)8-8222-5116
E-mail: Lorna.Mirabelli@sa.gov.au

ANZ Hyperbaric Medicine Group 2018 Course report

The 2018 two-week ANZHMG Introductory Course in diving and hyperbaric medicine moved to Fremantle, Western Australia (WA) after many years at the Prince of Wales Hospital, Sydney. The course, convened by Ian Gawthrope and Neil Banham was attended by 27 participants. Course activities included a visit to the Submarine Escape facility at HMAS Stirling, a day focussed on diver retrieval at the Jandakot Royal Flying Doctor Service base and a morning on the water rescuing the injured diver. Faculty included staff from the local Fiona Stanley Hospital Hyperbaric Medicine Unit as well as 'wise men from the East' including Simon Mitchell, David Smart, Mike Bennett, Andrew Fock, Ken Thistlethwaite and Iestyn Lewis – many thanks to all! The course prize was awarded to Dr Alan Gault, WA.

A comprehnsive list of contact details for all Hyperbaric Units in Australia and New Zealand will soon be available on the SPUMS website.

Dr Neil Banham, Chair ANZHMG
neil.banham@health.wa.gov.au

A new hyperbaric facility for Tasmania

Saturday 05 May marked a milestone that was particularly satisfying for the author personally; the commencement of a new era for the staff of the Royal Hobart Hospital Department of Diving and Hyperbaric Medicine. Our 14 m long, 66 tonne, rectangular, triple-lock hyperbaric chamber was lifted into its new home on the third floor of the Royal Hobart Hospital redevelopment, where it will be located for the next 30+ years. Except for the Royal Darwin Hospital, the RHH hyperbaric facility is the last teaching hospital in Australia to receive an upgrade to a rectangular-format hyperbaric chamber. We have had the advantage of learning from other facilities around Australia and New Zealand, and our staff have been fortunate to be involved from the commencement of the project. This has allowed us to design the chamber to meet the foreseeable future needs of the Tasmanian community, but also to design a department around the chamber that will allow excellence in patient care and unique research capabilities.

The chamber is a triple-lock facility with compartment 1 working pressure up to 4 ATA, and compartment 2 (entry lock) and compartment 3 able to be pressurised to 6 ATA. Hobart's current medical treatment capacity is five patients simultaneously. Compartment 1 will allow up to eight patients to be treated simultaneously. Compartment 3 will accommodate two stretcher patients or four patients sitting, and has mixed gas capability. Also, we will maintain possession of our two 42-inch monoplace chambers for ultimate flexibility in delivery of patient care. The department has been designed to maximise efficiency of patient flows and amenity for emergency and routine treatment, wound care and also equipment transit and cleaning. There is even allocated space to house the Carl Edmonds library, which he so kindly donated to our facility in June 2014.

Bureaucrats initially questioned the need for a triple-lock chamber (given our existing twin-lock), but we countered their arguments with an engineering solution for dealing with emergencies at the same time as routine care. Imagine if it were that simple in operating theatres, where acute trauma cases lead to cancellation of elective surgery. In March 2017, the "*McCartney*" hyperbaric chamber (designed by Dr Peter McCartney, our founding director) was retired having served us well for 25 years. Since then until January 2020 when the new Hospital block will be opened, the department must operate a temporary facility.

The last three years has seen huge upheaval in our department. In addition to designing the future department from scratch, our staff weathered a complete demolition and rebuild within our existing department, whilst still maintaining full clinical services. At one stage, we had five operational chambers (two twin-lock multiplace and three monoplace chambers). The dust has now settled, and we are operating a 30+ year old chamber, before moving to the new department. I am proud of all our hyperbaric medical team at RHH, for their patience, good humour, professionalism and, most importantly, focus on patient care during challenging times. The chance to write the future happens infrequently, and this is currently our privilege.

David Smart, Medical Co-Director, Royal Hobart Hospital Department of Diving and Hyperbaric Medicine
david.smart@ths.tas.gov.au

Key words
Hyperbaric medicine; General interest

Figure 1
Hyperbaric chamber being lifted into the 3rd floor of the new Royal Hobart Hospital; photo courtesy David Smart

Figure 2
Professor David Smart in the "*McCartney*" chamber

EUBS Notices and news

EUBS notices and news and all other society information is to be found mainly on the society's website: www.eubs.org

EUBS 2018 Annual Scientific Meeting

In 2018, the EUBS ASM will be held in Durban, South Africa. The second Tricontinental Meeting on Diving and Hyperbaric Medicine (TRICON2018) will be held 23–29 September, co-organised with SPUMS and SAUHMA. There will be no other EUBS meeting in 2018. This will be a once-in-a-long time occasion to meet people you mostly only hear or read about, and in a most exciting environment. Besides enjoying a full academic programme, you will have the opportunity to go diving with sharks on Aliwal Shoal, or drive among elephants, rhinoceros, giraffe and buffalo in Hluhluwe National Park.

For full information go to http://www.tricon2018.org (please remember to type the "http://" otherwise you may receive a security warning when using certain web browsers); or by visiting the EUBS website. Earlybird registration has now ended, but abstracts are still being accepted. Please submit an abstract; your input is an important part of the success of our meetings.

EUBS Affiliate Society agreements

For the year 2018, Affiliate Agreements have been made with the following scientific societies in the field of diving and hyperbaric medicine:
Belgian Society for Diving and Hyperbaric Medicine (SBMHS-BVOOG; www.sbmhs-bvoog.be)
French Society for Diving and Hyperbaric Medicine (MEDSUBHYP; www.medsubhyp.com)
German Society for Diving and Underwater Medicine (GTUEM, www.gtuem.org)
Italian Society for Diving and Hyperbaric Medicine (SIMSI; www.simsi.org)
Swiss Society for Diving and Hyperbaric Medicine (SUHMS; www.suhms.org)
Undersea and Hyperbaric Medicine Society (UHMS; www.uhms.org)
Scott Haldane Foundation - Diving Medicine Education, The Netherlands (www.scotthaldane.org)

If you are a member of an Affiliate Society you can benefit from a 10% discount on EUBS membership – please indicate this on the membership application or renewal form, and upload a PDF or scanned copy of your "*other membership*" proof). As a member of EUBS, you will also benefit from a membership discount with UHMS, so you have many reasons to start connecting professionally around the world.

European Data Protection Law: General Data Protection Regulation

The Data Protection Directive (officially Directive 95/46/EC) on the protection of individuals with regard to the processing of personal data (PII (US) and on the free movement of such data) is an European Union (EU) directive adopted in 1995 which regulates the processing of personal data within the EU. It as been an important component of EU privacy and human rights law.

The General Data Protection Regulation (GDPR; EU 2016/679) is a regulation in EU law on data protection and privacy for all individuals within the European Union. It also addresses the export of personal data outside of the EU. It is an important component of EU privacy and human rights law.

The GDPR aims primarily to give control to citizens and residents over their personal data and to simplify the regulatory environment for international business by unifying the regulation within the EU. The GDPR was adopted in April 2016, superseding the 1995 Data Protection Directive, and will be enforceable starting on 25 May 2018. From this date, all businesses and organisations within the EU should have reviewed data protection procedures and updated privacy policies.

EUBS has taken the necessary steps to comply with these regulations, and on the EUBS website you can now find and download a document formally describing the data that are being stored for each EUBS member, what is our policy for distributing these data and to whom, what your rights are as to the storage and use of your personal data by EUBS and how you can modify your data, either yourself or through the EUBS Secretary, peter.germonpre@eubs.org.

EUBS website

A new "*EUBS History*" section has been added under the Menu item "*The Society*". There is still some information missing in the list of EUBS Meetings, Presidents and Members-at-Large. Please dig into your memories and help us complete this list! By popular demand, EUBS Members can now also download the complete Abstract Book of previous EUBS Meetings from the Members' Area.

The EUBS President's message

Jacek Kot, President, EUBS

Usually I notice the change of season from Winter to Spring by having less carbon monoxide intoxication cases in my hospital. This is perhaps a somewhat narticulate observation clearly explained by the most frequent origin of poisoning in our geographic location, which is home gas heaters.[1] Then as Spring evolves, the number of patients with sudden deafness increases; once again, an observation that has been confirmed scientifically.[2] I am not sure whether you share the same feeling as I have, but Spring usually finds me tired and somewhat bored by long winter night duties at the hospital. I felt this earlier this year than previously. Fortunately, science has again helped me in finding an excuse – the long-term observation that Spring is coming earlier year by year![3] Yet another change in my clinical practice – more patients with severe necrotising soft tissue infections and decompression injuries – will enhance the need for summer holidays and preparation for the next Annual Scientific Meeting (ASM) of our Society.

This year, the ASM will be shared again with our partner Societies, namely SPUMS and the South African Underwater and Hyperbaric Medicine Association (SAUHMA), at TRICON2018 in Durban, South Africa. I am confident that those who remember TRICON2013 on Reunion island will be glad to repeat this experience in a different location and those who were not there should be willing to taste the atmosphere of joint activity. For sure this is the best platform for personal discussions, charging your internal batteries by exchanging ideas and finding new partners for future projects. Go to the TRICON2018 website at http://www.tricon2018.org and register as soon as possible.

I would also like to remind you about the elections in our Society. One for Member-at-Large and the other for the President-Elect. Even if the EUBS has some kind of positive inertia which keeps it stable in its operation, new blood is vital and always welcome. Remember, whoever your proposed candidates will be, either young and revolutionary or old and conservative (or any other combination of those), this is your call. Do not miss it, check the website frequently and read the News being distributed directly to your mailbox.

Now, enjoy the current issue of the Journal online.

References

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3 Crabbe RA, Dash J, Rodriguez-Galiano VF, Janous D, Pavelka M, Marek MV. Extreme warm temperatures alter forest phenology and productivity in Europe. Sci Total Environ. 2016; 563–564:486–95. doi: 10.1016/j.scitotenv.2016.04.124.

Key words
Medical society; General interest

EUBS Executive Committee 2018 elections

Around the time of publication of this issue of DHM, the election process for the 2018 ExCom members (Vice-President, Member-at-Large) of EUBS will have commenced.

Member-at-Large: we will be saying goodbye to Karin Hasmiller as Member-at-Large 2015; however, she will remain working with ExCom as a Member of the DHM Journal Governance Committee and in the Research and Education Committee. ExCom does extend their thanks to Karin for her work for the Society thus far. Members-at-Large serve for three years.

Vice-President: during the next EUBS General Assembly (during TRICON2018), Ole Hyldegaard will take over the Presidency of EUBS from Jacek Kot. Therefore, we need to elect a new Vice-President, someone who is ready to serve the Society for 12 years (three each as Vice-President, President, Immediate Past President, and Past President). Such are the directives set forth by our Constitution.

Candidates for these positions will be presenting themselves on the EUBS website with a picture and short CV, and you will receive soon, by e-mail, an internet ballot where you can cast your vote. If you have not received such an e-mail by the end of June, please notify us at secretary@eubs.org, and we will work with you to find out the reasons why. As the system works via e-mail, it is possible the message might end up in your spam folder. There may be other reasons but usually we are able to solve them.

Scott Haldane Foundation

Dedicated to education in diving medicine, the Scott Haldane Foundation has organized more than 250 courses over the past 20 years, increasingly targeting an international audience with courses worldwide. The courses Medical Examiner of Diver (part I and II) 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).

SHF Course Calendar second half 2018
14–15 September: HBOT and decompression (level 2d), Nunspeet, NL
09–16 November: Medical Examiner of Divers part 1, Palau
16–23 November: 26th In depth course, Diving Medicine (level 2d), Palau
23–30 November: 26th In depth course, Diving Medicine (level 2d), Palau
tbd: Refresher course, Organization diving medical, NL
On request: Internship different types of diving (DMP), NL
On request: Internship HBOT (DMP certification), NL/Belgium

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

Hyperbaric Oxygen, Karolinska

Welcome to: http://www.hyperbaricoxygen.se/
This site, supported by the Karolinska University Hospital, Stockholm, Sweden, offers publications and high-quality lectures from leading investigators in hyperbaric medicine. Please register to obtain a password via e-mail. Once registered, watch online, or download to your iPhone, iPad or computer for later viewing.
For further information contact:
E-mail: folke.lind@karolinska.se

German Society for Diving and Hyperbaric Medicine (GTÜeM)

An overview of basic and refresher courses in diving and hyperbaric medicine, accredited by GTÜeM according to EDTC/ECHM curricula, can be found on the website:
http://www.gtuem.org/212/Kurse_/_Termine/Kurse.html

DAN Europe

DAN Europe has a fresh, multilingual selection of recent news, articles and events featuring DAN and its staff.
Go to the website: http://www.daneurope.org/web/guest/

Capita Selecta Diving Medicine Academic Medical Centre, University of Amsterdam, The Netherlands Programme 2018–2019

The Capita Selecta Diving Medicine annually offers symposia on diving medicine presented by speakers of national and international renown to a multinational audience of diving physicians, paramedics and highly educated diving instructors. The level of the presented material is advanced, i.e., Level 1 and 2d, and often beyond that. The lectures are in English.

27 October 2018: The ageing diver
Topics include the physiology of the healthy, ageing heart, lung and muscular system, clinical aspects of diving – cardiology, ageing of sensory system and the brain, DCI and age, the medical exam of the older diver.
Speakers include: Olga de Bakker, NL; Jacques Regnard, FR; Rienk Rienks, NL and Nico Schellart, NL

Registration opens 08 July
www.capitaselectaduikgeneeskunde.nl

30 March 2019: Diving medicine of women, children and divers with a disability
Topics include menstruation, pregnancy, menopause; mental and physical maturation (cognition and psychology, CNS, muscular system and skeleton) of children and adolescents; diving with mental and physical disabilities and, for all groups, safety aspects.
Speakers include: Selina Haas, AT, Ulrike Preiml, AT and Guy Vandenhoven (BE)

British Hyperbaric Associ Annual Scientific Meeting

Dates: 08–09 November
Venue: Danubius Hotel Regent's Park, London
07 November: Historical event
Hosts: London Diving Chamber
http://www.londondivingchamber.co.uk/
Further information:
http://www.ukhyperbaric.com/

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

Royal Australian Navy Medical Officers' Underwater Medicine Course 2018

Dates: 08–19 October
Venue: HMAS Penguin, Sydney

The MOUM course seeks to provide the medical practitioner with an understanding of the range of potential medical problems faced by divers. Emphasis is placed on the contraindications to diving and the diving medical assessment, together with the pathophysiology, diagnosis and management of common diving-related illnesses. The course includes scenario-based simulation focusing on the management of diving emergencies and workshops covering the key components of the diving medical.

Cost: AUD1,355 without accommodation
(tbc with accommodation and meals at HMAS Penguin)

For information and application forms contact:
Rajeev Karekar, for Officer in Charge,
Submarine and Underwater Medicine Unit
HMAS Penguin
Middle Head Rd, Mosman
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Fax: +61-(0)2-9647-5117
E-mail: Rajeev.Karekar@defence.gov.au

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The Historical Diving Society 2018 Annual Conference

Date: 03 November
Venue: RNLI College, Poole, UK

The HDS was formed in the UK in 1990 with the aim of preserving and protecting diving heritage. Since then the Society has grown into an international organisation with affiliated national societies across the world. It produces a newsletter, the *Historical Diving Times*, and the *International Journal of Diving History*. It publishes facsimile monographs of important works on diving.

Email: chairman@thehds.com
Websites: www.thehds.com
www.divingmuseum.co.uk

Asian Hyperbaric and Diving Medical Association 14th Annual Scientific Meeting

Dates: 27–28 July 2018
Venue: Anantara Riverside Bangkok Resort, Thailand

Guest Speaker: Assoc Prof Dr Jacek Kot, General Secretary of the European Committee for Hyperbaric Medicine (ECHM).

Pre-ASM courses: 26 July 2018
Topics: 1. Diving Medicine Refresher Course (for doctors with EDTC Level 2D or equivalent).
2. Wound Care Course

Call for abstracts: closes 15 June 2018
Registration: http://www.ahdma.org/annual-scientific-meeting/asm-2018/

20th International Congress on Hyperbaric Medicine 2020

Dates: 13–16 September 2020
Venue: Rio de Janeiro, Brazil
For preliminary information contact:
Dr Mariza D'Agostino Dias
Email: mariza@hiperbarico.com.br

Advertising in *Diving and Hyperbaric Medicine*

Companies and organisations within the diving, hyperbaric medicine and wound-care communities wishing to advertise their goods and services in *Diving and Hyperbaric Medicine* are welcome. The advertising policy of the parent societies appears on the journal website:
www.dhmjournal.com

Details of advertising rates and formatting requirements are available on request from:
E-mail: editorialassist@dhmjournal.com

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P O Box 347, Dingley Village
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E-mail: hdsaustraliapacific@hotmail.com.au
www.classicdiver.org

Diving and Hyperbaric Medicine: Instructions for Authors

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, 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.

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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 user name 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 word count); include an informative **Abstract** of no more than 300 words (excluded from word count); structure of the article and abstract is at the author(s)' discretion.

Case reports, Short communications, Work in progress reports, etc: maximum 1,500 words, and 20 references (excluded from word count); include an informative **Abstract** (structure at author's discretion) of no more than 200 words (excluded from word count).

Educational and historical articles, Commentaries, Consensus and other meeting reports, etc., 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.

Formatting of manuscripts

All submissions must comply with the requirements set out in the full instructions on the DHM website. Non-compliant manuscripts will be suspended whilst the authors correct their submission. Guidance on structure for the different types of articles is given above.

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

- Instructions for authors
- DHM Key words 2018
- DHM Mandatory Submission Form 2018 (downloadable)
- Trial design analysis and presentation
- Conflict of interest statement
- English as a second language
- Guideline to authorship in DHM 2015
- Helsinki Declaration revised 2013
- Is ethics approval needed?

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

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 onto the SPUMS website, click on "*Resources*" then on "*GTÜeM 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.

DIVER EMERGENCY SERVICES PHONE NUMBERS

AUSTRALIA
1800-088200 (in Australia, toll-free)
+61-8-8212-9242 (International)

NEW ZEALAND
0800-4DES-111 (in New Zealand, toll-free)
+64-9-445-8454 (International)

ASIA
+81-3-3812-4999 (Japan)

EUROPE
+39-6-4211-8685 (24-hour hotline)

UNITED KINGDOM
+44-7740-251-635

SOUTHERN AFRICA
0800-020111 (in South Africa, toll-free)
+27-828-106010 (International, call collect)

USA
+1-919-684-9111

The DES numbers (except UK) are generously supported by DAN

DAN ASIA-PACIFIC DIVE ACCIDENT REPORTING PROJECT

This project is an ongoing investigation seeking to document all types and severities of diving-related incidents. All information is treated confidentially with regard to identifying details when utilised in reports on fatal and non-fatal cases. Such reports may be used by interested parties to increase diving safety through better awareness of critical factors. Information may be sent (in confidence unless otherwise agreed) to:

DAN Research
Divers Alert Network Asia Pacific
PO Box 384, Ashburton VIC 3147, Australia
Enquiries to e-mail: research@danasiapacific.org

DAN Asia-Pacific NON-FATAL DIVING INCIDENTS REPORTING (NFDIR)

NFDIR is an ongoing study of diving incidents, formerly known as the Diving Incident Monitoring Study (DIMS). An incident is any error or occurrence which could, or did, reduce the safety margin for a diver on a particular dive. Please report anonymously any incident occurring in your dive party. Most incidents cause no harm but reporting them will give valuable information about which incidents are common and which tend to lead to diver injury. Using this information to alter diver behaviour will make diving safer.

The NFDIR reporting form can be accessed on line at the DAN AP website:
www.danasiapacific.org/main/accident/nfdir.php

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