Diving and Hyperbaric Medicine

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SPUMS

Volume 50 No. 3 September 2020

EUBS

Endothelial function after air diving

Chain of events in scuba diving fatalities

Pressure in water-filled endotracheal tube cuffs

Assessing insulin sensitivity during HBOT

In-chamber sound levels during HBOT

HBOT in breast late radiation tissue injury

HBOT in pelvic late radiation tissue injury

Thermal balance in divers with spinal injury

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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

This is the third issue of *Diving and Hyperbaric Medicine* (DHM) in a profoundly turbulent year. The SARS-CoV-2 pandemic has caused global mayhem in a way that many (except students of history) had never contemplated. This writer, living in New Zealand and working within the sharp-end medical workforce, has been thus far lucky enough to be largely unaffected in any material way; but there are countless people everywhere who have lost family, friends or livelihoods. We should never lose sight of the terrible cost of this event.

There have been unexpected consequences for the journal. One has been a profound increase in the number of submissions received. By the end of July we had received as many submissions in 2020 as we did for the entire year in 2019. There are no economies of scale in processing submissions, and each new submission adds linearly to the workload. I wish to acknowledge the hard work of our editorial assistant Nicky Telles in coping with what has effectively been a doubling of work, and also the support of the society executive committees who have backed the increased journal activity through financial support. A healthy number of submissions has been a good 'problem' to have, but it stretches resources, including our cohort of semi-regular reviewers who I gratefully thank for almost always accepting review invitations. The most obvious explanation for the surge in submission activity is an almost paradoxical increase in available time for author activities over the pandemic period during shut-downs and reduced clinical loads as hospitals have constrained elective work. An increase in my own non-clinical time has certainly helped me cope with the increase in journal work.

Another consequence of the pandemic for our medical field has been the loss of our usual opportunities to gather, learn, exchange ideas and socialise. Specifically, the annual scientific meetings of EUBS (Prague), SPUMS (New Zealand), UHMS (San Diego), and the AHDMA (Viet Nam) were all cancelled this year, though several of these meetings hopefully will go ahead in the same venues in 2021. The need to cancel carefully planned meetings will have been heart-breaking for convenors, but we trust that members of all societies will be motivated to make those meetings well-attended and memorable when they eventually do take place. Our societies are the glue that holds the field together and we must support them through membership and meeting attendance in these financially difficult times.

Returning to the journal, in late June 2020 the 2019 impact factor (IF) was released revealing an increase from just under 1.2 to 1.5. An IF of 1.5 is seriously good for a niche specialty journal like DHM. It maintains our pre-eminent position among comparable journals, though for reasons probably well known to most readers the IF is a flawed and potentially capricious metric for comparing journals, and I advise members against becoming fixated on it. It may fluctuate in future years; especially given we are publishing greater numbers of papers as a consequence of greater numbers of submissions as described above. For now, it provides us reassurance that we at least on a satisfactory track.

In this issue of the journal we publish our first paper related to SARS-CoV-2; a pragmatic attempt to guide evaluation of diving candidates or divers returning to diving after suffering a Covid-19 infection. Charlotte Sadler and the San Diego group promulgated the basic elements of this guideline on-line earlier in the pandemic, and this paper provides a reasoned explanation for their recommendations. The on-line version will continue to be updated as required. Diving by Covid-19 sufferers who may exhibit residual pulmonary effects is likely to become a vexed issue in our community.

A second guideline appearing in this issue is the result of a SPUMS workshop convened with the aim of rationalising and modernising the approach to screening divers for cardiovascular disease. This is an important subject given evidence that the diving population is aging, and that a cardiac event is the disabling injury in a significant proportion of diving fatalities. The workshop held at the SPUMS Annual Scientific Meeting in 2019 generated the key elements of the guideline which was then refined by a focus group led by Australian cardiologist Nigel Jepson and which included diving academics and cardiologists from both Australia and Europe. This guideline is intended to fill a void that has opened up as evaluation for coronary disease has evolved over the last 20 years.

The remainder of this issue is a true blend of articles focusing on the diving and hyperbaric sides of the field, with some fascinating case reports. There is a thought-provoking review written by Dick Clarke of the debate over whether decompression illness should be treated in monoplace chambers. This has some relevance to the emerging situation in the USA where the number of hyperbaric units continues to expand but the number of units providing a 24-hour emergency service for treating divers (or which treat divers under any circumstances) slowly shrinks.

Professor Simon Mitchell
Editor, Diving and Hyperbaric Medicine Journal

Front cover

Composite multi-time-lapse light-painting photo from the Pearse Resurgence cave, Nelson New Zealand, pushed to 245 m by Harris and Challen 2020. The buoyed structure is a gas filled habitat anchored in the cave at 40 m for dry decompression stops; by Simon Mitchell, who appears (slightly blurred) in his own photo.

Original articles

The effect of hyperbaric oxygen treatment on late radiation tissue injury after breast cancer: A case-series of 67 patients

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Key words
Radiotherapy; Soft-tissue radionecrosis; Hyperbaric medicine; Pain

Abstract
(Spruijt NE, van den Berg R. The effect of hyperbaric oxygen treatment on late radiation tissue injury after breast cancer: A case-series of 67 patients. Diving and Hyperbaric Medicine. 2020 September 30;50(3):206–213. doi: 10.28920/dhm50.3.206–213. PMID: 32957121.)
Introduction: Late radiation tissue injury (LRTI) after breast cancer may benefit from hyperbaric oxygen treatment (HBOT). This study aimed to report the LRTI symptom scores up to 12 months after HBOT and identify risk factors for poor scores.
Methods: A case-series of 67 patients who underwent a mean of 44 sessions of HBOT was analysed. LRTI symptoms were scored at four time points using the LENT-SOMA scale (Late Effects in Normal Tissues – Subjective, Objective, Management, and Analytic), a visual analog scale for pain, and the range of shoulder motion.
Results: Between starting HBOT and 12 months after HBOT 57 patients (85%) reported at least one point improvement in their LENT-SOMA score. Median pain and fibrosis scores improved significantly between the start and end of HBOT ($P < 0.001$), and remained stable three and 12 months after HBOT. The median breast oedema score improved significantly 12 months after HBOT ($P = 0.003$). Median shoulder abduction increased significantly from 90 to 165 degrees ($P = 0.001$) and median shoulder anteflexion increased significantly from 115 to 150 degrees ($P = 0.004$). Various risk factors were identified for poor scores despite HBOT; the most common risk factor was a poor score at start of HBOT.
Conclusions: In this case-series, patients who underwent HBOT for LRTI after breast cancer reported significant improvement in pain, fibrosis, oedema, and shoulder movement. The improvement persisted up to 12 months after HBOT. A poor score at the start of HBOT was predictive for a poor score 12 months after HBOT.

Introduction

Breast cancer is the most common form of cancer affecting women in the Netherlands. One in seven women will get breast cancer during their lives.[1] It is often diagnosed at an early stage and has a good prognosis; the five-year survival is 87%.[1] Survivors face several late sequelae of treatment including late radiation tissue injury (LRTI). Radiation injury can be divided into acute and late tissue injury.[2] Acute injury occurs during or a few months after radiotherapy and is usually self-limiting, including haematoma, dermatitis, breast pain and implant infection.[3] Late injury is that persisting at six months or occurring over six months after radiotherapy, and usually worsens with time.[4]

The most common LRTI symptoms after breast cancer are pain, oedema, fibrosis, and limited range of motion of the arm at the shoulder joint.[5] The prevalence of LRTI increases with time.[4,6,7] For example, five years after radiotherapy for breast cancer 15–19% of patients have moderate to marked breast fibrosis, and 10 years after radiotherapy the incidence increases to 22–28%.[7] Although breast pain is common after breast-conserving surgery and radiotherapy (47% of patients report having pain), over 85% of patients consider it tolerable.[8]

For those who suffer from LRTI, hyperbaric oxygen treatment (HBOT) has been shown to improve symptoms.[9–11] Both the European Committee for Hyperbaric Medicine and the Undersea and Hyperbaric Medical Society accept LRTI as an indication for HBOT.[2,12] During HBOT, stem cells within irradiated tissues are induced and mobilised and angiogenesis is stimulated, improving tissue oxygenation and decreasing fibrosis.[2] The objective of this study was to report the effects of HBOT on LRTI symptoms after breast cancer up to 12 months after completing HBOT and to identify risk factors for persistent symptoms after HBOT.

Methods

Data were collected prospectively, recorded in the patients' medical records and analysed retrospectively. In accordance with the Health Code of 2005 based on the Code of Good Conduct 1995, our institutional review board grants a universal waiver for retrospective chart reviews, such as this study.

Figure 1
Diagram showing the compression, oxygen breathing periods, air breaks, and decompression time of a treatment session. msw – metres' seawater equivalent 'depth'

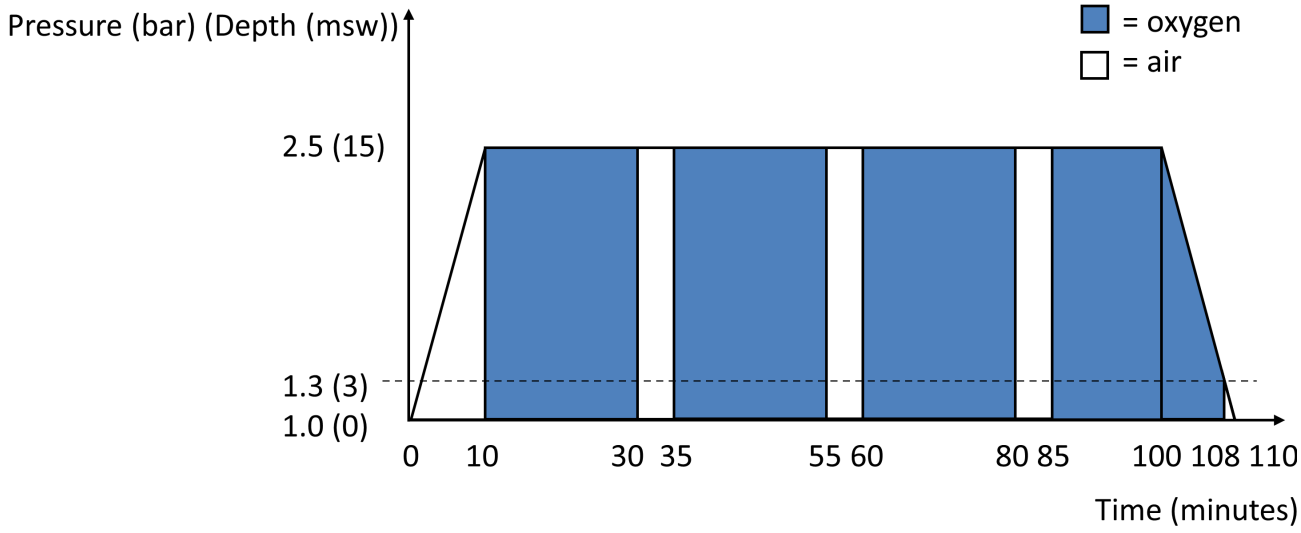


Table 1
Definition of LENT-SOMA scores[5]

Symptom	0	1	2	3	4
Pain	Absent	Rarely, minimal	Intermittent, tolerable	Permanent, intense	Always, excruciating
Fibrosis	None	Barely palpable	Definite increased density and firmness	Marked density, retraction, fixation	
Breast oedema	None	Asymptomatic	Symptomatic	Secondary dysfunction	

All patients who were referred for HBOT for LRTI after breast cancer were candidates for HBOT. Patients underwent assessment of their fitness for hyperbaric exposure and were prescribed 40 sessions of HBOT. Patients were treated in a multi-place hyperbaric chamber capable of accommodating up to 12 patients (IHC Hytech, Raamsdonkveer, the Netherlands) at an ambient pressure of 253 kPa (2.5 atmospheres absolute). At this pressure, 100% oxygen was breathed via a mask during four periods for a total of 83 minutes, interspersed by three 5-min air breaks (Figure 1). Including compression and decompression time, the total duration of each session was 110 minutes. Patients underwent one session per day, five days per week. Per protocol, 40 sessions were prescribed, but treatment was continued until patients chose to prematurely stop the treatment, the symptoms did not show any further improvement, or the maximum number of treatments which are reimbursed by the health insurance was reached (60 sessions). Side effects of HBOT were recorded including problems equalising the ears, changes in vision and fatigue.

LRTI symptoms were scored by two dedicated nurses at four time points: in person before commencing HBOT and upon completion of the course of HBOT, and by telephone three and 12 months after completing HBOT. Three scoring systems were used to quantify LRTI symptoms: 1) the LENT-SOMA (Late Effects in Normal Tissues – Subjective, Objective, Management, and Analytic) scoring system;[5] 2) a visual analog scale (VAS) to quantify the intensity of pain; and 3) the range of motion in degrees at the shoulder joint on the affected side. Since the range of motion could only be assessed clinically, this was only scored at the start and end of HBOT, and not at the three- and 12-month follow-ups. The LENT-SOMA scoring system is considered the most effective validated tool to analyse the late effects of radiotherapy.[13] It is comprised of 12 questions about pain, oedema, fibrosis, telangectasias, atrophy/retraction and ulceration, each of which is scored on a 2–5-point scale. In this study we only report on the primary symptoms for referral to our clinic which were pain, breast oedema, and/or fibrosis (Table 1). The LENT-SOMA scores for pain (0–4), breast oedema (0–3), and fibrosis (0–3) were also summed (score range 0–10).

STATISTICS

We used descriptive statistics to describe the patient characteristics, the HBOT course, side-effects of HBOT and outcomes after HBOT. We reported the median scores of the LRTI symptoms at each time point. The Kruskal-Wallis

Table 2
Patient characteristics (n = 67)

Characteristic	Mean (range)
Age (years)	59 (43–79)
Body mass index (kg·m^{-2})	27.8 (18.8–43.9)
	***n* (%)**
Smoking:	
Never	25 (37)
Stopped	34 (51)
Current	8 (12)
Breast surgery:	
Breast-conserving	50 (75)
Mastectomy	17 (25)
Axillary nodes:	
Sentinel node removal	36 (54)
Axillary clearance	25 (37)
Axillary radiotherapy	6 (9)
Chemotherapy	46 (69)
Time since radiotherapy:	
< 1 year	26 (39)
1–3 years	20 (30)
3–5 years	2 (3)
> 5 years	19 (28)

test was used when comparing the median pre- and post-HBOT scores of more than two groups with a P-value limit of significance of < 0.05. If this overall test was significant, the Mann-Whitney U test was then used to compare pairs of groups. The Spearman correlation test was used for evaluation of correlations between risk factors and the LENT-SOMA and VAS scores 12 months after HBOT. Step-wise multivariate logistic regression analyses were used to assess associations between risk factors and LENT-SOMA pain, fibrosis, or breast oedema score ≥ 2 and VAS score ≥ 5 at 12 months after HBOT. No correction for multiple testing was employed. All analyses were performed using SPSS software (SPSS inc. version 22.0, Chicago, IL).

Results

Between November 2015 and December 2017 a total of 101 patients presented with LRTI after breast cancer; 97 were offered HBOT and 91 accepted treatment. Ten of these patients (11%) prematurely stopped the treatment: three due to recurrent breast cancer; four due to anxiety/hyperventilation; and three for other reasons. The supervising physician was always consulted when patients prematurely stopped treatment. Of the 81 patients who completed the HBOT, data were missing for 14 patients (17%) and complete for 67 patients (83% follow-up).

All 67 patients were women whose characteristics are listed in Table 2. Fifty patients (75%) had breast-conserving surgery, 36 patients (54%) only had sentinel node clearance, and 46 patients (69%) had chemotherapy. The time interval between radiotherapy and starting HBOT was less than one year for 26 patients (39%) and longer than five years for 19 patients (28%).

The patients underwent a mean of 44 HBOT sessions (range 26–60). During the HBOT five patients (8%) had difficulty equalising the ears requiring referral to an otorhinolaryngologist for grommets. Upon completion of the course of HBOT, 46 patients (69%) reported increased fatigue. Transient vision changes were reported in 56 patients (84%). Four patients (6%) had vision changes which persisted three months after completion of the HBOT and were referred to an ophthalmologist: two were found to have cataracts and two had refraction changes, probably not related to the HBOT, requiring new glasses.

IMPROVEMENT WITH HBOT

The median LENT-SOMA pain, fibrosis, their sum, and VAS scores improved significantly between start and end of HBOT (P < 0.001), and remained stable at three and 12 months after HBOT (Figure 2). Compared to the score at the start of HBOT, the median LENT-SOMA breast oedema score was not significantly lower at the end of HBOT (P = 0.188) nor after three months (P = 0.066), but was significantly lower 12 months after HBOT (P = 0.003).

Among the patients who had limited range of motion of the arm at the start of HBOT, median shoulder abduction increased significantly from 90 to 165 degrees (n = 22, P = 0.001) and median shoulder anteflexion increased significantly from 115 to 150 degrees (n = 19, P = 0.004) at the end of HBOT (Figure 3).

Between the start and end of HBOT, 54 patients (81%) reported at least one point improvement in their LENT-SOMA summed score. Between the end of HBOT and three months after HBOT, 29 patients (43%) reported at least one point improvement in their LENT-SOMA sum score. Between three and 12 months after HBOT 23 patients (34%) reported at least one point improvement in their LENT-SOMA sum score. Overall, between the start of HBOT and 12 months after HBOT, 57 patients (85%) reported at least one point improvement in their LENT-SOMA sum score, 44 (66%) reported at least one point improvement in their LENT-SOMA pain score, 50 (75%) reported at least one point improvement in their LENT-SOMA fibrosis score and 29 patients (43%) reported at least one point improvement in their LENT-SOMA breast oedema score.

UNIVARIATE CORRELATIONS

Associations between risk factors and LENT-SOMA and VAS scores 12 months after HBOT were analysed by Spearman correlations (Table 3). The LENT-SOMA pain score 12 months after HBOT was correlated with the axillary

Figure 2
Median LRTI symptom scores at the four time points (n = 67)

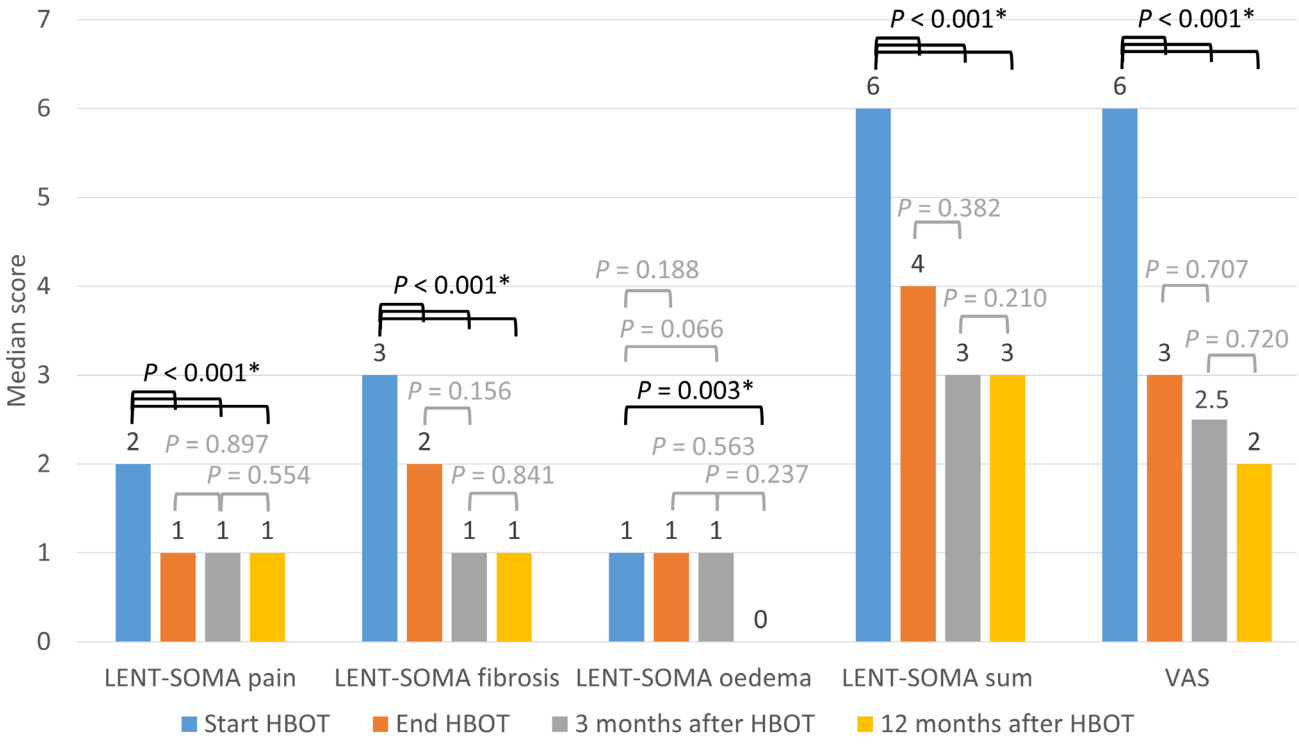
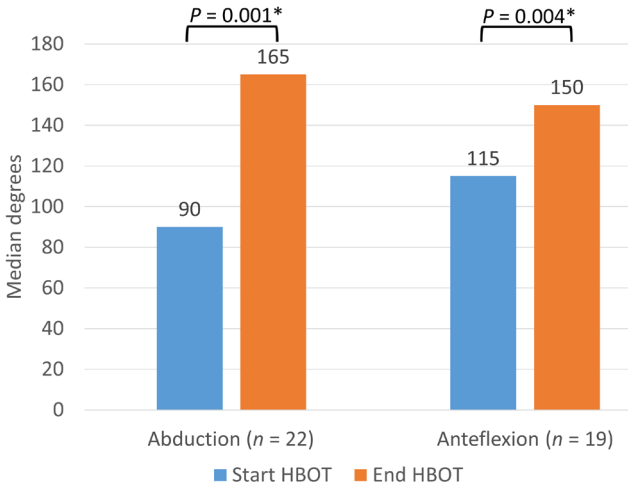


Figure 3
Range of motion from the shoulder joint on the affected side at the start and end of HBOT



node treatment (rho = -0.244, P = 0.046), sessions of HBOT (rho = 0.261, P = 0.033), and the LENT-SOMA pain score at the start of HBOT (rho = 0.684, P < 0.001). The LENT-SOMA fibrosis score 12 months after HBOT was correlated with chemotherapy (rho = -0.246, P = 0.045) and the number of sessions of HBOT (rho = 0.243, P = 0.048). The LENT-SOMA breast oedema score 12 months after HBOT was correlated with age (rho = -0.302, P = 0.013), body mass index (rho = 0.249, P = 0.043), time since radiotherapy (rho = -0.431, P < 0.001), and the LENT-SOMA breast oedema score at the start of HBOT (rho = 0.647, P < 0.001). The VAS score 12 months after HBOT was correlated with the axillary node treatment (rho = -0.248, P = 0.043), the number of sessions of HBOT (rho = 0.357, P = 0.003), and the VAS score at the start of HBOT (rho = 0.476, P < 0.001).

MULTIVARIATE ANALYSES

Step-wise multivariate logistic regression analyses showed significant associations between specific risk factors and LENT-SOMA pain, fibrosis, or breast oedema score ≥ 2 and VAS score ≥ 5 at 12 months after HBOT (Table 4).

The prevalence of a LENT-SOMA pain score ≥ 2 decreased from 38 patients (57%) at the start of HBOT to 22 patients (33%) 12 months after HBOT (Figure 4). In the multivariate analyses, only a LENT-SOMA pain score ≥ 2 at start HBOT was a risk factor for LENT-SOMA pain ≥ 2 at 12 months after HBOT (OR 15.0, 95% CI 3.1–72.2).

The prevalence of a LENT-SOMA fibrosis score ≥ 2 decreased from 59 patients (88%) at the start of HBOT to 31 patients (46%) 12 months after HBOT (Figure 4). In the multivariate analyses, only current smoking at the start of HBOT was a risk factor for LENT-SOMA fibrosis ≥ 2 at 12 months after HBOT (OR 10.2, 95% CI 1.2–88.4).

The prevalence of a LENT-SOMA breast oedema score ≥ 2

Table 3
Correlation between patient characteristics and LENT-SOMA and VAS scores in 67 women 12 months after HBOT; * = statistically significant; # = LENT-SOMA pain, fibrosis, breast oedema and VAS scores

Characteristic	LENT-SOMA pain	*P*-value	LENT-SOMA fibrosis	*P*-value	LENT-SOMA oedema	*P*-value	VAS	*P*-value
Age	-0.162	0.191	-0.208	0.092	-0.302	0.013*	-0.156	0.207
Body mass index	-0.090	0.471	0.053	0.672	0.249	0.043*	-0.004	0.976
Smoking	0.073	0.558	0.019	0.877	0.004	0.971	0.012	0.920
Breast surgery	-0.234	0.057	-0.100	0.421	-0.057	0.648	-0.129	0.297
Axillary nodes	-0.244	0.046*	-0.231	0.060	0.070	0.571	-0.248	0.043*
Chemotherapy	-0.065	0.604	-0.246	0.045*	-0.052	0.676	-0.077	0.537
Time since radiotherapy	-0.038	0.758	-0.142	0.253	-0.431	< 0.001*	-0.031	0.806
Sessions of HBOT	0.261	0.033*	0.243	0.048*	-0.063	0.612	0.357	0.003*
Respective score# at start HBOT	0.684	< 0.001*	-0.063	0.612	0.647	< 0.001*	0.476	< 0.001*

Table 4
Odds ratios for patient characteristics and LENT-SOMA and VAS scores in 67 women 12 months after HBOT; * = statistically significant; # = LENT-SOMA pain, fibrosis, breast oedema and VAS scores

Characteristic	LENT-SOMA pain ≥ 2	*P*-value	LENT-SOMA fibrosis ≥ 2	*P*-value	LENT-SOMA oedema ≥ 2	*P*-value	VAS ≥ 5	*P*-value
Age	0.064	0.800	0.926	0.336	0.388	0.533	1.029	0.477
Body mass index	1.069	0.301	0.435	0.510	1.082	0.298	0.989	0.125
Smoking	0.021	0.885	10.21	0.035*	0.039	0.843	1.831	0.489
Breast surgery	0.495	0.482	0.253	0.615	0.044	0.834	3.928	0.139
Axillary nodes	0.057	0.812	3.379	0.066	0.191	0.662	0.350	0.227
Chemotherapy	0.061	0.805	3.548	0.060	1.634	0.201	1.117	0.739
Time since radiotherapy	1.131	0.288	1.230	0.267	2.686	0.101	0.102	0.025*
Sessions of HBOT	0.227	0.634	2.844	0.092	0.125	0.724	1.047	0.314
Respective score# at start HBOT	15.00	0.001*	0.039	0.844	23.06	0.004*	6.295	0.027*

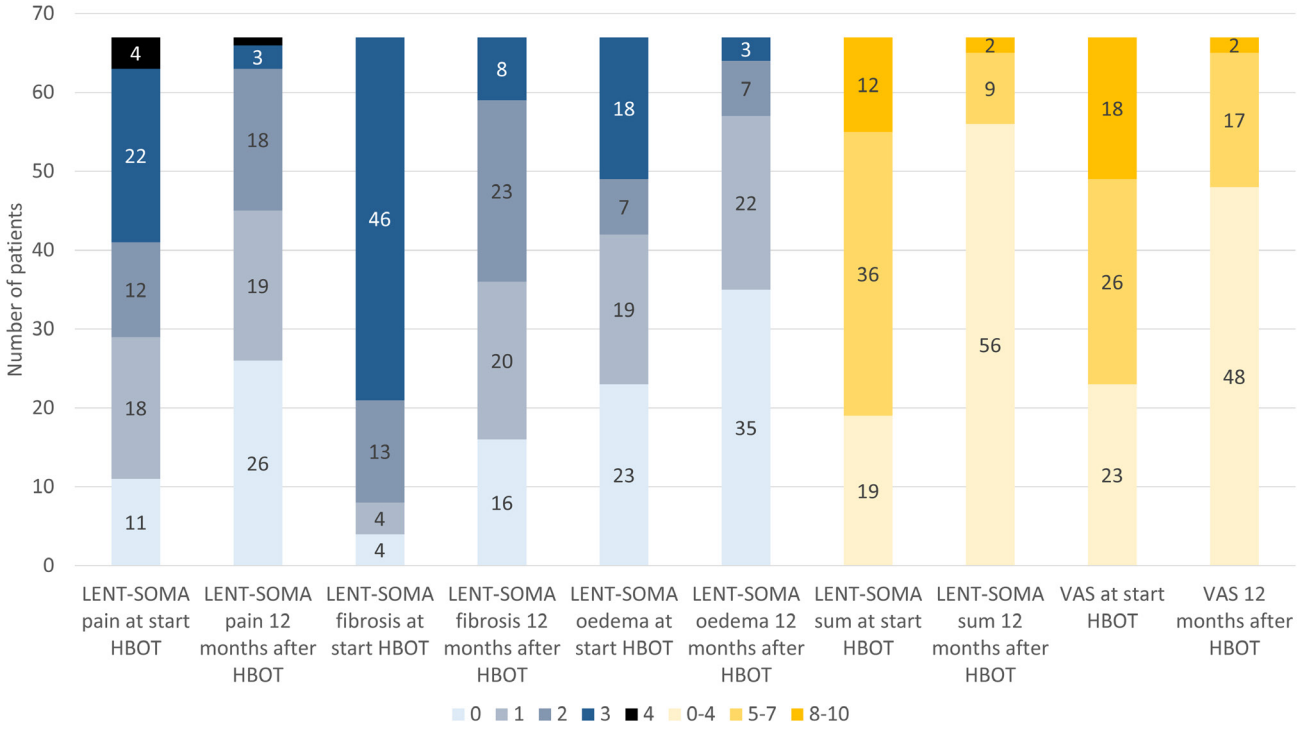
decreased from 25 patients (37%) at the start of HBOT to 10 patients (15%) 12 months after HBOT (Figure 4). In the multivariate analyses, only a LENT-SOMA breast oedema score ≥ 2 at the start of HBOT was a risk factor for LENT-SOMA pain ≥ 2 at 12 months after HBOT (OR 23.1, 95% CI 2.7–197.1).

The prevalence of a VAS score ≥ 5 decreased from 43 patients (64%) at the start of HBOT to 19 patients (28%) 12 months after HBOT (Figure 4). A VAS score ≥ 5 at the start of HBOT (OR 6.3, 95% CI 1.2–32.1) and a time interval ≥ 3 years between radiotherapy and start HBOT (OR 0.1, 95% CI 0.01–0.8) were risk factors for VAS score ≥ 5 at 12 months after HBOT in the multivariate analyses.

Discussion

In this study we add to the literature that shows that symptoms of LRTI after breast cancer improve after HBOT.[9–11] Pain and fibrosis scores improved between the start and end of HBOT, and remained stable up to 12 months after HBOT. Shoulder range of motion improved between the start and end of HBOT. Breast oedema scores did not improve significantly between the start and end of HBOT, but 12 months after HBOT they were significantly lower than at the start of HBOT. Risk factors were elucidated for high scores 12 months after HBOT. As in previous studies, we chose the cut-off point to be LENT-SOMA scores of ≥ 2.[6,14–16]

Figure 4
Prevalence of LRTI symptom scores at the start of HBOT and 12 months after HBOT in 67 women; colour codes refer to LENT-SOMA scores or score intervals as indicated



HBOT is not the first line of treatment for LRTI symptoms after breast cancer. Patients are referred for HBOT when analgesics, oedema therapy, and/or physiotherapy have not led to satisfactory symptom improvement. In general, patients are very pleased with the improvement in LRTI symptoms following HBOT. They do not expect all the symptoms to resolve with HBOT, and are delighted when the quality of life improves because of decreased pain, fibrosis, or oedema and increased range of motion from the shoulder. We noticed that some patients seem to benefit more from HBOT than others and sought predictive factors. Overall, the most common risk factor for higher scores 12 months after HBOT was a higher score at the start of HBOT (Table 3). Higher scores 12 months after HBOT were correlated with more sessions of HBOT, which corresponds to our intent to tailor the treatment duration to the severity of the patient's symptoms.[10]

Specifically, we found that higher pain scores (LENT-SOMA and/or VAS) at 12 months after HBOT were correlated with more aggressive axillary node treatment, a higher pain score at the start of HBOT, and more sessions of HBOT (Table 3). Multivariate analyses showed the risk factors for higher scores 12 months after HBOT were higher pain scores at the start of HBOT (OR 15.0 for LENT-SOMA score and OR 6.3 for VAS score) and a shorter interval between radiotherapy and the start of HBOT (OR 0.1). Other studies have shown that the LRTI pain score is associated with the radiotherapy dose[8,15,17] larger breast volume,[15] shorter time since radiation,[8] and hormone therapy.[8]

We found that higher fibrosis scores at 12 months after HBOT were correlated with chemotherapy and more sessions of HBOT (Table 3). Multivariate analyses showed the only risk factor for higher fibrosis scores at 12 months after HBOT was current smoking at the start of HBOT (OR 10.2). Other studies have shown that the LRTI fibrosis score is associated with chemotherapy,[17,18] larger irradiated volume,[18] and increased time after radiotherapy.[18]

We found that higher breast oedema scores at 12 months after HBOT were correlated with lower age, higher body mass index, a shorter time interval between radiotherapy and start of HBOT, and a higher oedema score at the start of HBOT (Table 3). Multivariate analyses showed the only risk factor for higher breast oedema scores at 12 months after HBOT was a higher oedema score at the start HBOT (OR 23.1). Other studies have shown that LRTI breast oedema scores were associated with axillary clearance, breast ptosis, and a bra cup size larger than C.[17]

The prevalence of LRTI symptoms after breast cancer varies per study, which can be expected given the variety of variables that may influence outcome.[19] A strength of this study is the heterogeneous population of patients who had undergone different surgeries and types of

radiotherapy representing the full spectrum of patients who suffer late sequelae of radiotherapy. On the other hand, the heterogeneous population is also a limitation of this study since it does not answer the question whether a specific patient will benefit from HBOT. Another limitation is that we do not know details about the radiotherapy our patients had, which has been shown to be an important prognostic factor for LRTI symptoms in other studies.[6,8,15,17,18] Since the follow-up scores after three and 12 months were collected by telephone, the reported oedema and fibrosis scores could not be verified with physical examination. Another limitation is that we performed a per-protocol evaluation rather than intention to treat (10 patients prematurely stopped treatment). Furthermore, we cannot definitively attribute the improvement of symptoms to the HBOT since we did not inventory concomitant treatment such as the use of analgesics, physiotherapy, or compression. Finally, the greatest limitation of this study is the lack of a control group preventing us from excluding a placebo effect. Given the known progression of LRTI symptoms with time,[4,6,7] we assume a control group who did not undergo HBOT would report worsening of symptoms during a 12-month follow-up period. Therefore, any future study should be in the form of a randomised, controlled trial.

In conclusion, we found significant improvement in pain, fibrosis, oedema, and shoulder movement scores among patients with LRTI after breast cancer who underwent HBOT. Shoulder movement was not followed up after completion of the HBOT. The improvement in pain, fibrosis, and oedema persisted up to 12 months after HBOT. Clinicians should be aware of this treatment option for patients with LRTI after breast cancer.

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Endothelial function may be enhanced in the cutaneous microcirculation after a single air dive

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Scuba diving; Circulation; Skin; Endothelium; Doppler; Iontophoresis

Abstract
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Introduction: The effects of scuba diving on the vessel wall have been studied mainly at the level of large conduit arteries. Data regarding the microcirculation are scarce and indicate that these two vascular beds are affected differently by diving.
Methods: We assessed the changes in cutaneous microcirculation before an air scuba dive, then 30 min and 24 h after surfacing. Endothelium-dependent and independent vasomotion were successively elicited by iontophoretic administration of acetylcholine and sodium nitroprusside respectively, and cutaneous blood flux was monitored by laser Doppler flowmetry.
Results: The response to sodium nitroprusside was significantly lower 30 min after surfacing than before diving (50 (SEM 6)% of the pre-dive values, $P = 0.0003$) and returned to normal values 24 h post-dive (102 (29)% of the pre-dive values, $P = 0.113$). When compared to pre-dive values, acetylcholine elicited a hyperaemia which was not statistically different 30 min after surfacing (123 (17)% of the pre-dive values, $P = 0.230$), but significantly increased 24 h post-dive (148 (10)% of the pre-dive values, $P = 0.005$).
Conclusion: Microvascular smooth muscle function is transiently impaired after diving. On the contrary, microvascular endothelial function is enhanced for up to 24 h after diving. This further suggests that the microcirculation reacts differently than large conduit arteries to scuba diving. The impact of modifications occurring in the microvascular bed on the physiological effects of diving merits further study.

Introduction

Impaired vasomotion after self-contained underwater breathing apparatus (scuba) diving was first reported in human large conduit arteries when decreased flow mediated dilation (FMD) of the brachial artery was observed after a single simulated air dive at 280 kPa.[1] Post-dive decreased FMD was further confirmed following a single open sea diving with various breathing mixtures including air,[2–5] nitrox[6] or trimix.[7] Although less investigated, the vasodilation induced by direct stimulation of the vascular smooth muscle (VSM) with nitric oxide (NO) donors is also decreased after diving.[3,5] Altogether, these data indicate that scuba diving impairs both the endothelium and the VSM in large conduit arteries. Additionally, impairment of FMD is maximal 30 min after surfacing and recovers progressively.[4] Indeed, FMD was still significantly reduced 48 h after the dive and needed three days for returning to pre-dive values.[4]

The microcirculation accounts for about 99% of blood vessels in adults. It is the smallest part of the vascular system and includes vessels with a diameter of less than 150 µm, i.e., arterioles, capillaries and venules. The microcirculation ensures the exchange of molecules between blood and tissues, as well as the regulation of blood pressure and the control of tissue fluid and oedema.[8] All of these actions are known to be influenced by the constraints induced during diving.[9,10] We and others have reported previously decreased microvascular reactivity after a single scuba dive.[3,5,11–13] However, we also reported that post-dive, macro- but not microvascular impairment was not present when bubble formation was prevented,[5] suggesting that scuba diving acts differently on these two vascular beds. In the present study, we assessed microvascular endothelium-dependent and independent reactivity and report data suggesting that microvascular endothelial function is enhanced 24 h after an air dive.

Methods

All experimental procedures were conducted in accordance with the Declaration of Helsinki and were approved by the Ethics Committee of the Haute Ecole Paul Henri Spaak, Brussels, Belgium (acceptance number:ISEK – 2009 – 12 –

14 – vs1). The study was conducted during training exercises performed by a local search and rescue dive unit. Dives were performed during September 2012 at Lake Lago D'Orta in Pettenasco, Italy, at an altitude of 294 m.

STUDY POPULATION

Six male divers (age 47 (SEM 4) years; body mass index 27 (0.8) $kg.m^{-2}$) volunteered for this study. The subjects were all experienced divers and had a valid medical certificate for diving at the time of the study. None had experienced decompression sickness (DCS) in the past. All but one were non-smokers. Prior to the experimental protocol, subjects abstained from any physical activity and diving for 72 h. The study participants were not taking medication except one subject who was receiving antihypertensive treatment. Subjects were not asked to fast; however, tea, coffee, alcohol and smoking were prohibited for 6 h prior to the test. Potential risks were explained to all subjects in detail and they gave their written informed consent before the experiment.

DIVE PROTOCOL

Each diver undertook an air dive in field conditions (water temperature 4°C). They were divided into three teams of two divers each. To account for any possible differences due to diving conditions or circadian variations which could influence microvascular reactivity, all dives were performed on the same day always in the morning. The diving site was located near the field laboratory, and divers were transported to the site by a power boat during a 10 min ride. The dive profile was planned as part of a training exercise for local rescue divers. Dive time was 20 min at 30 metres' fresh water (mfw) depth. Then, the ascent rate was 15 $m.min^{-1}$ with a 150 s pause at 12 mfw according to GAP DivePlanner (2003–2010), version 3.0.425.6, model RGBM, conservatism + 2 (recreational). Subjects used masks and fins and were dressed in dry suits with hood, boots, and gloves. Depth and dive time were monitored by each diver's personal dive computer.

LASER DOPPLER FLOWMETRY

To assess cutaneous microvascular endothelial function we performed iontophoresis with pharmacological agents coupled with laser Doppler flowmetry (LDF) as described previously.[3] Cutaneous blood flux (CBF) was recorded in a stable temperature room (22 (1)°C) before the subjects started to equip themselves for the dive and 30 min after surfacing. Each subject was therefore used as his own control. For each diving team, the two subjects were examined in parallel, the entire LDF measurement lasting 15 to 20 min. Before any measurement, subjects were asked to empty their bladder and to remain in the supine position until the end of the measurement. LDF measurements started after at least 15 min of rest. A multifibre laser probe (PF 450-PI, Perimed, Järfälla, Sweden) specially designed to make possible simultaneous current application and CBF recording was placed at the ventral side of the forearm, 5 cm below the elbow bend to avoid site to site variation.[14] CBF was measured from a small volume of skin (1 mm^3) using a laser beam at 780 nm wavelength which provides good skin penetration independently of skin color and oxygen saturation.[15] The probe was connected to a LD flowmeter (Periflux PF 5001, Perimed, Järfälla, Sweden).

IONTOPHORETIC STIMULATION

Iontophoresis is a method for non-invasive transdermal drug delivery based on the principle that a charged drug in solution will migrate across the skin under the influence of a direct low-intensity electric current.[15] It makes possible local delivery of small amounts of pharmacological agents, thus avoiding potential systemic effects while delivering drugs in the area of CBF measurement. The laser Doppler probe used had a chamber where we positioned a 0.6 cm^2 sponge filled with 100 µl of the drug solution. The iontophoretic sponge was connected to a battery powered current supply (Perilont PF 382; Perimed, Järfälla, Sweden), allowing for the delivery of regulated-intensity currents for programmable durations. It allowed the measurement of CBF in the middle of the stimulated region through a hole at the center of the sponge.

Endothelium-dependent vasodilation was first induced with a 1% acetylcholine (ACh) chloride solution administered with an anodal current (35 s, 0.10 mA). After CBF had returned to baseline values, a cathodal current (35 s, 0.10 mA) was used to deliver a 1% sodium nitroprusside (SNP) solution in order to assess endothelium-independent vasoreactivity. A stable baseline blood flow was measured for 2 min before the current was applied. Measurement of the peak increase in CBF was performed at the peak of the maximal plateau reached after each stimulation to represent the increase in vasodilation. Responses to ACh and SNP were presented as percentages of basal perfusion values measured before the iontophoretic stimulation. All traces were visualised on a personal computer using Perisoft V.5.10 (Perimed Software) and stored for later analysis.

STATISTICAL ANALYSIS

Statistical analyses were performed with the Statistica 10 software programme (Tulsa, Oklahoma, USA). For all data, the Shapiro-Wilk test was first used to test normality. As normality was confirmed for all data, they were presented as mean (SD and SEM). A paired *t*-test was used to compare values obtained for each diver before and after the protocol. Statistical significance was set *a priori* at $P < 0.05$. However, in order to take into multiple comparisons (three for each substance), a Bonferroni correction was applied and, therefore, $P < 0.016$ was considered statistically significant for the comparison of the time of measurement on each substance.

Figure 1

Response of cutaneous blood flow to sodium nitroprusside (SNP) iontophoresis at 30 min and 24 h after an air dive. The increase in cutaneous blood flow is expressed as a percentage of blood flow variation before the dive. Thin lines are individual values; the thick line represents the mean (SEM) value; $P < 0.016$ between before and after diving protocol at 30 min; $n = 6$

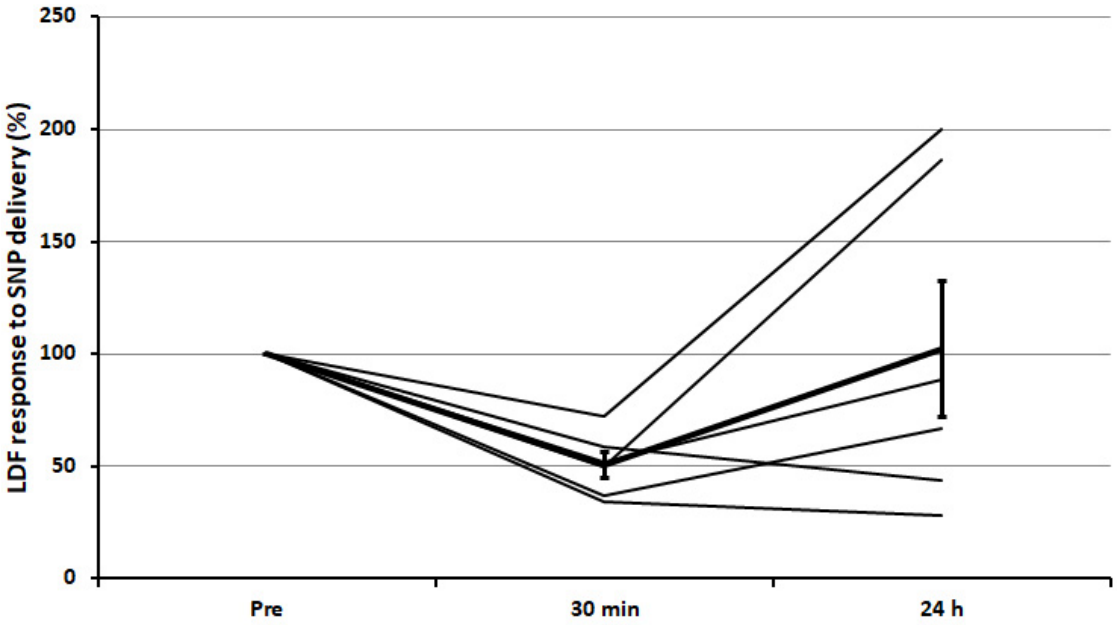
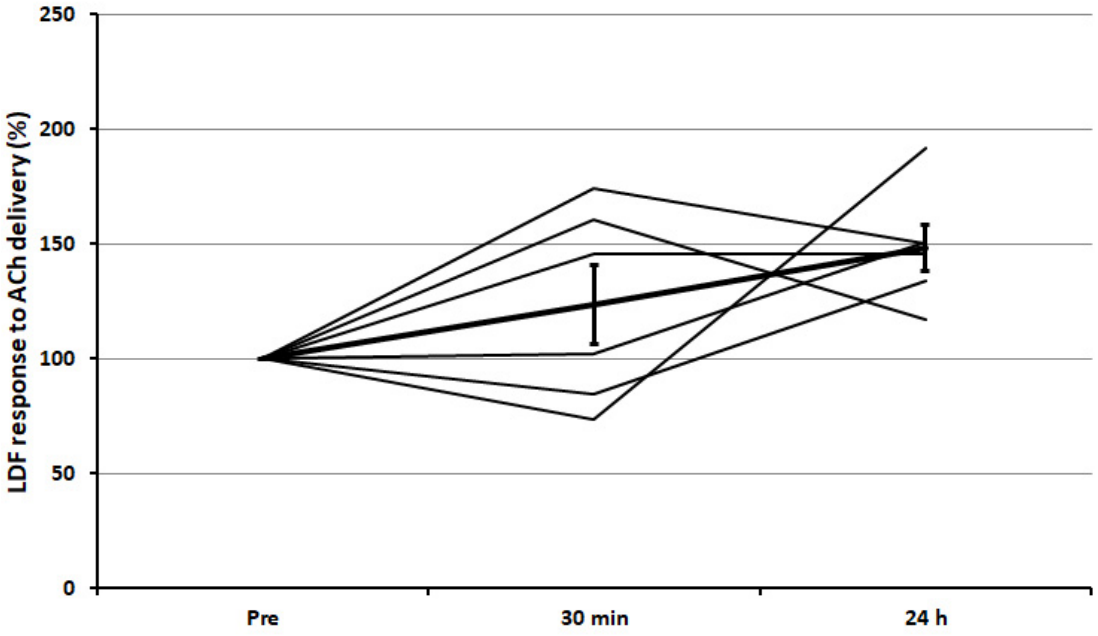


Figure 2

Response of cutaneous blood flow to acetylcholine (ACh) iontophoresis at 30 min and 24 h after an air dive. The increase in cutaneous blood flow is expressed as a percentage of blood flow variation before the dive. Thin lines are individual values; the thick line represents the mean (SEM) value; $P < 0.016$ between before and after diving protocol at 24 h; $n = 6$



Results

The cutaneous microvascular response to SNP at 30 min post dive was significantly decreased compared to pre-dive measurements (50 (SEM 6)% of the pre-dive values, $P = 0.0003$), as shown in Figure 1. At the same time, endothelium-dependent hyperaemia elicited by ACh averaged 123 (17)% of the pre-dive values and was not significantly changed ($P = 0.230$; Figure 2). However, 24 hours after this first dive, response to SNP was no longer

different (102 (29)% of the pre-dive values, $P = 0.113$) while ACh-induced hyperaemia was significantly higher than pre-dive values (148 (10)% of the pre-dive values, $P = 0.005$).

Discussion

The main result of the present study is that the impairment of microvascular smooth muscle post-dive lasts less than 24 h and that the microvascular endothelium-dependent response to ACh is increased 24 h post-dive.

The effects of scuba diving on the vascular wall have previously been assessed mainly at the level of the brachial artery. Globally, data from these studies demonstrate that scuba diving decreases both the FMD and the vasodilation induced by NO donors, indicating that the endothelium and the vascular smooth muscle are both impaired.[1,3,5] The present study assessed vascular function at the level of the cutaneous microcirculation. Only the response of vascular smooth muscle to NO was impaired 30 min after surfacing whereas the response to endothelial stimulation by ACh remained unchanged. This observation agrees with previous studies. Indeed, although they all reported a decreased response to NO donors,[3,5,13] the response to ACh remained unchanged in two studies[5,13] while two others reported decreased response to endothelium stimulation by ACh[3] or shear stress.[11] In the present study, microvascular smooth muscle function was no longer impaired 24 h after the dive, which is shorter than the three days needed for complete recovery of the FMD.[4]

Laser Doppler is based on the reflection of a beam of laser light. The light undergoes changes in wavelength when it hits moving blood cells. The magnitude and frequency distribution of these changes in wavelength are related to the number and velocity of blood cells.[16] LD does not directly measure cutaneous blood flow, but provides an index of skin perfusion, quantified as the product of average red blood cell velocity and their concentration, often referred to as flux. The arbitrary units correspond to the voltage of the analog signal of the LD flowmeter, with the zero-value corresponding to the blood flow value during arterial occlusion. However, it is considered accurate at detecting and quantifying rapid changes in CBF in response to a given stimulus.[14] Additionally, although plasma volume changes have been reported after scuba diving[17] we have shown that this has no effect on the LDF signal, at least in this range.[13]

Besides its action through endothelium, a C-fibre (axon reflex)–mediated mechanism has also been reported during iontophoresis of ACh.[18] However, this mechanism occurs only when the iontophoretic current is equal to or higher than 5.10^{-2} mC·mm^{-2}. Given the surface of the drug-delivery electrodes (113 mm^2), we used a charge density equal to 3.10^{-2} mC·mm^{-2} only. A recent study further confirmed that the change in cutaneous microvascular blood flow in response of iontophoresis of 2% ACh in deionised water with 0.1 mA during 30 s was due to ACh only.[19] Thus, it is unlikely that an axon reflex occurred in our study and the increase in CBF is therefore a response to ACh stimulation. In these conditions, the changes in the cutaneous response post-dive reflect the modifications of microvascular reactivity to ACh.

SNP is known to react with tissue sulfhydryl groups under physiologic conditions to produce NO and thereby directly stimulate VSM relaxation, whereas the mechanisms by which iontophoretic administration of ACh increases CBF in humans are still under debate. Prostanoids, endothelium derived hyperpolarising factors and NO have all been reported to be involved.[15,20,21] Nevertheless, it was confirmed that ACh and SNP increase skin blood flow through endothelium-dependent and endothelium-independent mechanisms, respectively.[21,22] Therefore, the increased response to ACh with unchanged response to SNP we observed 24 h post-dive is typical of enhanced endothelium-dependent vasodilation with normal endothelium-independent vasodilation. Moreover, the transient decrease of the response to SNP in the present study clearly indicates that VSM reactivity is impaired 30 min after the dive and recovers within 24 h. As a consequence, the unchanged response to ACh 30 min after surfacing despite a decreased response to SNP suggests that the impairment of vascular smooth muscle function is compensated by enhanced endothelial function. Taken together, our data suggests that air scuba diving might enhance microvascular endothelial function and that this effect may last up to 24 h.

Many mechanisms can explain these changes and hypotheses based on our results are speculative. Because post-dive impairment of FMD results in part from the diving-induced increase of ROS production[4] and also in part from decompression-induced bubbles,[5,23] it is tempting to hypothesize that the same mechanisms mediate the effect of diving on the cutaneous microcirculation. However, ROS were shown to decrease the response to ACh iontophoresis without altering that to SNP in the human cutaneous microcirculation,[24,25] whereas reducing the amount of circulating bubbles has no effect on the post-dive changes of cutaneous microcirculation.[5] Although we did not measure skin temperature, the immersion must have induced a decrease in the forearm skin temperature, especially in cold water and despite the use of dry suits. Given its role in thermoregulation, this should have resulted in decreased CBF. However, although it was previously shown that a moderate decrease of skin temperature results in progressive decrease of both ACh- and SNP-induced vasodilation,[26] in our study only the response to SNP (but not ACh) was decreased at 30 min post-dive. This suggests that the changes in microvascular reactivity we observed are unlikely to result from modifications of the skin temperature. Similarly, we previously reported that neither hyperbaric hyperoxia nor immersion alter the cutaneous responses to iontophoretic administration of both ACh and SNP.[13] It was also shown that the increased activity of the sympathetic autonomous nervous system, previously reported after diving,[27] has no

effect on the amplitude of the forearm skin response to iontophoretic administration of ACh or SNP.[28] Finally, an increase of plasma NO was reported during scuba diving.[29] Although, the reason and origin of this increased NO production are still unknown, it could explain the decrease in vascular smooth muscle response to the exogenous NO donor SNP through desensitisation of the soluble guanylyl cyclase.[30]

Enhanced microvascular endothelial function after diving contrasts with the previously reported impaired endothelial-dependent relaxation of conduit arteries as assessed by FMD. This further confirms that the macro and microcirculation can be affected differently by scuba diving. This is consistent with previous reports showing that the brachial artery and the cutaneous microcirculation are affected differently by various conditions such as decompression-induced circulating bubbles[5,23] and physical exercise training.[31] The explanations for these territorial differences are still unclear. Differences in vessel calibre-dependent blood rheology and/or signalling pathways may be involved. Indeed, shear stress is lower in the microcirculation than in conductance arteries.[32] Along with that, shear stress increases endothelial-dependent vasodilation through an increased NO-dependent mechanism only in the brachial artery[33] and through endothelial NO-dependent and independent pathways in the cutaneous microcirculation.[34]

LIMITATIONS

The low number of subjects included in the study is certainly its main limitation. Studies including more divers are needed before definitive conclusions can be made. However, differences between the times of measurements were statistically significant. Moreover, at 30 min post-dive, our results agreed with several previous studies from our group and others. As already stated in the discussion, we did not measure skin temperature at the site of measurement, nor did we regulate the temperature of the room where measurements were made. However, although an effect of temperature cannot be ruled out in our study, the room temperature did not vary throughout the experiment, and the previously reported effects of changes in skin temperature are not confluent with the changes observed in our study. In our study, we presented relative amplitudes of the increase of CBF, which gives information about the contribution of each component to the total variability observed. It was reported that wavelet analysis from haemodynamic signals makes possible the determination of different influences on skin blood flow.[21] This represents a challenge in future research in this field.

Conclusions

Microvascular endothelial function may be enhanced up to 24 h after a SCUBA dive. This contrasts with changes which occur at the level of large conduit arteries and indicate that these two vascular beds may react differently to diving. However, the reasons for this difference are still unknown. Additionally, whether the changes observed at the level of microcirculation could influence the divers' response to diving and/or decompression needs to be better understood. The impact of modifications occurring in microvascular bed on the physiological effects of diving is worth further studies.

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Scuba diving fatalities in Australia 2001 to 2013: Chain of events

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Abstract
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Introduction: We aimed to identify the possible chain of events leading to fatal scuba diving incidents in Australia from 2001–2013 to inform appropriate countermeasures.
Methods: The National Coronial Information System was searched to identify scuba diving-related deaths from 2001–2013, inclusive. Coronial findings, witness and police reports, medical histories and autopsies, toxicology and equipment reports were scrutinised. These were analysed for predisposing factors, triggers, disabling agents, disabling injuries and causes of death using a validated template.
Results: There were 126 known scuba diving fatalities and 189 predisposing factors were identified, the major being health conditions (59; 47%), organisational/training/experience/skills issues (46; 37%), planning shortcomings (29; 23%) and equipment inadequacies (24; 19%). The 138 suspected triggers included environmental (68; 54%), exertion (23; 18%) and gas supply problems (15; 12%) among others. The 121 identified disabling agents included medical-related (48; 38%), ascent-related (21; 17%), poor buoyancy control (18; 14%), gas supply (17; 13%), environmental (13; 10%) and equipment (4; 3%). The main disabling injuries were asphyxia (37%), cardiac (25%) and cerebral arterial gas embolism/pulmonary barotrauma (15%).
Conclusions: Chronic medical conditions, predominantly cardiac-related, are a major contributor to diving incidents. Divers with such conditions and/or older divers should undergo thorough fitness-to-dive assessments. Appropriate local knowledge, planning and monitoring are important to minimise the potential for incidents triggered by adverse environmental conditions, most of which involve inexperienced divers. Chain of events analysis should increase understanding of diving incidents and has the potential to reduce morbidity and mortality in divers.

Introduction

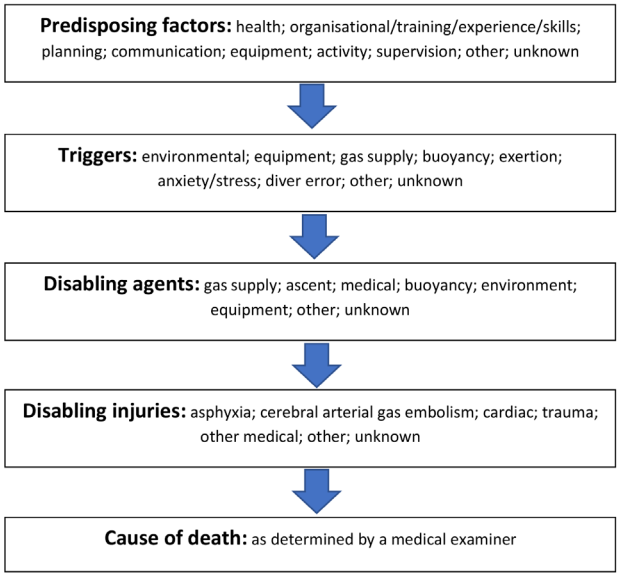
Scuba diving safety can be influenced by a broad range of factors that present before, during and sometimes after the dive. Such factors include: health and fitness; organisation; planning; communication and supervision; equipment problems; decisional factors; and various environmental factors.[1,2] A diving incident usually involves a trigger which may lead to a cascade of related events, some precipitated by the diver and some circumstantial, which may lead to morbidity or mortality. Several studies of diver fatalities have utilised a sequential or 'chain of events' analysis (CEA) to describe the suspected sequence of events within the incident. This began with a landmark report on US fatalities which divided the CEA into four categories.[1] These were defined as the trigger, disabling agent (DA, an action or circumstance following the trigger which caused injury or illness), disabling injury (DI, directly responsible for death or incapacitation leading to drowning), and cause of death (specified by a medical examiner). The methodology described was adapted and subsequently applied to a large series of Australian fatalities.[2]

However, preceding the trigger may be factors which predispose to such an event. Some reports have highlighted the role that human factors may play in diving incidents.[3,4] Such factors, among others, have been included in a revised CEA template as an additional category called 'predisposing factors'.[5] This revised template was validated and subsequently used to analyse this current series of scuba diving fatalities. Such an analysis helps to identify common features in the incidents and can be used to devise appropriate countermeasures.

Methods

This was a case series of scuba diving-related fatalities that occurred in Australian waters from 2001 to 2013, inclusive.

Figure 1
Flowchart of the chain of events analysis of a scuba diving accident



Approval for the study was received from the Human Research Ethics Committees of the Victorian Department of Justice, the Royal Prince Alfred Hospital, the Coroner's Court of Western Australia, the Queensland Office of the State Coroner and Deakin University, Melbourne.

SEARCH AND REVIEW

The methodology for identifying relevant cases is described in detail elsewhere.[6] In brief, it comprised a comprehensive key word search of the National Coronial Information System (NCIS)[7] to identify scuba diving-related deaths reported to various state coronial services for the years 2001 to 2013, inclusive. Cases identified were matched with those collected by the Divers Alert Network Asia-Pacific (DAN AP) via the media or the diving community in order to minimise the risk of over- or under-reporting. Relevant coronial findings, witness statements, police, autopsy and equipment reports and medical histories were reviewed, and annual case series were prepared and published.[8–19]

CHAIN OF EVENTS ANALYSIS

This chain of events analysis for the scuba fatalities was based on the criteria and templates previously published.[5] Event categories and sub-categories are shown in Figure 1, and categories are defined as follows:

Predisposing factor: A relevant factor(s) present prior to the dive, and/or prior to the trigger occurring, and which was believed to have predisposed to the incident and/or to key components in the accident chain (e.g., the trigger or disabling agent).

Trigger: The earliest identifiable event that appeared to transform an unremarkable dive into an emergency.

Disabling agent: An action or circumstance (associated with the Trigger) that caused injury or illness. It may be an action of the diver or other persons, function of the equipment, effect of a medical condition or a force of nature.

Disabling injury: Injury or condition directly responsible for death or incapacitation followed by death from drowning.

Cause of death: As specified by a medical examiner, which could be the same as the disabling injury or could be drowning secondary to injury.

After planning, one author (JL) applied the published templates to the data and obtained the reported results. Both authors were involved in writing the report. A hypothetical example of the application of such a template is: a diver with a faulty tank pressure gauge (predisposing factor) runs out of air (trigger), makes an emergency ascent (disabling agent), suffers a cerebral arterial gas embolism (disabling injury), becomes unconscious and subsequently drowns (cause of death).

STATISTICAL ANALYSIS

Descriptive analyses based on means (standard deviation) or medians (range) as appropriate were conducted using SPSS Version 25 (IBM, Armonk, New York). Comparisons of proportions employed odds ratios (OR) accompanied by 95% confidence intervals (95% CI). The level of significance was considered as $P \leq 0.05$.

Results

There were 126 scuba diving fatalities during the study period. The mean (SD) age was 44 (13) years and 99 (79%) of the victims were male. Autopsy reports were available to the researchers for 123 (98%) of the cases.

PREDISPOSING FACTORS

One hundred and eighty-nine predisposing factors were identified as possible or likely contributors to the 126 deaths (Table 1). No predisposing factors were identified in seven deaths. The main factors were related to the victims' health and/or organisational/training/experience/skills-related factors prior to diving.

Pre-existing medical conditions: Forty-six divers (37%) were identified as having chronic medical conditions which likely contributed to their incident. These were predominantly cardiac conditions such as ischaemic heart disease (IHD), but also included respiratory conditions, hypertension, diabetes and epilepsy, among others. They are the subject of a separate report.[20]

Organisational/Training/Experience/Skills: Inexperience and the associated relatively poor diving skills were implicated in 30 of the 46 deaths in this category. However,

Table 1

Predisposing factors (n = 189) associated with 126 scuba fatalities; some deaths involved multiple predisposing factors, hence the number of predisposing factors exceeds the number of cases; * = absence of items such as wetsuit, knife, fins, snorkel when needed

Predisposing factor	Subgroup	*n*	Mean (SD) age	Male/Female
Health		59 (47%)	50 (12)	47/12
	Significant medical history	46		
	Fatigue	7		
	Drug / medication intake	4		
	Obesity	2		
Organisational/training/ experience/skills		46 (37%)	39 (13)	32/14
	Inexperienced overall	30		
	Poor organisation	6		
	Lack of training / skills for dive	5		
	Lack of recent experience	5		
Planning Poor pre-dive choice of:		29 (23%)	40 (12)	24/5
	Conditions	15		
	Solo diving in poor conditions	6		
	Location	4		
	Other	4		
Absence of appropriate equipment or use of faulty equipment		24 (19%)	41 (12)	19/5
	Faults	9		
	Absence*	9		
	Over-weighting	4		
	Other	2		
Activity		14 (11%)	41 (12)	13/1
	Penetration	6		
	Deep diving with CCR	3		
	Seafood collection	3		
	Other	2		
Supervision Poor supervision by:		14 (11%)	37 (12)	7/7
	Buddy	8		
	Instructor	5		
	Divemaster / guide	1		
Communication Poor communication or co-ordination between:		3 (2%)	37 (8)	2/1
	Instructor and student	1		
	Diver and others	1		
	Dive shop and instructor	1		

the victims included five experienced divers who had not dived for extended periods. Among five divers with little relevant experience were three very experienced divers who died due to a lack of familiarity with new equipment which included a drysuit, technical diving equipment and a rebreather. The six cases associated with organisational issues included a poor internal process by a dive shop for the oversight of the progress and needs of trainees, poor systems for the organising of introductory scuba dives, unqualified staff giving inappropriate medical advice and an inadequate system for ensuring the appropriate screening and oversight of inexperienced divers.

Planning: These factors involved poor planning decisions, generally immediately prior to the dive. The majority involved a decision to dive in obviously unsuitable conditions which included rough water and/or surge, very poor visibility and/or strong currents. Six divers set out to dive solo in conditions that were obviously unsuitable, especially when alone, and another three intentionally separated in such circumstances. One diver failed to correctly plan his decompression requirements, and another his breathing gas requirements while diving in a cave. Other issues involved poor choice of instructor/student ratios and poor gas supply planning.

Absence of appropriate equipment or use of faulty equipment: Predisposing factors related to equipment were contributory to 24 incidents, some with multiple issues. Relevant faults were found in a variety of equipment which included the buoyancy compensation device (BCD), pressure gauge, regulator, alternative air source, oxygen sensors and tank valve. Incorrect gear configuration or assembly at the site contributed to four deaths, poor-fitting wetsuits to two

Table 2
Triggers (n = 139) associated with 126 scuba fatalities; some deaths involved multiple triggers; * = Various faults in regulators, tank valves, oxygen sensors, wet and dry suits

Trigger	Subgroup	*n*	Mean (SD) age	Male/Female
Environment		68 (54%)	45 (13)	52/16
	Conditions	37		
	Immersion effects	19		
	Entrapment	5		
	Marine animal	3		
	Other	4		
Exertion		23 (18%)	52 (9)	21/2
	Pre-dive	4		
	During dive	13		
	Post-dive	6		
Gas supply		15 (12%)	42 (9)	13/2
	Out of gas	8		
	Low gas	3		
	Incorrect mix	2		
	Contamination	1		
	Other	1		
Equipment*	N/A	10 (8%)	32 (12)	9/1
Anxiety	N/A	10 (8%)	39 (15)	6/4
Primary diver error	N/A	4 (3%)	37 (7)	2/2
Buoyancy	N/A	3 (2%)	51 (10)	1/2
Other	N/A	6 (5%)	43 (15)	5/1
Unknown	N/A	14 (11%)		

and weights in BCD pockets and unable to be ditched was contributory to one death. Substantial oil in cylinder air likely contributed to another.

Activity: Fourteen victims were undertaking activities that can potentially carry an increased risk of an incident. These included six penetration dives, three in freshwater caves and two in deep wrecks. In four of these, the victims became separated and ran out of breathing gas. Three incidents occurred during deep dives using a closed circuit rebreather (CCR). Another three incidents involved attacks by large sharks whilst the victims were either harvesting seafood, spearfishing or diving near where fishing was being conducted.

Poor supervision: In 13 of the 14 such incidents, poor decisions by the supervisor were made prior to the dive. Five of these involved a formal instructional situation and four involved divers who had very little or no experience. Another involved a diver (a non-instructor) teaching his girlfriend to dive. Most other incidents involved more experienced divers making poor pre-dive decisions, often about sites or conditions, which affected their inexperienced buddies.

Poor communication or co-ordination: Three incidents specifically involved poor pre-dive communication between divers and/or those overseeing them, although several cases in the preceding categories were also associated with communication or co-ordination issues.

TRIGGERS

One-hundred and thirty-nine possible or likely triggers were identified (Table 2).

Environmental: The main triggers were environment-related and were implicated in more than half of the fatalities. These were predominantly associated with adverse conditions which included current, rough seas, poor visibility, surge and depth. Twenty-three of the 37 divers with a conditions-related trigger were inexperienced, compared with 34 of 78 divers with other triggers. This indicates a higher association of conditions-related triggers among 'inexperienced' divers than with 'experienced' divers (OR 2.22, 95% CI 1.00–4.94; P = 0.05).

Nineteen fatalities were believed to have been associated with the cardiac-related effects of immersion; seven of these due to immersion *per se*, and twelve with the combination of immersion and exertion due to conditions. Five incidents involved entrapment due to environmental circumstances. Three deaths were associated with the presence of and subsequent attack by a shark.

Exertion: Exertion-related triggers were associated with exertion before, during or after a dive. This exertion was unrelated to adverse sea conditions and was what would normally be expected with swimming or walking whilst wearing scuba equipment. Seventeen of these 23

incidents resulted in a cardiac-related DI. Some causes included exertion before the dive (e.g., walking to the site wearing diving equipment), exertion during a dive (e.g., carrying heavy catch bags) and exertion post dive (e.g., long surface swims or boarding a boat).

Gas supply: Eleven of these 15 incidents involved low or exhaustion of breathing gas situations which occurred while hunting seafood, salvaging an anchor from a wreck at depth and during a cave penetration dive. Three incidents resulted from inappropriate breathing gas: one from contamination by oil; and two from incorrect breathing mixtures. One novice entered the water without his regulator in his mouth.

Equipment: The 10 equipment-related triggers included: one incident each of a faulty mouthpiece causing aspiration; faulty tank 'J-valve' causing loss of reserve air; tight wetsuit causing breathing restriction and subsequent panic; tank slippage causing loss of air supply; faulty mask causing leak and panic; alternate air source detachment causing loss of mask and panic; loss of fin causing mobility problems and panic; faulty oxygen sensors causing hyperoxia; faulty drysuit inflator causing problems at depth and during ascent; and faulty BCD inflator causing rapid ascent.

Anxiety: Anxiety is a likely trigger in a substantial number of diving-related deaths. However, it was only included as a factor in the CEA when there were specific witness accounts reporting that the victim displayed signs of anxiety, and this appeared to have led to panic and the subsequent fatality. There were 10 such accounts, all but one involving novice divers.

Buoyancy: Only three of the incidents appeared to have been triggered by a buoyancy-related problem. One involved a diver who had logged 55 dives but still had not mastered buoyancy control and who sank after venting too much air from her BCD during ascent. The other two involved experienced divers who were using relatively unfamiliar equipment; one a CCR and the other a drysuit.

Primary diver error: Many incidents involved diver error in the accident chain, some prior to the dive and others arising from poor decisions or arising subsequent to a problem. Four incidents are likely to have been triggered by primary diver error, in conjunction with other triggers. In one case, the diver failed to heed a repeated warning on his CCR. In another, a substantially over-weighted drysuit diver, relatively inexperienced in the use of her drysuit (although highly experienced otherwise), redescended alone with relatively little remaining air and inadvertently inverted while adjusting buoyancy. In the third incident, an experienced cave diver inadequately accounted for her ability to relocate and reach an alternative, and necessary, air supply on the other side of a narrow constriction. The final incident involved the diver entering the water without his regulator in place.

Other triggers: included three medical-related conditions, trauma, loss of dentures and inadequate decompression.

DISABLING AGENTS

There were 121 likely disabling agents identified in the 126 scuba fatalities (Table 3).

Medical: More than three-quarters of the medical-related disabling agents were likely due to cardiac conditions, predominantly ischaemic heart disease. The 'Other' category included two subdural haematomas, and one each from asthma and a pulmonary cyst.

Ascent: In at least 21 incidents, the disabling agents were clearly ascent-related, borne out by evidence of pulmonary barotrauma (PBT) or cerebral arterial gas embolism (CAGE). The actual ascent was unwitnessed in eight cases, including three where the diver had run out of breathing gas. There were other incidents in which the disabling agent may have been the ascent although this was unclear and, therefore, not included. Nine incidents were characterised by a witnessed rapid ascent, three of these involving exhaustion of the breathing gas. The incidents in which gas trapping occurred during ascent were likely associated with a pre-existing medical condition including asthma, chronic obstructive pulmonary disease and pleural effusion. These ascents were witnessed, and none were described as rapid.

Buoyancy: Eight victims became incapacitated by insufficient buoyancy on the surface as a direct consequence of failing to inflate their BCD and/or dump weights. Seven others were disabled while at depth and subsequently drowned due to poor buoyancy control. Two of these were drysuit divers who became inverted while trying to adjust buoyancy.

Gas supply: The identified events where the disabling agent was related to the lack of, or inappropriate supply of, breathing gas involved: direct exhaustion of breathing gas in eight; out of gas post-entrapment in three; loss of access to demand valve in four; and beginning the dive with inappropriate breathing gas in two (one involving suicide using pure helium and the other a hypoxic mix breathed at the surface).

Environmental: Seven deaths involved adverse sea conditions with six of the victims being disabled after heavy contact with rocks, and one drowning after being swept off the rocks. Three were disabled by shark attacks and another three were trapped and subsequently ran out of breathing gas as a direct result of entrapment.

Equipment: These incidents included one each of: incorrect fitting of an alternative air supply leading to detachment during the dive; equipment weight and bulk causing incapacitation in rough surface conditions; a ditched weight belt becoming entangled with the tank pressure gauge and

Table 3
Disabling agents (*n* = 121) associated with 126 scuba fatalities; * = Unwitnessed but evidence of an ascent complication

Disabling agent	Subgroup	*n*	Mean (SD) age	Male/Female
Medical		48 (38%)	50 (12)	42/6
	Cardiac disease/dysfunction	38		
	Oxygen seizure	3		
	Immersion pulmonary oedema	≥ 3		
	Other	4		
Ascent		21 (17%)	39 (13)	18/3
	Rapid ascent	9		
	Gas trapping	3		
	Inadequate decompression	1		
	Unwitnessed*	8		
Buoyancy		18 (14%)	42 (11)	9/9
	Negative at surface	8		
	Poor control underwater	7		
	Grossly overweighted	3		
Gas supply		17 (13%)	44 (13)	13/4
	Out of gas	11		
	Loss of regulator access	4		
	Inappropriate mix	2		
Environmental		13 (10%)	38 (11)	12/1
	Conditions	7		
	Shark attacks	3		
	Entrapment	3		
Equipment		4 (3%)	38 (12)	4/0
Unknown		11 (9%)		

the loss of a fin and mask subsequent to impact with a boat hull.

DISABLING INJURIES

The predominant disabling injuries identified were asphyxia, cardiac causes and CAGE with or without evidence of PBT (Table 4). Others were immersion pulmonary oedema (IPE), trauma, and decompression sickness (DCS). In 21 cases, no clear disabling injury could be identified. In nine of these, there were indicators of a possible cardiac-related incident, although other factors, such as signs of drowning or CAGE, hampered a clear determination.

The numbers of victims in each of the Australian states identified with a cardiac-related disabling injury were: Queensland (10/29); Western Australia (5/19); South Australia (5/17); New South Wales (7/32); Victoria (4/20) and Tasmania (1/9). Among victims aged 45 years or more, 26 of 66 had a cardiac-related disabling injury, compared with six of 60 victims younger than 45 years. This indicates a strong association between being at least 45 years old and having a cardiac-related disabling injury (OR 5.85, 95% CI 2.20–15.55; $P = 0.0004$). Twenty-five of the 32 deaths attributed to a cardiac disabling injury were associated with exertion, compared with 18 of 94 non-cardiac deaths (78% vs. 19%). This indicates a strong association between a disabling cardiac injury and preceding exertion (OR 6.31, 95% CI 2.52–15.80; $P = 0.001$).

Rough conditions were a trigger in 15 of 47 deaths attributed to asphyxia as the DI, and 6 of 79 of the deaths attributed to other disabling injuries (32% vs. 8%). This indicates a strong association between the trigger of rough conditions and asphyxia as the disabling injury (OR 5.70, 95% CI 2.03–16.04; $P = 0.001$). There were no other significant associations.

CAUSES OF DEATH

The predominant causes of death identified were drowning, which was reported in 64 (51%) of the incidents, cardiac causes (23, 18%) and PBT/CAGE (14, 11%). Others included trauma (three, 2%), IPE (two, 2%) and DCS (two, 2%). In 18 (14%) cases, no clear cause of death was identified by the pathologists.

Figure 2 compares the likely disabling injuries as identified by the COE analysis with the causes of death reported by the pathologists. Drowning has traditionally been (and still is in many places) recorded as the default cause of death when a lifeless diver was recovered from the water and no other obvious cause of death was apparent on autopsy. The difference between the 51% drowning as the cause of death

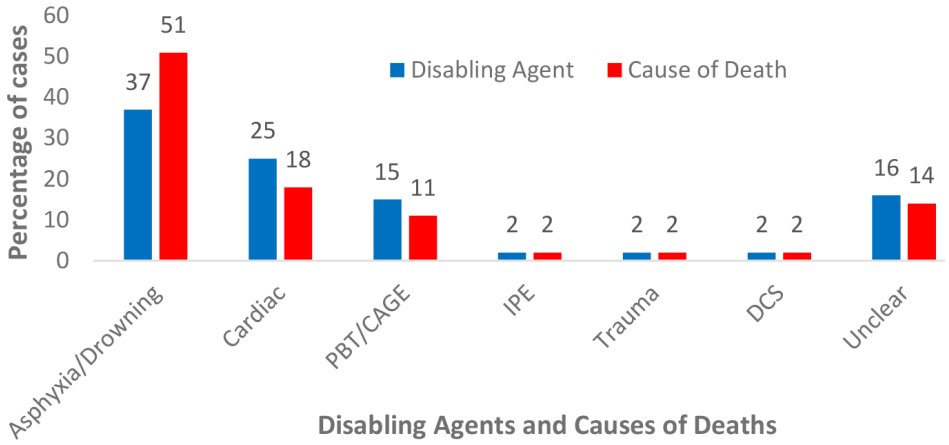
Table 4

Relative occurrence of disabling injuries in 126 scuba fatalities. Ages are in years. Data are mean (SD) unless otherwise specified. N/A – not applicable; PBT/CAGE – pulmonary barotrauma/cerebral arterial gas embolism; IPE – immersion pulmonary oedema; DCS – decompression sickness

Disabling injury	*n* (%)	Male/Female	Age (all)	Age (males)	Age (females)
Asphyxia	47 (37)	33/14	42 (12)	42 (10)	40 (15)
Cardiac	32 (25)	31/1	52(11)	52 (11)	46 (0)
CAGE/PBT	19 (15)	16/3	40 (14)	42 (14)	28 (12)
IPE	3 (2)	0/3	50 (1)	N/A	50 (1)
Trauma	3 (2)	3/0	29 (6)	29 (6)	N/A
DCS	1 (1)	1/0	45 (0)	45 (0)	N/A
Unclear	21 (17)	16/5	47 (12)	49 (12)	41 (11)

Figure 2

Comparison of disabling injuries and causes of death in 126 scuba fatalities; PBT/CAGE – pulmonary barotrauma/cerebral arterial gas embolism; IPE – immersion pulmonary oedema; DCS – decompression sickness



and 37% asphyxia as disabling injury may reflect cases where drowning was secondary to a cardiac arrhythmia or an injury such as CAGE.

Discussion

Various factors played an important part throughout many of these fatal events. These included pre-existing health conditions, poor fitness, lack of experience, organisational shortfalls, poor planning or supervision and inadequate equipment maintenance. In addition, inattention, carelessness, inappropriate attitude, poor decision-making and inappropriate actions, whether prior to or during an incident, can all influence the outcome. The results of this CEA highlight the value of the addition of the link for 'predisposing factors' which identified almost 200 factors that were present prior to the dives and which likely, or possibly, contributed to these fatalities. Almost one half of the predisposing factors identified were health-related and these are discussed in a separate report.[20] More than one third were associated with organisational factors, training, experience and skills; whilst one quarter were planning-related. Many diving incidents involve more than one factor within and between the various categories in the CEA template, as clearly demonstrated here.

ORGANISATIONAL/TRAINING/EXPERIENCE/SKILLS FACTORS

An important organisational-level consideration is the need for improved education of the diving and medical communities in fitness-to-dive considerations, and how certain health conditions can impact diving safety. Another organisational issue that was apparent in several incidents was the need for dive operations to have clear guidelines on staff responsibilities as well as hand-over to ensure appropriate communication between staff about a customer in their care. The fact that almost three quarters of the victims in this series were found wearing their weight belts[6] suggests that more needs to be done during and after basic scuba

training to highlight the need to attain positive buoyancy in an emergency and to consolidate the required skills.

However, the predominant factor in this category was inexperience, which is discussed in detail in a previous paper.[6] Although more than 90% of the victims were certified or undergoing training at the time of their fatal incident, approximately one half had done fewer than 30 lifetime dives and were, therefore, relative novices. As such, it is unsurprising that lack of skills and inexperience contributed to many deaths. In addition, lack of recent diving is likely to affect current competency and appears to have been contributory to some accidents in both experienced divers and novices.

The template on which this CEA was based[5] is dynamic and should to be adjusted when necessary to improve its utility. In future studies, rather than utilising the combined category of organisational/training/experience/skills as defined in the template, it may be simpler and more effective to separate this into two separate categories; 'organisational' and 'individual diving capacity' with the latter incorporating an individual's training status, experience and skill level.

PLANNING-RELATED FACTORS

One quarter of the incidents involved poor planning decisions such as diving in patently adverse conditions, and/or diving solo or with an obviously ineffective buddy system. The latter is discussed in more detail in a previous paper.[6] Poor planning was a factor in at least four of the seventeen deaths which occurred during diver training. Dive planning should always allow for adverse events ('Murphy's Law'); a history of trouble-free practice of a procedure is no guarantee of lack of future problems. Past poor practice where there have been no obvious repercussions can lead to 'normalisation of deviance' whereby it becomes acceptable not to follow best practice and so narrows the margin of safety.[4]

ENVIRONMENTAL FACTORS

Various environmental factors appear to have been the triggers in almost one half of the incidents. The majority of these were associated with adverse sea conditions including, singularly or in combination, strong currents, poor visibility, rough surface conditions and underwater surge. Inexperienced divers, especially those with poor aquatic skills and lack of comfort in the aquatic environment, can more easily underestimate and be overcome by what might be mild to moderate sea conditions for those with more experience and skill. Even diving veterans can become over-confident in what they believe to be manageable conditions. In this series, inexperienced divers (defined in this study as having done fewer than 30 dives) were twice as likely to have had a conditions-related trigger than those who had done more than 30 previous dives. Sea conditions are dynamic, and appropriate local knowledge, planning and monitoring are important to minimise the potential for such problems.

EXERTION

Exertion was the trigger, or a co-trigger, in over a quarter of these fatal incidents. Dependent on the wearing of weighty equipment, diving inherently involves some level of exertion which can be multiplied manyfold by the presence of a current and rough conditions. Given the potential cardiac demands associated with diving, it is unsurprising that cardiac-related disabling injuries were six times more likely to be associated with preceding exertion than were other disabling injuries.

EQUIPMENT-RELATED PROBLEMS

Although post-incident equipment examination, when conducted, revealed faults in the equipment of one third of the victims, these appear to have been directly contributory as a trigger in a relatively small percentage (8%) of the deaths.[6] This is lower than the 18% previously reported for Australia (1972–2005),[2] 15% for the USA (1992–2003),[1] and 20% in the UK (1998–2009).[21] The apparent reduction in equipment triggers may be due in part to improvements in the design and quality of equipment or in its maintenance and appropriate use by divers over time, as well as regional differences in equipment use. A recent report from a survey of European divers indicated an overall incidence of equipment malfunction in 2.7% of 39,099 dives.[22] However, in the UK fatality series,[21] over half of the equipment-related deaths involved the use of rebreathers which are more commonly used in the UK than in Australia and many other countries. The likely over-representation of rebreathers in 'near-misses' reported by some Australian divers[23] is testament to the greater complexity of these devices, and the increased need for training, maintenance and vigilance.[24–26] Many fatal incidents involving rebreather divers appear to result from human error. However, a considerable number have also been attributed to design faults in the devices, something that is being progressively identified and addressed.[24,27,28]

All the equipment-related deaths in this series were preventable. For example, one inexperienced diver failed to secure his alternative-air second stage during assembly, such that it came detached during a cave dive. If the dive operator had secured the fitting, the death would likely not have occurred. Other problems arose from lack of maintenance. BCD inflator/deflator mechanisms have historically been a common cause of mishaps,[29] and continue to be so. Faulty submersible tank pressure gauges should have been identified well before the planned dive and repaired or replaced.

The use of pre-dive checklists should be invaluable in the prevention of a variety of diving mishaps, including those related to equipment, and should be strongly encouraged throughout the diving community, but especially among those using more complicated equipment, such as rebreathers.[30–32]

GAS SUPPLY PROBLEMS

Gas supply-related issues comprised 12% of triggers and 13% of the disabling agents in this series, usually resulting in CAGE or asphyxia. Despite the ubiquity and relatively low cost of good quality submersible tank pressure gauges, breathing gas depletion remains a problem.[6] This is usually a result of inexperience, inattention, poor planning or faulty equipment. All of these are preventable with appropriate equipment maintenance, careful gas supply planning and greater situational awareness prior to, and during, the diving. Technical divers need to be very careful about their choice of breathing gases to avoid using an inappropriate mixture during any part of a dive. Several deaths in this series were associated with the use of unsuitable breathing mixtures, either inadvertently or through poor planning.

Although contamination of breathing gas was not definitively identified as the direct cause of any death in a scuba diver in Australia between 2001 and 2013, it has been subsequently.[33] However, oil contamination was a likely contributor to one death in this series, in which the diver appeared to have become nauseated, made a rapid ascent and suffered a CAGE. Deficiencies in the required purity of the breathing air were found in over 8% of cases, so this is an area requiring vigilance. Correct compressor placement, maintenance and appropriate oversight by knowledgeable personnel are important; the addition of carbon monoxide monitoring is highly desirable.

LIMITATIONS

As with any uncontrolled case series, the collection and analysis of fatality data are subject to inevitable limitations and uncertainties associated with the incident investigations. Given that many incidents go unwitnessed, assertions in the reports are sometimes speculative. Important information may not have been available in some cases, which rendered CEA data incomplete, thus limiting the conclusions that could be drawn. Even with the use of a template, classification of cases into a sequence of five events in the CEA is imperfect and remains vulnerable to some subjectivity. The chain of successive events is a simplified representation of incidents that may be the result of parallel events and more factors than fit into the five categories used. Therefore, misclassification of factors into such categories is possible. However, this should not prevent identification of modifiable factors in what were, ultimately fatal events.

Conclusions

Chronic medical conditions, predominantly cardiac-related, are a major contributor to diving deaths. It is important that divers with such conditions, indeed all 'older' divers, undergo fitness-to-dive assessments, preferably with doctors with dive medical training. Other common predisposing factors involved organisational shortcomings, inadequate training, experience and skills, and poor planning and supervision. Appropriate local knowledge and monitoring are important to minimise the potential for the many incidents triggered by adverse environmental conditions, most of which involve inexperienced divers. An increased understanding of the impact of the many contributing factors by using chain of events analysis will enhance education about diving fatalities throughout the medical and diving communities. This has a considerable potential to reduce morbidity and mortality in divers.

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Evaluation of pressure in water-filled endotracheal tube cuffs in intubated patients undergoing hyperbaric oxygen treatment

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Key words
Intensive care medicine; Mechanical ventilation; Cuff pressure; Patient monitoring; Ventilators

Abstract
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Introduction: Inflating endotracheal tube cuffs using water instead of air before hyperbaric oxygen treatment (HBOT) is common. The objective of this study was to assess cuff pressure (P_{cuff}), when the cuff was inflated using water, in normobaric conditions and during HBOT.
Methods: This was a prospective, observational study taking place in hyperbaric centre and intensive care unit of the University Hospital of Lille. Every patient who required tracheal intubation and HBOT at 253.3 kPa (2.5 atmospheres absolute [atm abs]) was included. P_{cuff} was measured using a pressure transductor connected to the cuff inflating port. Measurements were performed at 'normobaria' (1 atm abs) and during HBOT at 2.5 atm abs.
Results: Thirty patients were included between February and April 2016. Recordings were analysable in 27 patients. Mean P_{cuff} at normobaria was 60.8 (SD 42) cmH_2O. Nineteen (70%) of patients had an excessive P_{cuff} (higher than 30 cmH_2O). Coefficient of variation was 69%. Mean P_{cuff} at 2.5 atm abs was 51.6 (40.7) cmH_2O, significantly lower than at normobaria ($P < 0.0001$). Coefficient of variation was 79%. In only five (18%) patients was $P_{cuff} < 20$ cmH_2O at 2.5 atm abs.
Conclusions: In normobaric conditions, when the cuff was inflated using water and not specifically controlled P_{cuff} was not predictable. The cuff was typically over-inflated exceeding safe pressure. During HBOT P_{cuff} decreased slightly.

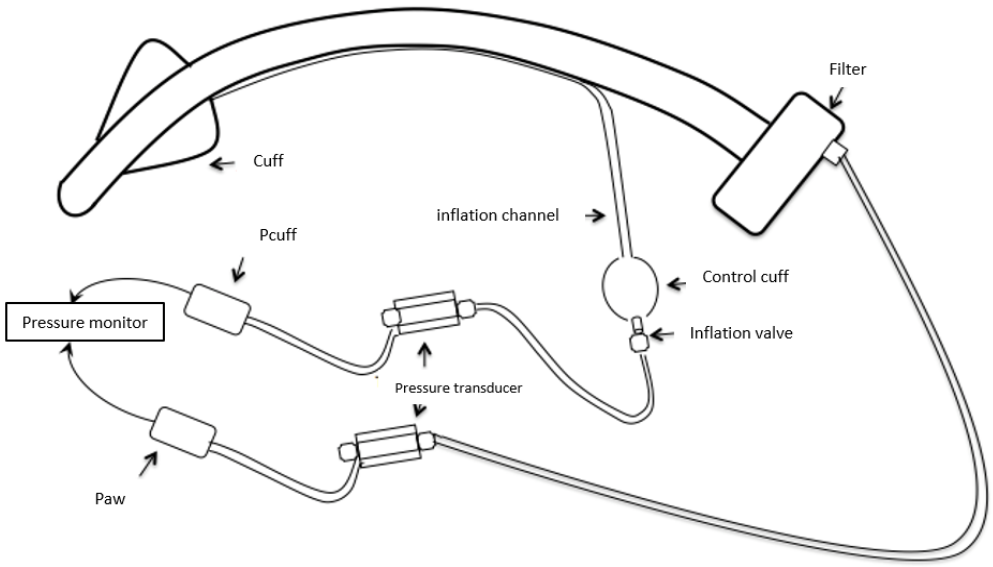
Introduction

Endotracheal tube cuff pressure (P_{cuff}) monitoring is recommended for ventilated patients.[1] Target levels for P_{cuff} should be between 20 and 30 cmH_2O, to avoid both under-pressure, causing micro-inhalations and ventilator associated pneumonia (VAP), and overpressure, which is a risk factor for tracheal mucosa ischaemia and tracheal stenosis.[2–3] In prehospital clinical practice, P_{cuff} is not routinely measured. In a series of 107 patients with air inflation of the cuff, a systematic analysis of P_{cuff} showed over-inflation in 79% of cases. P_{cuff} was 56 (SD 34) cmH_2O when intubation was performed outside the hospital and 69 (37) cmH_2O when intubation was performed within the hospital.[4] In another study in the operating theatre, when not controlled, P_{cuff} was measured at 58 (31) cmH_2O.[5] Without manometric control, cuff air inflating pressure is unpredictable and varies from one patient to another, however, over-inflation is usually observed.

During hyperbaric oxygen treatment (HBOT), patients are subjected to ambient pressure variations. If cuffs remain inflated with air, which is compressible, cuff volume will decrease during compression – following the Boyle -Mariotte Law – leading to leakage during positive pressure ventilation. During decompression, an increase in cuff volume can cause cuff rupture. Water is non-compressible, and replacing cuff air with water before HBOT sessions is the accepted and usual technique.[6] Ventilatory leakage is inversely correlated with P_{cuff}.[7] In current practice, the quantity of water injected into the cuff is whatever volume is required to prevent ventilatory leakage.

This technique is empirical. To our knowledge, P_{cuff} of water-inflated endotracheal tubes has never been evaluated, and P_{cuff} is not monitored in HBOT conditions. Initial pressure levels in normobaric and hyperbaric conditions are unknown. Moreover, the completeness of cuff air removal under these conditions is unknown. Air bubbles remaining in the inflation system could cause unexpected pressure variations. The aim of this observational study was to determine P_{cuff} in normobaric and hyperbaric conditions, when endotracheal tube cuffs are inflated with water within a standard care protocol. As water-filled cuffs have long been used in our unit and in other hyperbaric treatment centres with no adverse effects, we were expecting to find equivalent pressures in cuffs, whether water or air-inflated.

Figure 1
Schematic of the cuff pressure (P_{cuff}) and airway pressure (P_{aw}) monitoring system



Methods

This prospective observational pilot study was performed in the intensive care unit (ICU) and the hyperbaric centre of the Lille University Hospital in collaboration with the Clinical Investigation Centre – Technological Innovation of Lille (INSERM CIC-IT 807). Our study was considered by the Ethics Commission of the French Language Resuscitation Society (SRLF) as a low-risk, usual-care study for which waiver for consent was granted (CE SRLF 13-31). No changes in patient management were caused by our study since it was descriptive by design. Patients (or their families) were nevertheless informed orally and in writing. Although their consent was not required, they were free to refuse to be included at any time. The data were collected and processed anonymously.

The primary objective of this study was to evaluate the median P_{cuff} at 101.3 kPa ('normobaria', 1.0 atmosphere absolute [atm abs]) in water-inflated cuffs as compared to that in air-inflated cuffs. Secondary objectives were the assessment of median P_{cuff} at 253.3 kPa (2.5 atm abs) in both conditions, and any P_{cuff} variations during HBOT sessions.

During a three-month period, all intubated and ventilated adult patients receiving HBOT at 2.5 atm abs during working hours were included, whether intubated within the ICU or before admission.

The HBOT protocol used involved a 15-min pressure rise from normobaria to 2.5 atm abs, maintaining this pressure over 90 min, followed by a 15-min decompression period back to normobaria. As water is non-compressible, replacing air with water in endotracheal tube cuffs before HBOT sessions is the accepted and usual technique in our unit. The usual practice before compression is to aspirate the air and replace it with sterile water whilst maintaining the endotracheal tube in situ. The amount of water injected into the cuff is whatever volume is required to prevent ventilatory leakage.

P_{cuff} and airway pressure (P_{aw}) were monitored continuously during HBOT sessions beginning at normobaria, 15 min prior to compression (initial P_{cuff}), 15 min after the session was over (final P_{cuff}) and during treatment (120 min). P_{cuff} was measured in the way we measure invasive arterial pressure. The cuff was connected to an arterial pressure transducer (Edwards Lifesciences) connected to the Physiotrace® (Physiotrace 1.0, Estaris Monitoring, Lille, France) data acquisition board (Figure 1).[8] Pressure transducers and tubing were purged with water, then connected to the cuff using the inflation valve. Airway pressure (P_{aw}) was monitored continuously via a pressure transducer (Edwards Lifesciences) connecting the breathing circuit filter to the Physiotrace® acquisition station (Figure 1). Physiotrace® includes a blood pressure measurement module which enables calibration (performed before each measurement), acquisition and processing of the blood pressure transducer data. To meet HBOT safety requirements and reduce fire risks as much as possible, the acquisition station was placed outside the hyperbaric chamber and connected to the pressure transducers via conventional electrical wiring through sealed bushings. Signals were post-analysed by an expert in signal processing. Low quality signals or with artifacts were excluded from the study.

To answer a question raised by our initial results, we performed further experimental tests (cylindrical cuffed endotracheal tubes) at 1.0 and 2.5 atm abs under two conditions: 1) usual practice: aspirating the cuff air out then

Table 1
General demographic and clinical data. Data are *n* (%) or median [IQR]. ACV – assist-control ventilation mode; CO – carbon monoxide; HBOT – hyperbaric oxygen treatment; IP – inspiratory pressure; MV – mechanical ventilation; NSTI – necrotizing soft tissue infection; PEEP – positive end-expiratory pressure; PSV = pressure support ventilation mode; RR – respiratory rate; SAPS-2 – simplified acute physiology score; VAP – ventilator-associated pneumonia

Patients	27
Age (years)	48 [35–67]
SAPS-2	59 [39–64]
Weight (kg)	79 [67.5–88]
Male	22 (81)
HBOT indication	
Cervical NSTI	8 (30)
NSTI, other locations	11 (41)
Anoxic encephalopathy	6 (22)
Air embolism	1 (4)
CO intoxication	1 (4)
Delay between ICU admission and inclusion (days)	1 [1–4]
Intubation	
Orotracheal	24 (89)
Nasotracheal	3 (11)
Endotracheal tube size (mm)	7.5 [7–7.5]
Ventilator	
Siarétron®	24 (89)
Maquet®	3 (11)
ACV	25 (93)
Tidal volume (mL)	440 [420–480]
RR (breaths·min^{-1})	20 [16–25]
PSV	2 (7)
IP (cmH_2O)	14 and 16
PEEP (cmH_2O)	6 [6–8]
VAP	2 (22)
MV duration (days)	11 [5–16.5]
Time spent without MV (days)	2 [0–7.5]
ICU stay duration (days)	14 [8.5–23.5]
Mortality	8 (30)

replacing it with water; 2) aspirating absolutely all the air present in the cuff, its inflation channel, as well as in the control cuff by performing multiple fluid purges. The tests were performed in 'static' conditions, without ventilation, and in 'dynamic' conditions, with ventilation, on a test lung, at 1 and 2.5 atm abs.

The data collected were demographic (sex, age, weight) and clinical (HBOT indication, endotracheal tube used, mechanical ventilation (MV) specifics). Patient follow-up was continued until ICU discharge and clinical events related to the management of P_{cuff} such as clinical tracheal ischeamia, days without MV, length of stay in the ICU, and outcome were collected. Statistical analysis was performed using SPSS software (version 20.0, SPSS, Chicago, IL). Results for qualitative variables are presented as numbers (percentage) and for quantitative variables are expressed in median with interquartile range. Pressures before, during and after HBOT were compared using the Wilcoxon signed-rank method.

Results

Between 01 February and 28 April 2016, 59 ventilated patients received HBOT. Twenty-nine patients could not be included because HBOT was urgent or because the session pressure was above 2.5 atm abs. Thus, 30 patients were included in the study; owing to artifacts, only the P_{cuff} and P_{aw} of 27 patients could be analysed.

The median age of patients was 48 (IQR 35–67) years, 81% were men. The median simplified acute physiology score (SAPS-2) was 59 (IQR 39–64). The median time between ICU admission and HBOT was 1 (IQR 1–4) day. In eight of the 27 patients, HBOT was prescribed for cervical necrotizing soft tissue infections, in 11 for necrotizing soft tissue infections in other locations, in 6 for post-anoxic encephalopathy following self-attempted hanging, and in two for other indications (Table 1).

INTUBATION AND VENTILATION

Intubation was orotracheal in 24 (89%) patients, whilst three were intubated with a nasotracheal tube because of airway compression due to cervical necrotizing soft tissue infection. These three patients also required a reinforced endotracheal tube (Mallinckrodt ™ Lo-Contour reinforced). The median internal diameter of the endotracheal tube was 7.5 (IQR 7–7.5) mm. All patients were intubated before admission to our ICU, which is why nine different endotracheal tubes were identified. The Rüschelit® super safety clear™ tube was the most widely used (12/27, 44% of patients). All endotracheal tubes were made of polyvinyl chloride (PVC). Cuffs were cylindrical in 22 (81%) of cases, oval in four (Mallinckrodt™ Lo-Contour reinforced) and conical in one (Mallinckrodt™ Taperguard™ Evac). Mallinckrodt™ Lo-Contour reinforced tubes were the only tubes with high-pressure cuffs. A tube with subglottic suction (Mallinckrodt™ Taperguard™ Evac and Portex®SACETT™) was used for two patients (Table 2).

A Maquet Servo-i HBO® (Getinge, Solna, Sweden) ventilator was used for ventilating three patients; two in pressure support ventilation (PSV) mode because of ventilator weaning and one under assist-control ventilation (ACV) mode. Twenty-four patients were ventilated with a Siaretron 1000 IPER® ventilator (Siare Engineering International Group, Crespellano-Valsamoggia, Italy) in ACV mode. When the ventilatory mode was ACV, the median tidal volume (TV)

Table 2
Endotracheal tube characteristics and manufacturers

Endotracheal tubes	Patients	Cuff shape	Subglottic suction
Rüschelit® super safety clear™ Teleflex, Wayne, USA	12	cylindrical	–
Rüsch® safety clear plus™ Teleflex, Wayne, USA	2	cylindrical	–
Sheridan/HVT® Teleflex, Wayne, USA	1	cylindrical	–
Mallinckrodt™ Lo-Contour reinforced Covidien, Dublin, Ireland	4	oval	–
Mallinckrodt™ Hi-Contour Covidien, Dublin, Ireland	2	cylindrical	–
Mallinckrodt™ oral/nasal tracheal tube cuffed Covidien, Dublin, Ireland	1	cylindrical	–
Mallinckrodt™ Taperguard™ Evac Covidien, Dublin, Ireland	1	conical	+
Portex® clear PVC oral/nasal soft seal® cuff Smith Medical, Minneapolis, USA	3	cylindrical	–
Portex® SACETT™ Smith Medical, Minneapolis, USA	1	cylindrical	+

Table 3
Endotracheal cuff (P_{cuff}) and airway (P_{aw}) pressures at 1 and 2.5 atm abs for all patients (n = 27); Min = minimum; Max = maximum

Parameter	1 atm abs			2.5 atm abs			*P*
	Median [IQR]	Min	Max	Median [IQR]	Min	Max	
P_{cuff} (cmH_2O)	53.9 [24.6–84.7]	7.8	199	38.9 [22.6–61.5]	6.2	191	< 0.001
P_{aw} (cmH_2O)	11 [9.1–17]	6	21	12.2 [8.7–19.2]	6	23	0.024

was 440 (IQR 420–480) mL and the median respiratory rate (RR) was 20·min^{-1} (IQR 16–25). Median positive end-expiratory pressure (PEEP) was 6 (IQR 6–8) cmH_2O.

Median duration of MV was 11 (IQR 5–16.5) days and time spent without MV was 2 (IQR 0–7.5) days. The median stay in ICU was 14 (IQR 8.5–23.5) days; eight of the 27 patients died.

CUFF PRESSURE DATA

Before HBOT, the initial P_{cuff} was 52.9 (IQR 27.6–84.8) cmH_2O with a lowest value of 6.1 cmH_2O, and a highest value of 203 cmH_2O. Back at atmospheric pressure, the final P_{cuff} was 57.1 (IQR 24.6–84.5) cmH_2O. Initial and final P_{cuff} were not statistically different. The median normobaric P_{cuff} was 53.9 (IQR 24.6–84.7) cmH_2O with a lowest value of 7.8 cmH_2O and a highest value of 199 cmH_2O. At 1 atm abs, P_{cuff} exceeded the usual limit of 30 cmH_2O in 19 (70%) of patients, between 20 and 30 cmH_2O in five of patients (5/27) and < 20 cmH_2O in three patients. The average recording time at 2.5 atm abs was 90 min. At 2.5 atm abs the median P_{cuff} was 38.9 (IQR 22.6–61.5) cmH_2O, significantly lower than at 1 atm abs before and after the HBOT session (P < 0.001). At 2.5 atm abs, 18 (67%) of patients had a P_{cuff} > 30 cmH_2O, four between 20 and 30 cmH_2O and five < 20 cmH_2O. The median P_{aw} was 11 (IQR 9.1–17) cmH2O at 1 atm abs and 12.2 (IQR 8.7–19.2]) cmH_2O at 2.5 atm abs (P = 0.024) (Table 3).

PRESSURE DATA FOR RÜSCHELIT® SUPER SAFETY CLEAR™ TUBES

Initial P_{cuff} was 47.7 (IQR 27.1–67.3) cmH_2O. Final P_{cuff} was 47.6 (IQR 23.4–68.9) cmH_2O. Initial and final P_{cuff} were not statistically different. The median normobaric P_{cuff} was 47.2 (IQR 24.4–68.1) cmH_2O with a lowest value of 16.2 cmH_2O and a highest value of 102 cmH_2O. At 2.5 atm abs, the median P_{cuff} was 34.8 (IQR 18.7–49.2) cmH_2O, significantly lower than at 1 atm abs before and after the session (P = 0.002). The median P_{aw} was 12.5 (IQR 9.4–17.3) cmH_2O at 1 atm abs and 14 (IQR 9.1–19.4]) cmH_2O at 2.5 atm abs (P = 0.022).

Discussion

In complete contrast with the expected results, the P_{cuff} was high in water-inflated cuffs. The median P_{cuff} at 1 atm abs when cuffs were water-filled was

Figure 2
An example of P_{cuff} evolution as recorded in one patient during a session of HBOT at 2.5ATA

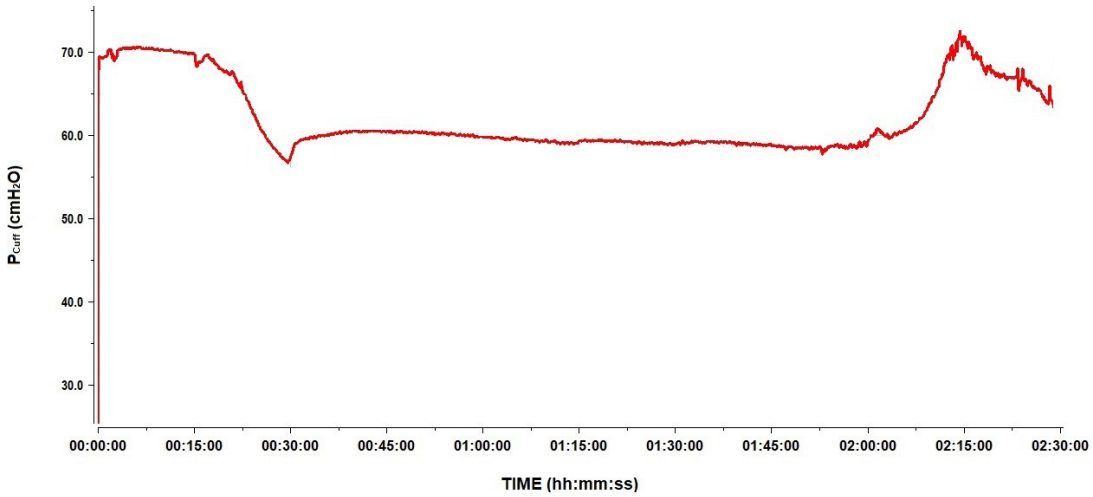


Figure 3
P_{cuff} variation at 1 and 2.5ATA following typical cuff air removal procedure

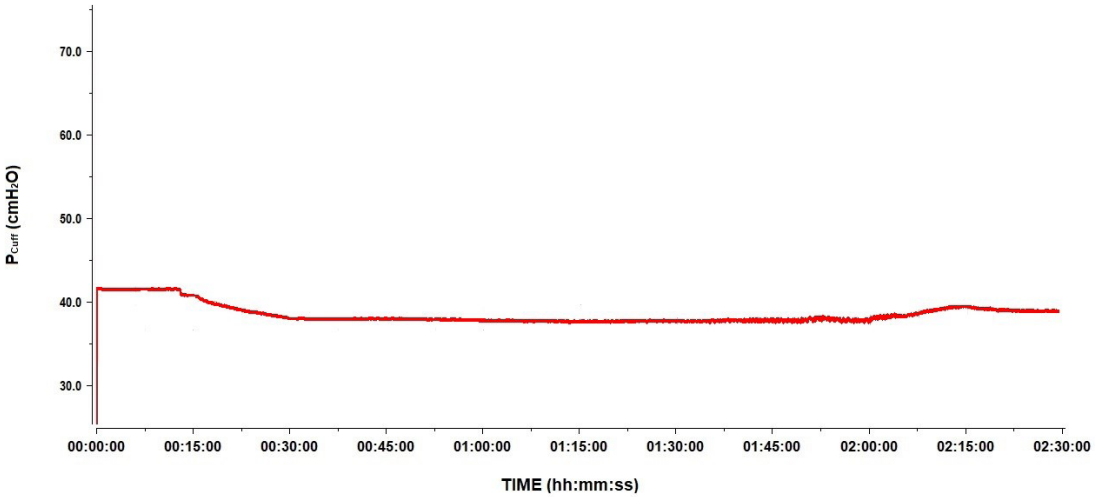
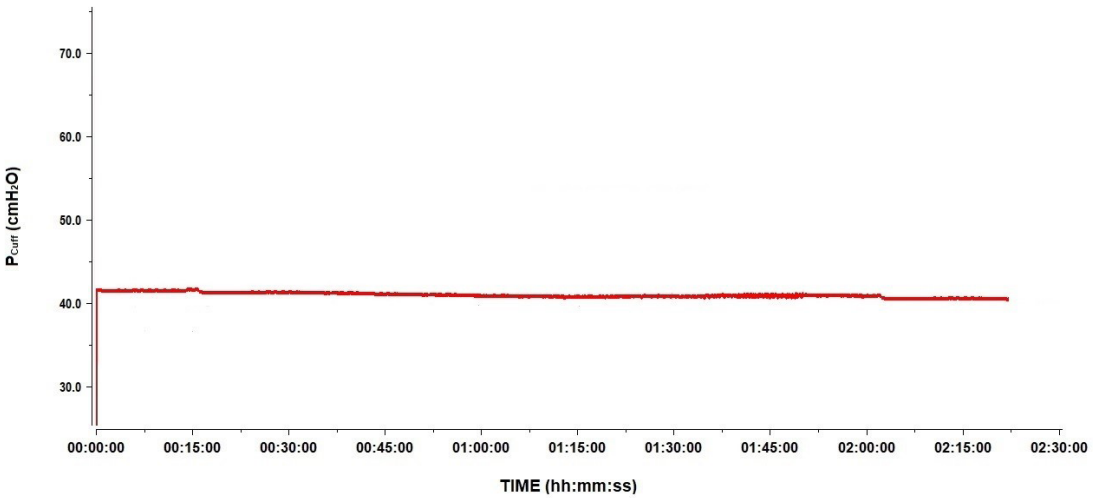


Figure 4
P_{cuff} variation at 1 and 2.5ATA following repeated cuff air removal procedures



53.9 (IQR 24.6–84.7]) cmH_2O (n = 27) and 47.2 (IQR 24.4–68.1) cmH_2O in the 12-patient group with Rüschelit® super safety clear™ tubes. To our knowledge, this was the first time that this parameter was assessed in these conditions. The gauges usually used to control and adjust the P_{cuff} operate only when the cuffs are inflated with air. The experimental system (Figure 1) devised to measure P_{cuff} with water is actually simple and could be made available on a routine basis.

The results are consistent with other data.[4] Without manometric control, cuff inflation with air or water is unpredictable from one patient to another and, therefore, tends to over-inflation.[4] This over-inflation might be explained by the lack of clinical detection. Since air leaks during mechanical ventilation are inversely correlated with P_{cuff}, cuff underinflation can be clinically detected as an audible leak, loss of volume from the ventilation circuit, or detected by the ventilator monitor.[7] This method for assessing pressure is unreliable and does not ensure that a level of P_{cuff} above 20 cmH_2O will be maintained as recommended to avoid microinhalations.[1] In contrast, the tracheal ischaemia potentially induced by cuff over-inflation is not clinically detectable. Nurses palpating the external ('control') cuff on the inflation tube to estimate P_{cuff} is not a reliable method.[9]

A secondary objective of the study was to describe the changes in P_{cuff} during HBOT sessions. At 2.5 atm abs, on average, P_{cuff} decreased by 15 cmH_2O to reach a median P_{cuff} of 38.9 (IQR 22.6–61.5) cmH_2O. This was also observed in the 12 patients intubated with the Rüschelit® endotracheal tube. Back at 1 atm abs, P_{cuff} returned to its original level (Figure 2). Water being non-compressible, we hypothesised an incomplete aspiration of air before replacing it with water in the cuffs. Since any remaining air bubbles are compressible, according to Boyle-Mariotte's Law, their presence results in the decrease in recorded P_{cuff}. To explore this hypothesis, we performed tests as explained in the Methods section. The results are shown in Figures 3 and 4. P_{cuff} remains stable at an ambient pressure of 1 atm abs, but there is a small but clear decrease in P_{cuff} when ambient pressure increases to 2.5 atm abs, with a recovery of the original P_{cuff} after returning to 1 atm abs, whether conditions were static or dynamic (Figure 3). Conversely, after repetitive air removal manoeuvers, P_{cuff} was totally stable, whether at 1 or 2.5 atm abs, under static or dynamic conditions (Figure 4).

Besides the volume of air or water injected, P_{cuff} can be influenced by other factors such as tracheal size and the ratio of tracheal diameter and cuff diameter, cuff type (high or low pressure), thickness, compliance, geometry, curvature of the tracheal tube and position in the trachea.[10–12] Since none of these factors vary during HBOT sessions, they cannot be blamed for hyperbaric P_{cuff} variations. Patient temperature also influences P_{cuff}.[13] In theory, patient cooling could lower cuff pressure, but since ambient temperature increases during a hyperbaric exposure this is unlikely. We conclude that imperfect purging of air from the cuff, is the most plausible cause of P_{cuff} reduction during the period at 2.5 atm abs.

High P_{cuff} appears to be a major risk factor for tracheal ischaemia. The main complication of tracheal ischaemia is the occurrence of tracheal stenosis. The occurrence of tracheomalacia, false obstructive tracheal membranes, tracheo-oesophageal or tracheo-innominate fistulas is unusual. Tracheal mucosa perfusion was reduced at a $P_{cuff} > 30$ cmH_2O and completely suppressed at > 50 cmH_2O.[3] An animal study found that superficial lesions appeared after 15 min of intubation at 27 cmH_2O tracheal pressure.[14] The median P_{cuff} at 1 atm abs for our patients was 53.9 (IQR 24.6–84.7) cmH_2O and median intubation time was 11 days (IQR 5–16.5). In our treatment protocol, most patients requiring HBOT were given two sessions a day. Their cuff remained inflated with water throughout the treatment, for several days. Yet no clinical event related to possible tracheal ischaemia was reported. Literature analysis shows that clinically detected consequences involving tracheal ischaemia are rare events, especially when tracheal ischaemia is not systematically sought. Its incidence has not been evaluated recently. But in tomography of 47 patients after tracheal intubation, a tracheal size reduction greater than 10% was found in 9 cases. None of those tracheal stenoses were symptomatic.[15] In another study, when tracheal stenosis was routinely sought three months after intubation, the incidence was 11%.[16]

One mitigating factor is that the measured P_{cuff} may be different to the tracheal mucosa pressure applied by the cuff. While for a high pressure endotracheal tube, a 30 cmH_2O P_{cuff} generates an equivalent pressure on the tracheal mucosa, the P_{cuff} for a low pressure tube, generates a lower pressure on the tracheal mucosa because of the elastic forces of the cuff material.[17] Half of our patients were intubated with tubes with high-volume low-pressure cuffs (Rüschelit® super safety clear™ and Mallinckrodt™ Taperguard™ Evac). Cuff inflation with water instead of air may still induce a tracheal mucosa pressure different from that expected with air for the same P_{cuff}. However, this has never been studied. Despite the very high P_{cuff} we measured, no clinical events due to tracheal ischaemia occurred. Since this study was observational, it lacks medium-term and long-term bronchoscopy follow-up to screen for tracheal stenosis.

Our study was observational and has not shown any clinical impact, neither regarding cuff overpressure during HBOT nor for P_{cuff} decrease at 2.5 atm abs. Nevertheless, in the light of previous clinical studies and current recommendations, a change in our practice is under consideration, with an evaluation of the effect this change would have. We feel our results should undergo validation by other centres following the same practice.

An alternative would be replacing water with air at the end of each HBOT session. However, if two HBOT sessions

are performed per day, this means replacing water or air in the cuff four times a day. These manipulations are known to be a risk factor for ventilator acquired pneumonia and are thus avoided in our practice. Water is replaced by air if hyperbaric treatment is stopped and the patient remains intubated. Alternatively, an endotracheal tube with an expansile foam cuff could be used (Bivona®Fome Cuff Wire Reinforced, Smith Medical, Minneapolis, USA). The foam cuff is connected to ambient air, thus inflating itself. Under standard conditions with air inflation, this type of endotracheal tube would create fewer tracheal ischaemic lesions than would a high-volume low-pressure cuff tube.[18,19] This system, however, has not been evaluated under hyperbaric conditions. Moreover if used, many ICU patients would require re-intubation since this may not be the usual device with which the patients were intubated. As an alternative, a smart Cuff Manager which monitors and regulates the internal pressure of high-volume, low-pressure cuffs is being tested. This seems promising for sessions at 2.5 atm abs but inefficient at 4 atm abs.

Several devices for the continuous control of tracheal cuff pressure have been successfully tested in ICU patients. They allow a more reliable control of P_{cuff} around a target value than intermittent control.[20–22] Among these devices, only pneumatic pressure regulators, unlike electrical pressure regulators, have shown effectiveness in reducing ventilator acquired pneumonia risk.[23] Such a regulator could be an interesting alternative in the control of initial P_{cuff} and could limit the depression of the P_{cuff} during HBOT. If the cuff is inflated with air, the device must be extremely reactive in order to avoid a major cuff overpressure and rupture during decompression. However, this system requires evaluation during HBOT. This device has never been tested when the cuff is inflated with water.

Finally a pressure transducer could be used to control P_{cuff} as in the present study. This method has previously been described outside HBOT.[24] P_{cuff} control could be continuous during HBOT sessions, with a detection of under- and over-pressure episodes, to which the inside attendant could provide the necessary adjustments. An initial pressure measurement at 1 atm abs may suffice if a complete purge of the air by the method described in the protocol is performed. This method has the advantage of being simple, inexpensive and does not require buying or testing any additional hardware.

There are some limitations to our study. The sample size is small. Also, patients were intubated with many different endotracheal tubes. For ethical reasons, we wished to provide a general overview of some points in our daily practice. The next stage should be to perform a larger study in terms of number of patients all fitted with the same endotracheal tubes. Our results, even though they are not necessarily generalizable, need to be confirmed by further study with a view to avoiding high P_{cuff} and potential tracheal ischaemias.

Conclusions

The median P_{cuff} at 1 atm abs, when the cuff is inflated with water and is not controlled by a dedicated device, is not predictable and usually far above the recommended standards. During HBOT sessions, the P_{cuff} drops, probably due to incomplete air purging of the inflation system. The clinical consequences of these observations have not been evaluated. Measuring water-inflated P_{cuff} is easy. It now remains to be proved whether a complete purge of air from the inflation system could reliably avoid the P_{cuff} drop observed at 2.5 atm abs.

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Assessment of insulin sensitivity during hyperbaric oxygen treatment

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Key words
Endocrinology; Hyperbaric research; Obesity; Metabolism; Physiology

Abstract
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Introduction: Previous studies using a hyperinsulinaemic, euglycaemic glucose clamp have demonstrated an increase in peripheral insulin sensitivity in men with and without Type-2 diabetes mellitus on the third and thirtieth hyperbaric oxygen treatment (HBOT) session. In two studies using different techniques for assessment of insulin sensitivity, we investigated the onset and duration of this insulin-sensitising effect of HBOT.
Methods: Men who were obese or overweight but without diabetes were recruited. One study performed a hyperinsulinaemic euglycaemic glucose clamp (80 $mU.m^{-2}.min^{-1}$) at baseline and during the first HBOT exposure (n = 9) at a pressure of 203 kPa. Data were analysed by paired t-test. The other study assessed insulin sensitivity by a frequently sampled intravenous glucose tolerance test (FSIGT) at three time points: baseline, during the third HBOT and 24-hours post-HBOT (n = 9). Results were analysed by repeated-measures ANOVA.
Results: There was a significant 23% increase in insulin sensitivity by clamp measured during the first HBOT exposure. The FSIGT showed no significant changes in insulin sensitivity.
Conclusions: The hyperinsulinaemic, euglycaemic glucose clamp demonstrated a significant increase in peripheral insulin sensitivity during a single, 2-hour HBOT session in a group of men who were obese or overweight but without diabetes. As an alternate technique for assessing insulin sensitivity during HBOT, the FSIGT failed to show any changes during the third HBOT and 24-hours later, however modification of the study protocol should be considered.

Introduction

While hyperbaric oxygen treatment (HBOT) is not used to treat diabetes mellitus *per se*, it has been observed that when people with diabetes undergo HBOT they may experience a decrease in blood glucose levels (BGL), potentially inducing clinical hypoglycaemia.[1,2] One study showed a substantial average BGL decrease of 3.5 $mmol.l^{-1}$ during a 2-hour HBOT session, with no change in serum insulin concentrations, suggesting an increase in insulin sensitivity as an underlying mechanism.[3]

Insulin resistance is defined as a relative impairment of the action of insulin on target tissues, particularly muscle and liver. The development of insulin resistance is the best predictor of those likely to develop type-2 diabetes mellitus (T2DM) in the future.[4] The inverse of insulin resistance is termed insulin sensitivity. In addition, obesity is strongly associated with the development of insulin resistance and T2DM via activation of a chronic inflammatory state.[5] The insulin resistance has effects on peripheral tissue glucose uptake as well as hepatic glucose production although an important effect is found in the peripheral tissues, specifically muscle.[6]

Of the many techniques available to assess insulin sensitivity, the hyperinsulinaemic, euglycaemic glucose clamp is the gold standard.[7,8] In a preliminary study of men (with and without T2DM) who were receiving a course of 30 HBOT sessions for medical indications, the glucose clamp technique revealed a substantial and significant increase in insulin sensitivity from baseline during their third (37% increase) and thirtieth (41% increase) HBOT sessions.[9] On subgroup analysis, this increase was significant only in those with T2DM, however numbers were small. A subsequent study, again using the glucose clamp technique, enrolled men who were obese or overweight (body mass index (BMI) > 25 $kg.m^{2}$), with and without T2DM.[10] This study demonstrated significant increases in insulin sensitivity during the third daily HBOT session in those with T2DM

(57% increase) and without (29% increase). The increased insulin sensitivity was still measurable 30-minutes after exit from the hyperbaric chamber.

Unanswered questions include how quickly the insulin-sensitising effect of HBOT occurs, how long it persists and its underlying mechanisms. To investigate this, we planned to assess insulin sensitivity during the first HBOT using the hyperinsulinaemic euglycaemic glucose clamp. However, while the glucose clamp technique is accurate, it is labour intensive and made more complicated by being performed within a hyperbaric chamber under pressure. We therefore designed a further study to assess an alternative, technically easier method of assessing insulin sensitivity in the chamber, which, if sufficiently accurate, could be more easily used for repeated studies on the same participant. Having previously shown that the insulin-sensitising effect could be demonstrated in men without T2DM, we designed these studies using men who were obese or overweight (BMI > 25 $kg.m^{-2}$) but without diabetes. This paper reports two studies: the use of the hyperinsulinaemic, euglycaemic glucose clamp to test the effect on insulin sensitivity during the first HBOT session and secondly, the use of a frequently sampled intravenous glucose tolerance test (FSIGT) to assess insulin sensitivity during HBOT and after 24-hours.

Methods

Both studies were approved by the Human Research Ethics Committee of the Royal Adelaide Hospital (RAH121212a, RAH140321) and the University of Adelaide and entered on a trial registry site (NCT02009813; NCT02136615). Both studies were carried out in accordance with the Declaration of Helsinki. All participants provided written, informed consent.

PARTICIPANT SELECTION

Both studies enrolled participants via local advertisement and a web-based recruitment company. Only men were studied as insulin sensitivity in women can vary throughout the menstrual cycle. Other inclusion criteria included age over 18 years with no history of diabetes; participants were obese or overweight (BMI > 25 $kg.m^{-2}$). Exclusion criteria included: prescribed or non-prescribed medication that may affect glucose homeostasis (e.g., corticosteroids); smoking; alcohol intake > 140 $g{\cdot}week^{-1}$; regular, high-intensity exercise (> twice weekly); blood donation or involvement in any other study within the last three months. All participants were assessed for fitness to undertake HBOT by a hyperbaric physician.

HYPERINSULINAEMIC EUGLYCAEMIC GLUCOSE CLAMP STUDY DESIGN

The hyperinsulinaemic, euglycaemic glucose clamp was first described by DeFronzo in 1979.[11] Insulin is infused at a constant rate that is above fasting levels, to stimulate glucose disposal in peripheral tissues but suppress hepatic glucose output. A variable dose glucose infusion is guided by regular blood sampling to measure BGL and 'clamp' the BGL at a pre-determined level (in this case, 6 $mmol.l^{-1}$). After running the infusions for a period of time, a steady-state can be reached where BGL and glucose infusion are stable. At this point, the glucose infusion rate (GIR) is equal to the glucose disposal rate. The GIR is a direct measure of whole body glucose disposal for a given level of hyperinsulinaemia.[8]

Ten participants were enrolled. A dual-emission X-ray absorptiometry scan (DXA) was performed at baseline for all participants to determine fat free mass (FFM). All participants attended the hyperbaric medicine unit after a 10 h overnight fast. Two intravenous cannulae were inserted into contralateral arms, one for the insulin and glucose infusions and the other for blood sampling. A primed insulin (Actrapid, Novo Nordisk, Baulkham Hills, Australia) solution (80 $mU{\cdot}m^{-2}{\cdot}min^{-1}$) was infused for 3.5 h as previously described.[10] Blood samples were taken at 5–10 min intervals and BGL measured by glucometer (Accu-Chek Performa, Roche Diagnostics, Sydney, Australia). BGL was maintained at 6 $mmol{\cdot}l^{-1}$ with a variable infusion of 25% dextrose (Baxter Healthcare, Old Toongabbie, Australia). Insulin sensitivity was determined by the GIR during two separate but consecutive 30-minute steady state (SS) periods in the last hour of the infusion; SS1 corresponded with 2.5–3 h and SS2 with 3–3.5 h. The GIR was standardised for FFM from the DXA scan.

The following day, all participants returned after overnight fasting and the 3.5 h glucose clamp was repeated using the same protocol, this time overlayed with a 2 h HBOT session. The twin-lock, multiplace hyperbaric chamber (Fink Engineering/Cowan Engineering, Australia, 1994) was compressed to 203 kPa followed by breathing 100% oxygen by mask or hood during 90 min at 203 kPa and a 30 min linear decompression to 101.3 kPa. Insulin sensitivity was determined by the GIR during the same two SS periods, so SS1 coincided with the last 30 min of the 2 h HBOT session and SS2 with the first 30 min after exit from the chamber. Statistical analyses were performed using Statistica (version 12, Statsoft, Tulsa, OK, USA). A paired *t*-test was used to compare GIR. Statistical significance was considered at $P < 0.05$.

FREQUENTLY SAMPLED INTRAVENOUS GLUCOSE TOLERANCE TEST STUDY DESIGN

An indirect measure of insulin sensitivity was developed by Bergman in 1979 using mathematical modelling of glucose and insulin data from an intravenous glucose tolerance test.[12] Following the glucose bolus, frequent measurement of blood glucose and insulin are made. The complex relationship between glucose and insulin in the disposal of glucose from the blood is built into pharmacokinetic models that are fit to the data. Parameters that provide best fit are derived.

This includes insulin sensitivity (S_I), defined as fractional glucose disappearance per insulin concentration unit.[8] Other parameters include: glucose effectiveness (S_G), the ability of glucose to promote its own disposal; the acute insulin response to glucose (AIR_G) or first-phase insulin response; the disposition index (DI), a product of insulin sensitivity and insulin secretion, which is a constant. The mathematics to calculate these parameters has been packaged into a commercially available software program (MINMOD Millennium, Pasadena, CA, USA). The FSIGT has shown reasonable correlation with the glucose clamp (r = 0.54).[7]

Twelve participants were enrolled. On the first study day (Day 1) all participants attended the hyperbaric medicine unit at the Royal Adelaide Hospital after a 10-hour overnight fast. A baseline FSIGT was performed in room air with the participant resting in a chair outside of the hyperbaric chamber according to the following protocol. Two intravenous cannulae were inserted into contralateral forearms and blood taken for time zero. A glucose bolus was given into one of the cannulae at time zero over one minute. The weight-dependant bolus used 25% dextrose (Baxter Healthcare, Old Toongabbie, Australia) at 300 mg·kg^{-1} to a maximum dose of 120 ml (30 g dextrose). Blood sampling from the other cannula was performed at 2, 4, 6, 8, 10, 12, 14, 16, 19, 22, 25, 30, 40, 50, 60, 70, 90, 120, 150 and 180 minutes.

Each participant then underwent three HBOT sessions on consecutive days (Days 2–4), with compression to 203 kPa breathing oxygen for 90 min and a 30 min decompression. During the third HBOT session on Day 4, another FSIGT was performed using the same protocol as on Day 1. Compression of the chamber to 203kPa takes 7 minutes and time zero for the dextrose bolus aligned with the start of oxygen breathing during the 90 min period at 203 kPa. A further FSIGT was performed 24 h later on Day 5, in air outside the hyperbaric chamber. The three FSIGTs were performed at a similar time of the day.

Blood samples taken at each of the time points were analysed for glucose and insulin. Insulin was measured by radioimmunoassay (Millipore, St. Charles, MO, USA). Glucose was measured using commercial enzymatic kits on a Beckman AU480 clinical analyser (Beckman Coulter, Brea, CA, USA). All samples for each subject were analysed within the same analytic run to minimise instrument variation. The glucose and insulin data were entered into the minimal model software to derive insulin sensitivity and the other parameters. These measures were statistically analysed by repeated measures ANOVA using SPSS for Windows (Version 22, SPSS, Chicago, IL, USA). Statistical significance was considered at $P < 0.05$.

Table 1
Demographics of participants in the glucose clamp study, $n = 9$; DXA – dual-emission X-ray absorptiometry scan

Parameter	Mean (SD)
Age	47 (5.7)
Height (cm)	176.4 (10.3)
Weight (kg)	97 (15.1)
Body mass index (kg.m^{-2})	31.1 (3.0)
DXA fat free proportion (%)	64.3 (0.1)
Baseline insulin sensitivity (mg.kgFFM^{-1}.min^{-1})	8.57 (3.02)

Results

HYPERINSULINAEMIC EUGLYCAEMIC GLUCOSE CLAMP

One participant sustained a minor middle ear barotrauma during compression at the start of the HBOT. He was removed from the hyperbaric chamber and excluded from the study. Characteristics of the remaining nine participants are shown in Table 1. The GIR data were normally distributed by Shapiro-Wilk and Kolmogorov-Smirnov tests. Figure 1A shows the GIR during SS1 (the last 30 min of the HBOT session). There was a significant increase in insulin sensitivity from Day 1 to Day 2, as measured by the GIR ($t = -2.89$, df = 8, $P = 0.02$). Figure 1B shows the GIR during SS2 (the first 30 min after leaving the chamber), the rise was not statistically significant ($t = -1.87$, df = 8, $P = 0.10$).

FREQUENTLY SAMPLED INTRAVENOUS GLUCOSE TOLERANCE TEST

One participant sustained a minor middle ear barotrauma at the start of compression and was removed from the hyperbaric chamber; another withdrew for personal reasons. On laboratory analysis, another participant had glucose and insulin levels on arrival for the FSIGT on the third HBOT and again 24 h later which suggested a failure to follow the fasting protocol, and these data were excluded. Characteristics of the remaining nine participants are shown in Table 2. The results of the minimal model analysis of the FSIGT are shown in Table 3. Data sets for all parameters showed large variances and there were no significant changes in any of the measured parameters.

Table 2
Demographics of participants in the FSIGT study, $n = 9$

Parameter	Mean (SD)
Age	37.1 (13)
Weight (kg)	99.3 (15.2)
Height (cm)	172.6 (3.8)
Body mass index (kg.m^{-2})	33.2 (4.1)

Figure 1
(**A**) Glucose infusion rate (GIR) at baseline vs. HBOT during SS1 (last 30 min in chamber); (**B**) GIR at baseline vs. HBOT during SS2 (first 30 min after HBOT); * $P = 0.02$

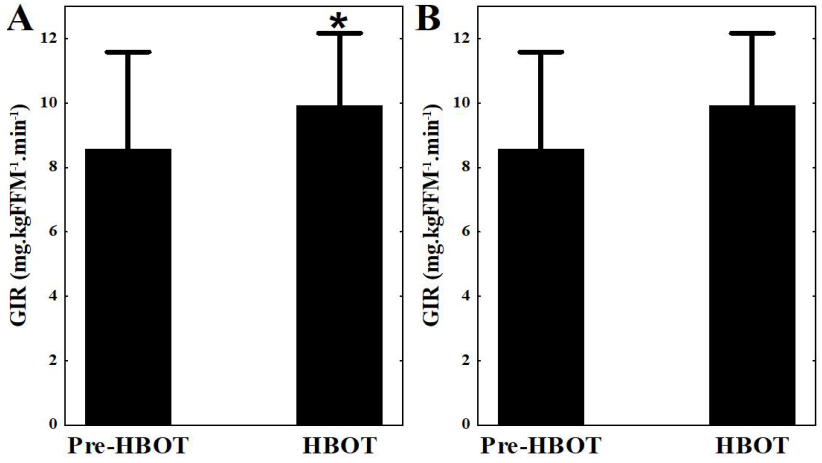

Table 3
Insulin sensitivity and other parameters derived from minimal model analysis; data are mean (SD); S_I = Insulin sensitivity, S_G = Glucose effectiveness, AIR_G = Acute insulin response to glucose

Parameter	Day 1	Day 4	Day 5
S_I ($mU \cdot l^{-1} \cdot min^{-1}$)	3.35 (1.27)	3.82 (2.09)	4.23 (3.38)
S_G (min^{-1} x100)	1.55 (0.79)	1.58 (0.92)	1.48 (0.82)
AIR_G ($mU \cdot l^{-1} \cdot min^{-1}$)	720 (462)	573 (275)	706 (364)
Disposition index	2304 (2004)	1862 (1115)	2165 (1089)

Discussion

Using an in-chamber hyperinsulinaemic euglycaemic glucose clamp technique, we have previously shown that routine HBOT typically used for clinical indications is associated with significant increases from baseline in peripheral insulin sensitivity on the third day of daily HBOT sessions.[9,10] Utilising the same clamp technique, we have now found that the HBOT-induced increase in insulin sensitivity occurs during the very first HBOT session. This study also confirms the previous findings that the insulin-sensitising effect of HBOT can be identified in overweight/obese men without diabetes and is not specific to those with diabetes. The findings that the effect can be identified during the first HBOT exposure and in men without diabetes should make future studies examining the effects of HBOT on insulin sensitivity and the effects underlying them easier to undertake.

In our previous study using the glucose clamp technique, HBOT significantly increased insulin sensitivity not only during the final 30 min of the 2 h spent under HBOT conditions, but also during the first 30 min after exit from the hyperbaric chamber, when performed on the third HBOT exposure.[10] The current study used the glucose clamp technique on the first HBOT and found significantly increased insulin sensitivity under hyperbaric conditions (during SS1). In contrast, there was not a significant increase over baseline insulin sensitivity during the first 30 min after leaving the chamber (SS2). There is a trend towards an increase in insulin sensitivity, however small sample size and large variance in the data make statistical significance more difficult to achieve. Another consideration as to why SS2 did not achieve significance in the current study could be that one HBOT has less impact than three; there was a 23% increase in insulin sensitivity during the first HBOT compared to a 29% increase in men without diabetes during the third HBOT.[10] There may be some accumulation of the HBOT effect with repeated exposures, however its duration of effect is not known. It is clear however, that one 2 h HBOT session is sufficient to see a change in insulin sensitivity. This finding is also consistent with clinical practice in hyperbaric medicine where anecdotally, people with diabetes have experienced a fall in their BGL during their first HBOT session.

Our previous studies performed the clamp on the third HBOT session for two reasons: to improve the chances of identifying an effect if some accumulated exposure was important, and also to give the participant the opportunity to practice middle ear equalisation manoeuvres that are required during pressurisation of the hyperbaric chamber,

prior to undergoing the glucose clamp procedure. While potential difficulty with ear equalisation was assessed during their initial medical review, middle ear barotrauma continues to be the most frequent complication associated with clinical HBOT (approximately 2%).[13] Indeed, one of our participants in this study had been established on his second glucose clamp with infusions of glucose and insulin when he was wheeled into the chamber only to find he could not satisfactorily equalise his ears on compression, resulting in his removal from the chamber and from the study. Despite the small sample size in this study, a significant increase in insulin sensitivity was identified, consistent with the two previously published studies.

Our attempts to replace the glucose clamp technique with the simpler FSIGT have not been successful. While the FSIGT requires frequent blood sampling over several hours, it avoids the necessity of passing samples through the medical lock for immediate glucometer analysis and the rapid decisions required to maintain blood glucose concentrations during a glucose clamp. However, under the same HBOT conditions as in our three glucose clamp studies, all of which showed increased insulin sensitivity during the first or third HBOT session, we found no significant effect of HBOT on insulin sensitivity when assessed by the FSIGT during the third HBOT and at 24 h later.

There are a number of reasons the FSIGT may have failed to pick up such an effect. First, the sample size was small and there was substantial variation in the data. Second, the FSIGT is known to be less reliable in people with insulin resistance. Several modifications to this technique have been suggested, such as giving tolbutamide or an insulin infusion early in the FSIGT, which has improved the correlation with glucose clamp studies.[7] However, in pursuit of a simpler technique and with a group of men without diabetes, we did not modify the FSIGT.

Third, and perhaps more likely, we performed the FSIGT too soon after the participants started their HBOT session. While we have demonstrated an increase in insulin sensitivity during steady state periods 2.5 to 3.5 h into the clamp (at the end of an HBOT exposure), we have not specifically tested insulin sensitivity earlier in the HBOT session using a glucose clamp technique. If the insulin-sensitising effect of HBOT requires some duration of exposure to activate, then giving the glucose bolus of the FSIGT at the beginning of the HBOT session may not be the best time. The bulk of the glucose disposal would have taken place in the early part of the HBOT session and missed a later-onset effect identified in the clamp studies. Future studies using the FSIGT should perform the procedure towards the end of the HBOT session. On a cautionary note, such a study design may create the potential for the fasting participant with diabetes to develop hypoglycaemia during their HBOT session prior to the FSIGT, and they would need regular monitoring of their in-chamber BGL. If hypoglycaemia occurred during the HBOT, intervention would be required and the FSIGT would not be able to proceed.

The third FSIGT performed 24-hours post HBOT also did not demonstrate an effect of HBOT on insulin sensitivity, but we cannot say whether this is because such an effect was not present (i.e., a stimulatory effect of the previous day's HBOT had worn off), or whether such an effect was present but could not be detected due to limitations with the FSIGT technique.

The FSIGT was chosen because it was anticipated to be easier to perform and more easily tolerated by the participant than the glucose clamp. In the end, both techniques were found to be labour-intensive in a hyperbaric chamber. Importantly for undertaking assessment of insulin sensitivity in the novel environment of a hyperbaric chamber, every endeavour was made to perform these techniques according to established protocols. The fasting participants were tested at the same time of the day. They were kept sedentary in comfortable chairs for the duration of the study and wheeled into and out of the hyperbaric chamber. The glucometer utilised a glucose dehydrogenase reagent as opposed to glucose oxidase, making it less sensitive to ambient oxygen pressures.[14]

Our hyperbaric facility, along with many others, manages potential hypoglycaemia in patients with diabetes by monitoring their BGL before they enter the hyperbaric chamber and by repeating it if clinically indicated. Continued investigation is warranted in this field, both for the safety of hyperbaric patients with diabetes but also for the potential to identify novel pathways of glucose control.

Conclusion

The glucose clamp performed during the first HBOT session demonstrated a significant increase in insulin sensitivity, earlier than in our previously published studies which showed an increase in insulin sensitivity in men with and without diabetes on the third and thirtieth HBOT.[9,10] The hyperinsulinaemic, euglycaemic glucose clamp appears to be a useful tool to undertake these investigations. The FSIGT in its current design is probably not a good tool to assess insulin sensitivity in a hyperbaric chamber.

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The evaluation of in-chamber sound levels during hyperbaric oxygen applications: Results of 41 centres

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Key words
Noise; Hyperbaric facilities; Health; Hearing; Noise-induced hearing loss (NIHL); Multiplace chamber

Abstract
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Introduction: Noise has physical and psychological effects on humans. Recommended exposure limits are exceeded in many hospital settings; however, information about sound levels in hyperbaric oxygen treatment chambers is lacking. This study measured in-chamber sound levels during treatments in Turkish hyperbaric centres.
Methods: Sound levels were measured using a sound level meter (decibel meter). All chambers were multiplace with similar dimensions and shapes. Eight measurements were performed in each of 41 chambers; three during compression, three during decompression, and two at treatment pressure, one during chamber ventilation (flushing) and one without ventilation. At each measurement a sound sample was collected for 25 seconds and A-weighted equivalent (LA_{eq}) and C-weighted peak (LC_{peak}) levels were obtained. Recorded values were evaluated in relation to sound level limits in regulations.
Results: The highest sound level measured in the study was 100.4 dB(A) at treatment pressure while ventilation was underway and the lowest was 40.5 dB(A) at treatment pressure without ventilation. Most centres had sound levels between 70 dB and 85 dB throughout the treatment. Ventilation caused significant augmentation of noise.
Conclusions: The chambers were generally safe in terms of noise exposure. Nevertheless, hyperbaric chambers can be very noisy environments so could pose a risk for noise-related health problems. Therefore, they should be equipped with appropriate noise control systems. Silencers are effective in reducing noise in chambers. Thus far, hyperbaric noise research has focused on chambers used for commercial diving. To our knowledge, this is the first study to investigate noise in hospital-based chambers during medical treatments.

Introduction

There are miscellaneous definitions for noise in acoustics or phonology, but it can simply be defined as unwanted sound. Basically, there is no difference between sound and noise. Sound waves can be perceived as speech, music or noise depending on the individual.[1] Noise in health sciences is accepted as a source of stress and has long been known to have physical and psychological effects on humans.[2] Hearing impairment known as noise-induced hearing loss (NIHL) is the most apparent impact, however, many other influences on body functions have been observed. It has been associated with high blood pressure and increased coronary heart disease risk as well as hormonal and psychosocial disturbances.[3–5] In addition, there is growing evidence that noise contributes to burnout and error risk related to impaired concentration and miscommunication.[6,7]

NIHL may develop after exposure to impulsive (instant high level) sounds. The human ear senses sounds between 0–140 dB. Whereas a noise of 120 dB causes discomfort in the ear, sounds between 125–135 dB cause pronounced pain. At 140 dB, tympanic membrane rupture may be seen and permanent damage might occur.[8,9] Prolonged and repeated exposure to lower sound levels can also deteriorate hearing and cause gradual impairment. To avoid damage, noise standards that set out exposure limits and measures to be taken for hearing protection have been determined.[2]

There are various sources of noise in daily life. Humans are exposed to noise from industry, transportation, recreation and work. According to the World Health Organization (WHO), the maximum level of noise exposure should not exceed 85 dB in daily life. Work is one of the places that humans spend most of their time and are exposed to noise. Therefore, regulations for worksites have also been developed and maximum sound levels at which an employee can work with respect to time are well defined. These regulations also mandate actions such as hearing protections or reducing sound at source when limits are exceeded. Noise standards

Table 1
Maximum daily exposure times with respect to sound levels according to Turkish regulations

Sound level (dB)	Exposure time (hours)
85	8
87	6
90	4
92	3
95	2
97	1.5
100	1
105	0.5
110	0.25

vary among countries, but generally, in developed countries the acceptable maximum noise level is 85–90 dB(A), for five days a week and eight hours per day. A'3dB doubling factor' which implies that an increase of three dB in sound level requires a reduction of exposure time by two, is applied to these limits.[10] Occupational noise standards in Turkey are defined in the legislation *Regulation on the Protection of Employees from Noise-related Risk*s and these are similar to other global standards.[11] The maximum allowed sound levels with respect to exposure times in Turkish regulations are given in Table 1.

Hospitals are worksites where occupational noise can be encountered. Medical equipment, alarms, portable vehicles, personnel activities, communication systems, and air conditioning and ventilation systems are some sources of noise.[12] Although not mandatory, there are recommendations for hospital noise. Sound levels should not exceed 30 dB and peaks should not be over 40 dB in hospitals according to the WHO. Similarly, the Environmental Protection Agency (EPA) recommends a maximum sound level of 45 dB(A).[1,13] It has been shown in many studies that these limits are exceeded, especially in intensive care units.[14,15]

Hyperbaric oxygen treatment centres can also be noisy environments. According to the European regulation for pressure vessels for human occupancy (EN 14931), the average sound level should not exceed 70 dB(A) at treatment pressure with (maximum) ventilation on, and 90 dB(A) during compression and decompression.[16] Studies have been performed in chambers used in diving operations but few studies have focused on sound levels in hospital-based chambers. The aim of this study was to measure in-chamber sound levels in different hyperbaric oxygen treatment (HBOT) centres in Turkey and to evaluate the possible effects on patients and health care providers by comparing the measured sound levels with international standards.

Methods

The study was approved by our Institutional Review Board. It was supported by Istanbul University Scientific Research Fund (Project No.: 20326).

All HBOT centres in Turkey were contacted either by phone or e-mail and the study was explained in detail. Sound level measurements were planned with centers that agreed to participate. Measurement days were randomly selected but were always weekdays on which treatments were conducted. All participating centres had cylindrical, steel multiplace chambers with similar dimensions. All chambers were equipped with similar furnishings, piping systems and internal instruments. Compression and decompression rates were similar for all chambers and ranged between 10–12 kPa·min^{-1} (equivalent to 1–1.2 metres' seawater [msw] per minute). Sound levels were measured in the chamber; three times during compression, three times during decompression and two times at treatment pressure (243 kPa, [2.4 atmospheres absolute [atm abs] pressure]), one with ventilation and one without. In this context 'ventilation' refers to a process where gas is flushed into and vented from the chamber at equivalent rates such that the pressure within remains constant. In many jurisdictions this is referred to as 'flushing'. Measurements during compression were performed between 15–30 kPa, 60–75 kPa and 120–134 kPa pressures (1.5–3 msw, 6–7.5 msw and 12–13.5 msw depth equivalents). Measurements during decompression were performed in reverse order.

Sound level measurements were performed using a Bruel & Kjaer Type 2240 sound level meter (SLM) (Bruel & Kjaer, Naerum, Denmark) and Bruel & Kjaer type 4231 sound level calibrator which is compatible with the SLM (Figure 1). This device is an integrated – average field

Figure 1
Bruel & Kjaer Type 2240 SLM (left) and Bruel & Kjaer type 4231 sound level calibrator (right) used for measurements

Type 1 sound meter and complies with International Electrotechnical Commission (IEC) 61672-1 standards. It can measure sound pressure levels between 30 to 140 dB(A) and frequencies between 20 Hz to 16 kHz. The device can operate between -10°C and 50°C and for 16 hours on two 1.5 Volt LR6/AA alkaline batteries. It weighs 245 g and is portable so can be carried easily to measurement spots. Information about the compatibility of device in hyperbaric conditions was provided by the manufacturer prior to performing the study.

All measurements were performed during routine HBOT sessions. The SLM was placed at least one metre away from the sides of the chamber and 130 cm above the floor, which would be the ear level of a sitting patient. At each measurement interval, a sound sample was collected for 25 seconds and A-weighted equivalent continuous sound levels (LA_{eq}) and C-weighted peak sound levels (LC_{peak}) were obtained. LA_{eq} defines the equivalent of total sound energy measured over a period of time and is basically the average sound level. LC_{peak} shows the instantaneous highest sound level. Before each measurement during compression and decompression the SLM was calibrated because the pressure in the chamber changes continuously. The calibration level was 94 dB.

Measured LA_{eq} and LC_{peak} values in dB(A) and dB(C), respectively, were recorded in Microsoft Excel® 2016. Recorded values are presented descriptively and evaluated in means of sound level limits in regulations. Statistical analysis was performed using the Med-Calc® for Windows (version 11.2.1.0). Data distribution was evaluated using the Kolmogorov-Smirnov test and Student's *t*-test was used to compare paired samples. Significance was accepted at $P < 0.05$.

Results

Forty-one HBOT centres from eight different cities participated in the study. The highest LA_{eq} (equivalent continuous sound level) measured in the study was 100.4 dB(A) at the treatment pressure during ventilation and the lowest was 40.5 dB(A) at treatment pressure without ventilation. The highest and lowest sound levels recorded at compression, treatment depth and decompression throughout the study are given in Table 2. The distribution of centres with respect to sound levels at each sample collection interval is given in Table 3.

Most of the centres had sound levels between 70 dB(A) and 85 dB(A) throughout the treatment, whereas only four were lower than 70 dB(A). These four were those with sound levels lower than 70 dB(A) at treatment depth both with ventilation on and off. Thirteen centres exceeded the 85 dB(A) limit at treatment depth with the ventilation on but all were below this limit when the ventilation was off. The sound levels were found to be significantly higher when the ventilation was on in all centres. ($P < 0.001$)

Other than the four centres that were below 70 dB(A) throughout treatment, another three and four centres were below 70 dB(A) all through compression and decompression, respectively. Three exceeded the 85 dB(A) in all three measurements of compression. Only one centre was over 85

Table 2

Highest and lowest L_{eq} and L_{peak} values during compression, treatment depth and decompression in the study; Vent. = ventilation

Parameter		Compression	At treatment pressure		Decompression
			Vent. on	Vent. off	
L_{eq} dB(A)	Highest	95.6	100.4	79.0	94.0
	Lowest	58.6	63.9	40.5	47.7
L_{peak} dB(C)	Highest	109.3	113.6	99.1	106.7
	Lowest	76.0	85.7	74.5	77.5

Table 3

Number of centres with respect to measured sound level in each sample collection interval. C1, C2 and C3 – measurement intervals at the beginning, midway through and towards the end of compression; D1, D2 and D3 – measurement intervals at the beginning, midway through and towards the end of decompression; Vent. = ventilation

Sound level dB(A)	Compression (*n*)			Treatment depth (*n*)		Decompression (*n*)		
	C1	C2	C3	Vent. on	Vent. off	D1	D2	D3
≤ 70	15	11	8	4	19	10	13	16
70.1–85	23	25	27	24	22	29	26	22
> 85	3	5	6	13	–	2	2	3
> 90	2	3	3	5	–	2	1	2

dB(A) all through decompression. Few exceeded 85 dB(A) in one or two measurement points during compression or decompression.

When the '3 dB doubling factor' was taken into account, a sound level of 95 dB(A) at treatment pressure and 105 dB(A) during compression and decompression could be permissible. In this case, only three centres exceeded the limit at treatment pressure with ventilation working. None remained over the limits all through treatment. Also, none exceeded the allowed LC_{peak} levels in any sample collection interval.

Discussion

Sound is a pressure wave that is formed by a vibrating object and travels through a medium by transferring energy from one particle to another. Sound pressure, which is the deviation in atmospheric pressure by a sound wave, is the most important parameter to understand its effects. The human ear can sense sound pressure between 20 µPa to 100 Pa. These two values are separated by a factor of more than a million, thus it is not practical to obtain sound pressure measurements in a linear scale of Pa since the range would be too wide. Accordingly, sound pressure level (SPL), which is the logarithmic ratio of a measured value to a reference value, namely 20 µPa is used for acoustic parameters. SPL is measured using a SLM and expressed in decibels (dB).[1]

Another parameter important in sound measurements is frequency weighting. Frequency is the number of sound waves passing a fixed point per second and measured in Hertz (Hz). The human ear can hear between 20 Hz to 20 kHz but is more sensitive to frequencies between 500 Hz to 8 kHz and less sensitive to very high and low pitches. A measurement device, on the other hand, does not have this selectivity. To ensure that a SLM measures what a human ear perceives, frequency weighting that filters the relative strength of various frequencies is used. The most common one is A-weighting, as it is accepted to be the most approximate frequency response to human hearing.[8] It cuts off the very low and very high frequencies that an average human cannot hear. C-weighting, on the other hand, also takes extreme high and low frequencies into account and is more commonly used for measuring peak sound levels. Measured sound levels are expressed as dB(A) or dB(C).

Hearing under pressure may differ from hearing at atmospheric pressure due to changes in acoustic parameters of the media through which a sound wave travels.[17] It has been shown that the hearing threshold increases underwater because bone conduction, which has less contribution to hearing compared with air conduction, becomes the major way sound is transmitted when the tympanic membrane is in contact with water (known as wet ear).[18] In other words, humans are less sensitive to sound underwater and higher sound levels would have less impact.[19] Despite this, studies have revealed divers may face noise-induced hearing impairment.[20,21] In dry hyperbaric environments, on the other hand, threshold shift has not been detected either with air or other gases, so susceptibility to noise is not thought to be different from normal air.[18] In addition, chambers are confined environments and can be noisy due to the turbulence generated from high pressure gas merging into still gas and passing through pipes during compression and ventilation. Also, cylindrical chambers are highly reflective for sound waves.[18,22] In fact, a study that questioned patient experience of hyperbaric treatment in Australia showed that noisiness in the chamber was one of the primary reasons for discomfort.[23] Yet, there are only a few studies discussing sound levels in chambers even though noise can reach sufficiently high intensities as to cause health hazards during hyperbaric interventions.

In a 1970 report, sound levels were measured in a US Navy chamber during compression and decompression with average rates of 210 kPa·min^{-1} (21 msw·min^{-1}) and 180 kPa·min^{-1} (18 msw·min^{-1}), respectively. The sound levels were over 100 dB(A) in both.[24] Later, sound levels were measured in a US Navy chamber during compression and decompression, both at 180 kPa·min^{-1} (18 msw·min^{-1}) and chamber ventilation at different depths. Almost all measurements read over 110 dB(A) and the highest sound level was 121 dB(A) at a pressure of 150 kPa (15 msw).[25] A series of measurements performed in British Royal Navy chambers revealed similarly high sound levels.[18] In recent decades, hospital-based chambers, which are generally operated at much lower compression and decompression rates than those used in the above studies and which are fitted with newer systems and equipment have prevailed. Until this study, there has been a lack of information regarding noise in these chambers, although they are mostly reserved for patients who are likely less used to and are expected to be more sensitive to noisy environments compared with industrial and navy divers.

In this study, sound levels in 41 different hyperbaric chambers were measured during compression, decompression and at treatment pressure. It was found that most of the chambers were under occupational noise level limits during treatment, although most exceeded the European pressure vessel standard at treatment pressure. Also, it was seen that ventilation increased the noise in the chamber significantly. Yet, chambers in this study can generally be considered safe in terms of noise for usual two-hour treatments. However, if longer treatment tables, such as the US Navy Table 6, are needed, some chambers may entail a risk. Noise has been shown to have adverse effects on patient outcomes, besides well-known noise-related health problems.[26] Studies investigating effects on patients suggest that prolonged noise exposure is related to slower healing, longer hospitalisation and increased pain medication.[27] In this regard, use of hearing protection may be considered for longer treatments or during ventilation in chambers in which higher sound levels are encountered.

The measured sound levels reported in this study are lower than in previous reports; however, the intended purpose of navy diving chambers, much higher compression rates and variability of measurement techniques are notable in terms of this comparison. A major difference that should not be ignored is the presence of advanced silencers in the chambers in which we performed measurements. Silencers and mufflers are effective ways of controlling noise in hyperbaric chambers. They are usually installed at inlets of air pipes or exhausts and reduce the sound transmission while allowing the free flow of air. Attenuation in the range of 20 to 40 dB was shown in a study performed in chambers and diving bells equipped with different designs of silencers.[22] Tests were conducted at pressures between 101.3 kPa (1 atm abs) in air to 608 kPa (6 atm abs) and heliox. In another study where four different silencers were compared during decompression from 506 kPa (5 atm abs), it was seen that the measured sound levels varied greatly.[17] Thus, the presence of a silencer and its design and attenuation capacity are all important for effective noise control. This may explain the variability of sound levels in our study since all chambers were equipped with silencers. Cladding chambers with sound absorbent materials might be another option for noise control; however, it should not represent a fire risk or cause hygiene problems.

Staff working around the chamber, especially chamber operators, may be exposed to noise, probably for long hours. The present study focuses on the noise in the chamber and does not reflect the exposure in the vicinity but such measurements may provide an insight. Sound levels around chambers should also be determined for the prevention of possible long-term health hazards for staff working in hyperbaric units.

LIMITATIONS

It is known that even small changes of conditions within a space may cause alterations in the sound field. Despite the similar structure of the chambers, the number of occupants during measurements was not the same. Also, the interior designs differed slightly. Therefore, a direct comparison of the chambers in terms of noisiness is not possible and was not the aim of the study. In addition, for a given chamber the measured sound level could have been different with a different number of occupants or a change in interior configuration, but the size of this effect is not predictable. The effects of chamber occupants and interior design on sound levels may be investigated in further studies.

Another important point in noise measurement is its effect on people. Even if the measured sound levels are within permitted limits, it is possible that patients and staff perceive it as disturbing due to hearing differences or confined space anxiety. Therefore, the impact of measured sound levels on comfort and health also needs to be evaluated. Further studies focusing on the perception of occupants should be conducted to claim that hyperbaric chambers are truly safe in terms of noise.

Conclusion

This study revealed that hyperbaric chambers can be noisy during ventilation and sound levels in the chamber may exceed safe limits when longer treatments are administered. In this regard, an assessment for compliance with noise regulations can be recommended for all hyperbaric chambers. Measures to minimize the impacts can be considered for chambers or operations that would pose a risk. Also, national legislations on hyperbaric chambers should be regulated for noise standards and chamber manufacturers should be obliged to comply with requirements. To our knowledge, this is the first study to focus on noise during treatments in hospital-based hyperbaric chambers and may serve as a pilot study for further research.

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An observational trial to establish the effect of hyperbaric oxygen treatment on pelvic late radiation tissue injury due to radiotherapy

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Abstract
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Introduction: Rates of pelvic cancer are growing globally with around half of these patients receiving radiotherapy. In a small proportion, radiotherapy results in significant late radiation tissue injury (LRTI) to surrounding tissue, most commonly affecting the bladder and bowel mucosa. We conducted a combined prospective and retrospective observational trial to establish the effectiveness of hyperbaric oxygen treatment (HBOT) in improving the symptoms and signs of LRTI in these patients.

Methods: Fifty-two patients were included after receiving radiotherapy for cancers of the bowel, bladder, cervix, prostate or vulva. They received HBOT at 203–243 kPa (2.0–2.4 atmospheres absolute (atm abs)) for 90 minutes with the median number of treatments being 30 (IQR 1). Late effects normal tissues – subjective, objective, management, analytic (LENT-SOMA) scores were recorded before and after treatment.

Results: The mean LENT-SOMA scores before and after HBOT were 11.7 (SD 5.3) and 8.1 (5.1) respectively. This reduction in score of 3.7 (95% CI 2.6 to 4.8) was statistically significant ($P < 0.001$). For radiation cystitis the mean reduction was 3.7 (95% CI 2.4 to 5.0, $P < 0.001$) and for radiation proctitis was 3.8 (95% CI 1.4 to 6.1, $P = 0.004$). There were no significant adverse effects recorded.

Conclusions: Hyperbaric oxygen treatment may be an effective and safe treatment for pelvic late tissue radiation injury.

Introduction

Cancer is a significant issue worldwide, causing nearly one in six deaths globally.[1] Pelvic tumours make up the largest group of solid cancers in the USA.[2] One of the most frequent modalities of treatment is radiotherapy with around half of cancer patients receiving either curative or palliative radiotherapy. Whilst effective at eliminating cancer cells, there is unavoidable damage to surrounding tissues. These effects are often divided into early (within weeks) and late (months to years). The early phase involves DNA damage and cell death (commonly during mitosis or through apoptosis) and is usually self-limiting.[3,4] It characteristically affects rapidly proliferating cells such as the mucosa of the bowel and bladder. In contrast, the late phase is driven in part by chronic oxidative stress and abnormal cytokine cascades.[5] This leads to chronic inflammation, progressive endarteritis, hypoxia and fibrosis.[6] Once again, the most affected tissues are the mucosal surfaces. Of all patients receiving pelvic radiotherapy around 5–18% will develop symptomatic late radiation tissue injury (LRTI).[7]

The clinical manifestations of this process are organ specific. In the rectum, they vary from mild (minor bleeding, excessive mucus production, tenesmus, diarrhoea and urgency) to severe (major bleeding, ulceration, stricture and fistula formation). In the bladder, frequency, incontinence and haematuria with clot retention are common. In severe cases of both rectal and bladder injury, blood loss can result in significant anaemia and require repeated blood transfusion and/or surgical removal of the organ. The severity of these symptoms is largely dependent on cumulative radiation dose and the area of tissue affected, and is often responsible for a significant reduction in quality of life.[8–10] Despite this, there is wide variability between patients who have received the same radiation dose.[11]

Advances in cancer treatment mean an ever-increasing number of survivors, with around half of patients being long-term survivors.[12] This suggests an increasing number of patients may suffer from LRTI in the future and has led to an increased interest in methods to reduce this substantial burden. Conventional approaches involve either medical or surgical symptom control, the cost of which commonly totals tens of thousands of dollars per year.[13] Unfortunately, these have limited efficacy or unpleasant side effects of their own. Hyperbaric oxygen treatment (HBOT) has for some time been reported as useful in LRTI.[14–17] However, there are also

data to support the contrary view. For example, a randomised controlled trial (RCT) published in 2016 demonstrated no improvement in chronic bowel dysfunction with HBOT.[18] High quality trials involving HBOT are difficult to undertake for a number of reasons and to date the only other four RCTs published in pelvic LRTI were crossed-over in the short term,[17] unblinded,[19,20] or both.[21] The majority of reports are non-controlled retrospective or observational studies, often vulnerable to regression to the mean and placebo effect.[15] In light of this, multiple authors have suggested that further research is needed.[14,22]

The aim of this study was to evaluate the effectiveness of HBOT for ameliorating the symptoms and signs of pelvic LRTI presenting to our clinical service. We hypothesised that HBOT is an effective treatment for these patients. We also aimed to evaluate the use of a long proposed, but little used, system for grading these symptoms and signs: the 'late effects normal tissues – subjective, objective, management, analytic (LENT-SOMA) scoring system'. In particular, we want to evaluate both the ease of use and practicality of this score of clinical severity for incorporation into a prospective registry under development.

Methods

The study was approved by the Prince of Wales Human Research Ethics Committee (HREC 17/010(LNR/17/POWH/24). Informed consent was waived on the basis that all data is obtained routinely from all patients in our unit. The study was conducted at the Prince of Wales Department of Diving and Hyperbaric Medicine. We recruited patients retrospectively who completed treatment from July to December 2017 and prospectively from January to April 2018. The study subjects were drawn from patients accepted for treatment during the study period. Inclusion criteria were: a diagnosis of pelvic LRTI made by the referring physician based on symptomatology or objective findings on endoscopy. Endoscopic evaluation is preferred as it allows for exclusion of recurrence of cancer, which can present similarly to LRTI. It also allows for objective assessment of treatment response when repeated during the post-HBOT period.

TREATMENT PROTOCOL

Treatments were once a day Monday to Friday, for six weeks (30 treatments planned in total). Most patients were treated in a multiplace chamber breathing oxygen using a hood or mask at 243 kPa (2.4 atmospheres absolute [atm abs]) for 90 minutes. The remainder were treated in a monoplace chamber, breathing 100% O_2 at 203 kPa (2.0 atm abs) for the same length of time. Both groups had a 5-minute air break at 45 minutes. Historically these two treatments have been considered roughly equivalent in terms of oxygen dose; allowing for some ambient air entrainment in the multiplace system. We aimed to minimise any gaps in treatment but sometimes this was not possible due to patient circumstances, appointments at other medical facilities or complications such as two to three days off recovering from barotrauma to the middle ear. After completion of this initial course, patients were discharged home and reviewed one month later for the consideration of a further course of treatment if required, to a maximum total of 50 sessions.

DATA COLLECTION

Symptoms were evaluated before starting and after finishing treatment. Where possible this was performed by the same doctor. We used the 'bladder' and 'bowel' domains of the original LENT-SOMA scoring system. This system was created in 1995 to address a need for a uniform scoring system applicable to LRTI in a wide range of tissue sites.[23] It has been validated for scoring the severity of LRTI in the pelvis and has been shown to correlate well with other scales for bladder and bowel symptoms.[24–26]

The score is the sum of three numerical domains: *subjective* (asking about symptoms such as pain), *objective* (documenting signs such as bleeding or observations on endoscopy) and *management* (asking about medical management such as iron therapy). Each domain asks about several relevant symptoms, objective findings and interventions respectively. For each of these, there is a possible score between 1 (the least) to 4 (the worst) possible manifestation of that item. Any field for which there is no contribution (e.g., no pain) does not contribute to the score. It was not practical to include the objective domain as few patients underwent endoscopy at meaningful intervals before and after treatment.

Age, sex, type of malignancy, site and dose of radiation, comorbidities, length of HBOT, complications and reasons for any early termination of the course were recorded.

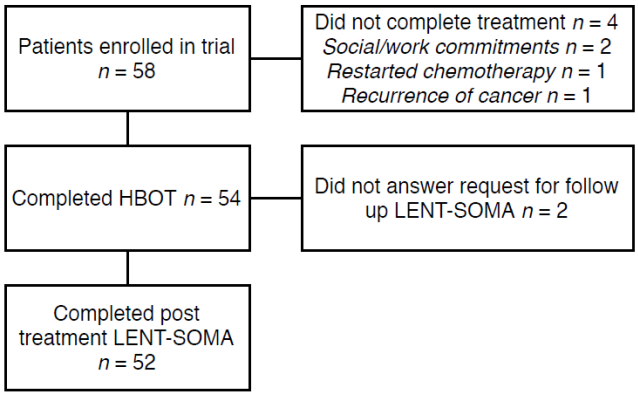
STATISTICAL ANALYSIS

Statistical analysis was performed using StatsDirect v3.1.11 (StatsDirect Ltd, Merseyside, UK). Parametric testing was deemed to be appropriate after visual inspection of the distribution of LENT-SOMA scores. Continuous data were assessed using paired *t*-tests and correlations were evaluated using simple linear regression and logistic regression as appropriate. A P-value < 0.05 was considered statistically significant. Data will be presented as mean and standard deviation or confidence interval, where appropriate. No formal sample size calculation was made on this opportunistic patient cohort.

Results

Fifty-eight patients were initially enrolled. Two withdrew because of work/social commitments. One had a recurrence of cancer and one had to restart chemotherapy. Two did not reply to our request for follow-up (see Figure 1). Data were therefore available for 52 patients. Of these,

Figure 1
PRISMA diagram demonstrating dropouts during the study



44 (84.6%) were male. The average age was 67.9 years (SD 10.1). The primary sites of LRTI were bladder (38 patients) bowel (13) and vulva (1). The primary malignancies were cancers of the prostate (41 patients), cervix (4), rectum (3), endometrium (2), bladder (1) and vulva (1).

Forty-eight of the 52 patients substantially completed the prescribed course of 30 treatments (27 to 31 sessions), while four completed a prescribed course of 20 treatments. Six of those completing an initial course of 30 treatments opted to have further sessions after the clinical evaluation of response (three had a further 20 sessions, two had eight and one had ten).

For the whole group, the mean LENT-SOMA score prior to HBOT was 11.7 (SD 5.3) while after completion of HBOT it was 8.1 (5.1). The maximum possible score was 44 in the rectum domain and 40 in the bladder domain. The mean reduction in score of 3.7 over the treatment period (95% CI 2.6 to 4.8) was statistically significant, $P < 0.001$. Subgroup analysis by affected site demonstrated a similar reduction for those with either proctitis or cystitis (mean reductions of 3.8 (95% CI 1.4 to 6.1); $P = 0.004$ and 3.7 (2.4 to 5.0); $P < 0.001$ respectively).

Simple linear regression demonstrated a statistically significant relationship between the severity of LRTI on presentation and the subsequent absolute reduction in LENT-SOMA scores ($P = 0.003$). There was no such clear relationship between the reduction in LENT-SOMA score and the number of HBOT sessions ($P = 0.71$), number of comorbidities ($P = 0.50$) or age ($P = 0.21$).

Thirteen of the 52 (25%) patients complained of ear pain during HBOT, of which two (3.8%) had clinically demonstrable barotrauma on examination. In both cases, this resolved and they were able to complete the course of treatment after a delay of two and three days respectively without further intervention. Four (7.7%) patients complained of myopia, which also resolved spontaneously. There were no other adverse events of therapy reported.

Discussion

The present findings are consistent with previous trials suggesting HBOT is an effective intervention for improving symptomatology in patients with LRTI. This was demonstrated by statistically significant improvements in scores using the LENT-SOMA grading system and our impression that the magnitude of these improvements is clinically important. A seemingly modest reduction of 3.7 can convey marked changes in a patient's quality of life. Examples from our series include a patient who had significant urinary frequency with recurrent admissions for clot-retention who became catheter-free and able to pass the night without having to urinate, and another who was housebound with social anxiety related to faecal incontinence is now able to socialise normally.

The most appropriate trial with which to compare the present proctitis scores is an RCT that demonstrated an improvement in LENT-SOMA scores after HBOT of 5.07 (from 12.55 to 7.48).[17] This is comparable to our mean improvement of 3.7 (from 11.7 to 8.1). We were unable to find any comparable trials using the LENT-SOMA system to assess symptoms of radiation cystitis. A recent RCT by Oscarsson et al. investigated HBOT treatment for radiation cystitis (as well as proctitis).[15] Their primary outcome was an improvement in expanded prostate cancer index composite (EPIC) scores, which have a large overlap with the subjective domain in the LENT-SOMA system. They observed an improvement in urinary symptoms of 22%, comparable to a 29.6% improvement in our group. We also demonstrated HBOT to be a safe intervention as evidenced by our low rate of side effects and absence of severe complications requiring early termination of the treatment course. HBOT has been shown to reduce the daily medical expenses for a patient with LRTI from AUD231.09 to AUD19.08.[13] The cost of a course of HBOT to treat LRTI at Prince of Wales Hospital has been estimated at AUD7153.[27] We believe this is a cost effective alternative to conventional treatments.

The present finding of a relationship between pre-treatment symptom severity and absolute improvement in LENT-SOMA scores makes clinical sense. The worst affected by any disease have the greatest potential for improvement. When we instead looked at the percentage improvement relative to the original score there was no trend, suggesting patients with worse symptoms did not improve disproportionately compared to those with mild disease. The absence of a relationship between number of treatments and change in LENT-SOMA scores probably reflects the fact that the majority received very close to 30 treatments (median 30, IQR one). It is also probable our study was not sufficiently powered to establish such a link. The six patients who had a further course of HBOT (an extra planned

10–20 treatments) had a smaller improvement in scores (1.5 vs. 3.9 for 'non-extenders') after completion of all treatments. Although this was not statistically significant it may represent a cohort of poor responders to HBOT.

THE LENT-SOMA SCORING SYSTEM

When the LENT-SOMA scoring system was released in 1995 the authors recommended taking the sum of all individual item scores and dividing by the number of items for which there was a score recorded, to give the overall severity score. Initial observations suggested this could lead to a misleadingly low overall score in a patient who had a high score in only one domain with low scores in all others.[28] It has become common practice over the years to report the sum of raw scores from each domain, as we have done in this report. This does not allow for comparison between different tissue types, as was the original aim of the system, but we feel it is a better representation of the impact of radiation injury on the individual.

Any system evaluating the side effects of a therapy must find a balance between high sensitivity and specificity for the diagnosis (e.g., mucosal changes on cystoscopy/sigmoidoscopy) and a representation of the impact on the patient (e.g., quality of life or functional assessments).[11] The four domains in the LENT-SOMA tables (subjective, objective, measured, analytic) was an attempt to strike this balance and it has been shown to have correlation with quality of life (QoL) scores.[29] Interestingly, several patients in the present cohort reported significant improvements during an informal discussion of QoL despite little improvement in their LENT-SOMA score and others reported the reverse.

Some studies have used cut-offs (e.g., an improvement of two points) as the minimum improvement likely to be important to an individual patient. Instead, we simply reported the mean changes in score, along with statistical significance testing of before and after scores. While any such assessment is very subjective, it is inferred from the observed changes over the course of treatment that many patients are improved in a clinically meaningful way. This investigation has prompted inclusion of a brief QoL assessment at first consultation, at treatment completion and at four week follow-up.

There were several other practical issues with the scoring system. Firstly, the scoring terminology was not very clear or intuitive, i.e., using criteria such as 'occasional' or 'intermittent' rather than clearly defined frequencies. Secondly, the inclusion of double criteria led to room for interpretation, i.e., dysuria could be 'occasional and minimal' (Grade 1) or 'persistent and intense' (Grade 3), but what if it was occasional yet intense? We feel in part this accounts for our anecdotal observations of inter-interpreter variability when different doctors scored symptoms in the same patient.

The objective and analytic domains in the LENT-SOMA tables were also sources of difficulty. Many patients had not had a recent cystoscopy/sigmoidoscopy or the results were very difficult to track down. As we re-assessed the patients shortly after they had completed treatment there was little opportunity for them to be re-evaluated objectively (on endoscopy).

Over the course of writing this paper, we have been introduced to an adaptation of the original LENT-SOMA tables that solve many of the above problems. For example, the separation of the frequency and severity of a symptom into two separate scores and the replacement of vague criteria such as 'occasional' with 'monthly'. In addition, the questionnaire has been divided into two sections. Subjective/management criteria are filled out as much as possible by the patient, removing interpreter bias. The clinician then fills out a questionnaire regarding objective findings (e.g., cystoscopic) where available. These are recommended for future use.[30]

LIMITATIONS

Aside from the difficulties with the LENT-SOMA scoring system, there were other issues requiring acknowledgement. The LENT-SOMA assessments were made by physicians involved in patient care and there was no comparator group with which to draw a comparison; either may bias the result favourably. As such, regression to the mean or a participation effect unrelated to any actual pathophysiological therapeutic benefit of HBOT cannot be ruled out. It is widely accepted that the 'ritual' of regular daily exposure over six to eight weeks to the chamber environment, supportive staff and one's fellow patients may make HBOT a powerful placebo procedure.[31] This effect may have been demonstrated in the Clarke et al. RCT, where 63% of the control group reported some response to sham treatment.[17]

Unfortunately having a control group is both technically challenging due to the nature of the treatment and highly consumptive of resources. Inevitably, in a busy service it means denying or delaying patients with accepted indications because of the fixed capacity of the chamber and attendant staff. A further ethical consideration surrounds the denial of what has become a routine accepted treatment to the putative control group. While we have demonstrated the effect we could anticipate in the active arm of a blinded, sham-controlled future study, we do not at this time have plans for a future controlled study.

A further limitation was the limited follow-up period of one month after treatment completion. This has two implications. Firstly, it has been shown that patients continue to improve for several months after HBOT, and there is potential to miss some improvements that manifested after follow-up.[17] Secondly, it is not possible to comment on whether or not any improvement in symptoms will have a lasting effect.

Conclusions

A review of the treatment outcome for LRTI patients in our unit suggests these patients are positively impacted by HBOT and this is consistent with most previous reports. There continues to be a good case for high quality, blinded and randomised trials, possibly in a group of patients for whom HBOT is not currently available due to geographical or regulatory constraints.

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Thermal balance of spinal cord injured divers during cold water diving: A case control study

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Scuba diving; Visual analogue scale; Disability; Diving; Disabled diver; Hypothermia

Abstract
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Introduction: This study compared the thermal balance of spinal cord injured (SCI) divers and able-bodied (AB) divers during recreational cold-water dives.
Methods: Ten divers (5 AB, 5 SCI) in matched pairs dived in a shallow lake (temperature 6°C) for 30 to 36 min wearing 5 mm 'Long John' neoprene wetsuits. A gastrointestinal temperature radio pill recorded gastro-intestinal temperature (T_{gi}) prior to, immediately after and at 5, 10, 15, 30, 60, 120 min post-dive. Subjective ratings of temperature perception were recorded concomitantly using a visual analogue scale (VAS).
Results: No difference between SCI and AB divers in T_{gi} before the dive was observed ($P = 0.85$). After the dive, SCI divers cooled significantly more than AB at all measured time intervals ($P < 0.001$). Post dive, the mean maximum fall in T_{gi} during the recovery phase in SCI divers was 0.85°C (SD 0.20) and in the AB group was 0.48°C (0.48). In addition, there was greater individual variation in SCI divers compared to AB divers. There were no statistically significant differences in temperature perception between the groups either before or at any time after the dives.
Conclusions: In contrast to AB divers, divers with SCI were unable to maintain T_{gi} during short shallow dives in 6°C water and their temperatures fell further post-dive. The reduction in T_{gi} was not reflected in the subjective ratings of temperature perception by the SCI divers. The study was too small to assess how the level of spinal injury influenced thermal balance.

Introduction

The annual incidence of traumatic spinal cord injury (SCI) in developed countries is estimated at 15 to 40 cases per million people.[1] The American Disabilities Act of 1990 and the British Disability Discrimination Act of 1995 have formed the basis for greater integration of SCI individuals into society. Today, individuals with spinal cord injury participate in sports and some even compete in high-level organised events such as the Summer and Winter Paralympic Games.[2] Recreational scuba diving is becoming a popular sport among SCI individuals. According to a survey of scuba diving for disabled divers, over 50% of British dive clubs had been involved in the training of disabled divers between 1998 and 2000.[3] Specialised training for SCI recreational divers is now available worldwide through national or regional associations for handicapped divers, as well as dive education organisations.[4]

SCI divers participate in both warm- and cold-water diving. One of the main issues for divers is thermal protection since the ratio of heat conductivity of water to air is approximately 24:1.[5] Although SCI individuals participate in winter paralympic sports (alpine skiing, ice sledge hockey, Nordic skiing, wheelchair curling, biathlon), the heat loss during these activities conducted in cold air is substantially less than that experienced during immersion in cold water.

Autonomic and behavioural responses are involved in the maintenance of deep body temperature in humans within a narrow range despite large variations in ambient conditions and activity level. Central foci initiate appropriate effector mechanisms in response to thermal afferent information from skin and core regions. Injury to the spinal cord abolishes thermal afferent information from regions innervated by nerves emerging from the spinal cord below the injury. Thus, although hypothalamic and cortical regions involved in autonomic and behavioural temperature regulation are not affected, their actions are limited to the regions above the spinal cord lesion. Consequently, the ability of SCI individuals to regulate body temperature and to sense cold is impaired.[6,7] The severity of impairment is directly related to the injury level and completeness of the lesion. Deep or central temperature receptors sensitive to cold can initiate

shivering above the level of the SCI.[6] Individuals with SCI show lower core temperature after cold exposure in comparison to able-bodied controls.[8,9] Central temperature mechanisms remain unaffected.[10]

As hypothermia is one of the risks in diving,[11] suitable thermal protection is part of the personal protective equipment of any diver. While in tropical waters neoprene rubber suits are the primary choice, drysuits are usually recommended for cold-water diving. However, in drysuit diving, gas must be delivered and released from the suit to maintain a constant volume during diving and to minimise the squeeze on descent or uncontrolled ascent due to excessive air. In general, drysuit diving requires specific training to control buoyancy. SCI divers need to use their arms to prevent uncontrolled rapid ascent and at the same time operate the suit and buoyancy compensator inflation and exhaust valves. The compensating air is usually taken from the breathing cylinder, decreasing diving time. Most dry suits have baggy trousers to allow passage of the feet to the boots. A large volume of air can be trapped in the legs, leading to body inversion. This can occur much faster in SCI divers who may have difficulty maintaining horizontal trim.[12] All the above might explain why SCI divers usually only use neoprene rubber wetsuits. The drawback of a wetsuit in contrast to a drysuit is that it does not offer the same level of thermal protection.

Several studies have investigated the issue of hyperthermia in SCI individuals during fever[13] and during activities conducted in warm air environments,[14,15] but there is only anecdotal information regarding the risk of cold injury among SCI individuals conducting activities in cold environments. During activities in cold air, the risk of local freezing and non-freezing cold injury to regions below the lesion and of hypothermia is most likely higher among SCI compared to able-bodied individuals. In contrast to activities conducted in air, where exposure time is essentially unlimited, during diving the exposure to the high heat loss environment is limited by the capacity of the breathing air supply.

In cold water diving the main problem is hypothermia.[16] Therefore, SCI divers would benefit from recommendations regarding planning such dives. The present study was conducted to compare gastrointestinal temperature as a valid surrogate marker of core temperature following a shallow dive in cold water in SCI and non-SCI divers. We hypothesised that SCI divers exposed to the same environmental conditions would demonstrate a larger reduction in core temperature than their able-bodied dive partners.

Methods

The study was approved by the National Committee for Medical Ethics at the Ministry of Health of the Republic of Slovenia. Written informed consent was obtained and it was made clear to all the participants that they could terminate the dive at any point. All volunteers were familiar with the methods and the diving protocol.

STUDY POPULATION

Five SCI and five able-bodied (AB) divers participated. All were experienced divers (between 100 and 300 logged diving hours), with previous experience in cold-water diving. All SCI divers were at least five years post-injury with levels of injury of T6 (two divers), T9 (one diver) and T12 (two divers), ASIA score A[17] and had no history of cardiovascular disease or diabetes. Paraplegia was the result of injuries sustained in either traffic or sports accidents, and in one case from a war injury. All five SCI divers received their diver training and certification after their spinal cord injury. Diving experience in the SCI group varied from three to 11 years and around 20 h per year of diving. Divers were matched by body mass index (BMI) and sex. Body surface area (BSA):[18]

$$BSA\ (m^2) = (height\ [cm] \times weight\ [kg]) / 3{,}600 \quad (1)$$

Lean body weight (LBW),[19] adult men:

$$LBW\ (kg) = (9{,}270 \times total\ body\ weight\ [kg]) / (6{,}680 + (216 \times BMI\ [kg \cdot m^{-2}])) \quad (2)$$

adult women:

$$LBW\ (kg) = (9{,}270 \times total\ body\ weight\ [kg]) / (8{,}780 + (244 \times BMI\ [kg \cdot m^{-2}])) \quad (3)$$

were calculated. Demographic data for all participants are summarised in Table 1.

DIVING PROTOCOL

The Adriatic Chapter of the International Association of Handicapped Divers (IAHD) organised a single recreational cold-water (6°C) dive at Gruener Lake in Tragoess, Austria, which is a shallow (maximum depth 5.4 metres' fresh water [mfw]) alpine lake at an altitude of 779 m. Appropriate altitude and fresh water considerations were included in the planning of the dives. Dives were limited to the maximum depth of the lake and a maximum of 35 min duration, and were conducted in pairs, each pair consisting of a SCI and a BMI-matched AB diver. SCI divers were instructed to start leisurely swimming after a 2 min descent and safety check and AB divers to adjust the swim pace accordingly. The pair swam for 15 min to the centre of the lake, turned around and swam back for 15 min. Table 2 lists pair-matched diving profiles.

All divers used two-piece 5-mm neoprene wetsuits, comprising 'Long John' trousers and long-sleeved jackets. The overlapping 'Long Johns' and jacket provided a 10-mm insulative layer of neoprene for the torso. The SCI divers used tailored suits (Kanoko® superstretch/ Pile® thermic inside, zipper to face, ELIOS, Italy), the AB divers used their own 5 mm thick neoprene wet suits, which were not standardised for the study. The dive profiles (depth and duration) and water temperature were recorded with dive computers worn by each diver (D-series, Suunto, Finland). The divers entered the water from the shore. The first post-

Table 1
Demographic data for spinal cord injured (SCI) and able bodied (AB) divers; BMI = body mass index; BSA = body surface area; LBW = lean body weight

Divers	Sex	Age	Height (cm)	Weight (kg)	BMI ($kg \cdot m^{-2}$)	BSA[17] (m^2)	BSA/weight ($m^2 \cdot kg^{-1}$)	LBW[18] (kg)	Injury level
SCI1	M	45	172	68	23.0	1.80	0.025	55	T9
SCI2	M	39	170	80	27.7	1.94	0.024	60	T6
SCI3	M	44	188	85	24.0	2.11	0.025	67	T12
SCI4	W	41	158	52	20.8	1.51	0.030	41	T6
SCI5	M	40	185	88	25.7	2.13	0.024	68	T12
AB1	M	23	180	76	23.5	1.95	0.026	61	/
AB2	M	51	180	89	27.5	2.11	0.024	67	/
AB3	M	60	175	76	24.8	1.92	0.025	59	/
AB4	W	35	168	60	21.3	1.67	0.028	47	/
AB5	M	48	173	78	26.1	1.94	0.025	60	/

Table 2
Pair-matched diving profiles for SCI and AB divers

Diver pair	Max. depth (m)	Average depth (m)	Duration (min)
SCI1	4	2.1	35
AB1	4.5	2.8	36
SCI2	5.3	2.8	36
AB2	5.4	3.3	35
SCI3	4.1	2.3	33
AB3	4.6	2.8	33
SCI4	5	2.9	30
AB4	5.2	3	30
SCI5	4.2	2.5	33
AB5	4.5	2.7	32
Mean (SD)	4.6 (0.51)	2.7 (0.34)	33.3 (2.2)

dive gastrointestinal temperature (T_{gi}) measurements were performed immediately after the divers reached the shore. Non-divers helped the SCI and AB divers with the diving equipment. SCI divers had help with the wheelchair transfer and were pushed from the shore to the observational terrace (50 m distant). All divers changed into dry clothes (long trousers, T-shirt, fleece jacket, Windstopper jacket, cap) and shoes after the dive and covered themselves with a provided fleece blanket. Both groups remained seated outdoors at a table for 120 min, chatting. Weather was partly cloudy, air temperature 17°C with light wind (1 $m \cdot s^{-1}$).

DEEP BODY TEMPERATURE ASSESSMENT

T_{gi} was used as a marker of core temperature using an ingestible radio pill (CorTemp® Temperature Sensor 262K15VSOHCO38075, HQInc, Palmetto, USA), which relayed the temperature information to an external receiver/recorder (CorTemp® Data Recorder 262K w/HR HT 130042, HQInc, Palmetto, USA). These pills contain temperature recording and radio frequency emitting electronic circuits, powered by a small battery. Because there are no data about the pressure resistance of the capsules, a laboratory pressure test was performed at Seabear Diving Technology, Graz, Austria. Capsules were tested 24 hours before the dive whilst recording at approximately 1 mPa in a pressure chamber filled with water. No mechanical deformities of pills or erroneous temperature measurements were detected.

The radio pill was ingested with a granola bar (Frutabela, Fructal, Slovenia) one hour before the dive. Variable gut motility among the subjects may cause the T_{gi} temperature to correspond to different regions of the gastrointestinal (GI) tract. According to normal gastric emptying (up to 2 h)[20] and small bowel transit time (up to 2 h in more than 80% population),[21] the radio pill was most likely positioned in the jejunum during measurements. The location of a GI radio pill may vary among subjects and it has been shown in one individual that the detected temperature changed with the GI location.[22] In contrast, no difference in measurements were seen when a sensor was ingested 24 h or 40 min before the first reading.[23] Since the gastrointestinal radio pill temperature system is designed for use in air, the radio receiving unit which receives and stores the amplitude

Figure 1
Individual T_{gi} before the dive (predive), immediately after the dive (pd 0) and maximal post-dive fall (maxdrop) for SCI divers (green) and AB divers (blue)

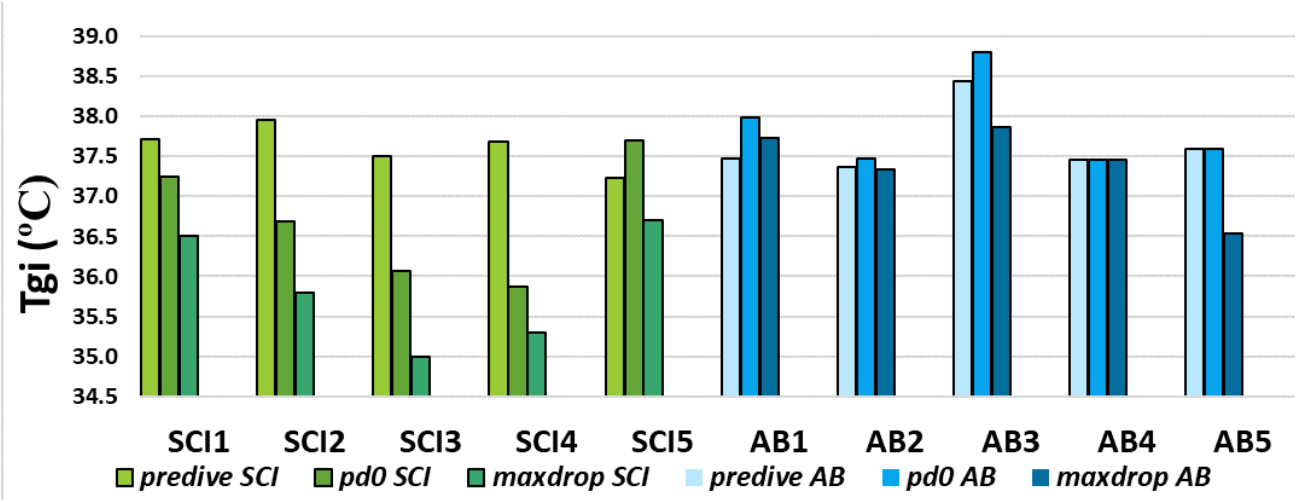


Table 3
Mean (SD) T_{gi} for SCI and AB divers pre dive, immediately post dive and at 5, 10, 15, 30, 60 and 120 min post-dive; the lowest temperature for each SCI diver is in bold; PD = post dive

Tgi (°C)	Pre-dive	PD 0	PD 5	PD 10	PD 15	PD 30	PD 60	PD 120
SCI1	37.7	37.2	37.1	37.1	37.1	37.0	37.4	**36.5**
SCI2	38.0	36.7	36.4	36.2	36.0	36.0	**35.8**	36.7
SCI3	37.5	36.1	35.6	35.3	35.0	**35.0**	35.6	36.5
SCI4	37.7	35.9	35.9	35.6	35.4	**35.3**	35.9	36.7
SCI5	37.2	37.7	37.5	37.3	37.1	36.9	36.8	**36.7**
Mean (SD)	37.6 (0.3)	36.7 (0.8)	36.5 (0.8)	36.3 (0.9)	36.1 (0.9)	36.0 (0.9)	36.3 (0.8)	36.6 (0.1)
Tgi (°C)	**Pre-dive**	**PD 0**	**PD 5**	**PD 10**	**PD 15**	**PD 30**	**PD 60**	**PD 120**
AB1	37.5	37.9	37.8	37.7	37.7	37.8	38.0	37.9
AB2	37.4	37.5	37.4	37.4	37.4	37.4	37.4	37.3
AB3	38.4	38.8	38.5	38.6	38.4	38.6	38.0	37.8
AB4	37.4	37.5	37.4	37.5	37.6	37.6	37.7	37.5
AB5	37.6	37.6	37.4	37.3	37.3	37.4	36.9	36.5
Mean (SD)	37.7 (0.4)	37.9 (0.6)	37.7 (0.5)	37.7 (0.5)	37.7 (0.4)	37.8 (0.5)	37.6 (0.5)	37.4 (0.6)
***P*-value**	0.85	0.027	0.017	0.015	0.010	0.006	0.012	0.014

modulated signal from the pill only works in air. T_{gi} were recorded prior to, directly after and at minutes 5, 10, 15, 30, 60 and 120 after the dive. During this post-dive period, the subjects were requested not to eat or drink.

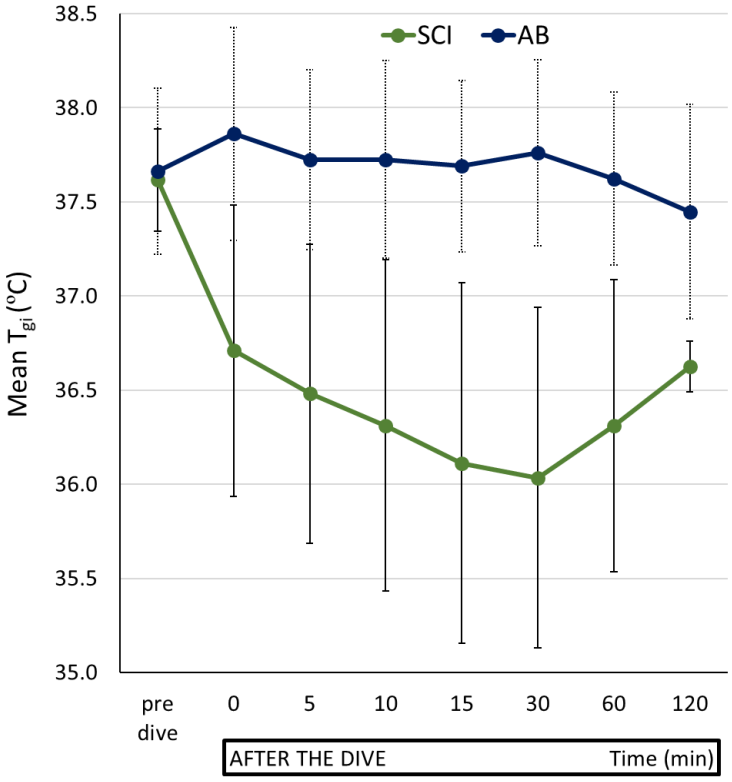
TEMPERATURE PERCEPTION

Divers were requested to provide overall ratings of temperature perception on a visual analogue scale (VAS) comprising a 10 cm horizontal line with 0 representing no perception of cold and 10 the perception of severe cold. The subjects rated their sensation of cold in parallel to the core temperature recordings pre-dive and at 0, 5, 10, 15, 30 and 60-min post-dive. In addition, it was noted if a subject was shivering.

STATISTICAL ANALYSES

After test for normality (Shapiro-Wilk test) a two-way mixed ANOVA statistical model, calculated with IBM SPSS® 25.0 software, was used to assess the effect of time, group and interaction between time and group on T_{gi}. Paired sample *t*-tests were used to establish whether the mean T_{gi} recorded at specific times were different between the groups; data are presented as mean (SD). It was not possible to assess changes in T_{gi} against injury level in the SCI divers because of the small numbers. Subjective ratings of temperature perception of the SCI and AB divers were evaluated with Mann-Whitney U tests (M-WU); these data being presented as median and inter-quartile range (IQR). The alpha level of significance was set at 0.05.

Figure 2
Mean (SD) T_{gi} in five SCI (green) and five AB (blue) divers before and at different times out to 120 min post-dive after a 6°C dive



Results

All divers completed the dives according to the dive plan. There were no untoward events either during or immediately after the dives and all planned temperature measurements in all divers were documented successfully.

DEEP BODY TEMPERATURE

Individual pre-, immediate post-dive and lowest post-dive T_{gi} are shown in Figure 1. There was no difference in pre-dive T_{gi} between the groups (SCI: 37.6°C (SD 0.2); AB: 37.7°C (0.4); $P = 0.85$) but a significant difference post-dive (SCI: 36.7°C (0.77); AB: 37.9°C (0.56); $P = 0.02$). Table 3 lists all measured T_{gi}.

Post-dive, the mean maximum fall in the recovery phase in T_{gi} in SCI divers was 0.85°C (0.2) and in the AB group 0.48°C (0.48). In addition, there was greater individual variation in SCI divers (mean max difference (SD): 1.75°C (0.6)) compared to AB divers (0.75°C (0.4)) in the recovery phase. The dive did not significantly alter the deep body temperature of the AB group (before: 37.7°C (0.4); after: 37.4°C (0.6); $P = 0.47$).

ANOVA revealed that there were significant main effects on T_{gi} for time ($F[7.56] = 9.789$; $P < 0.001$), group ($F[1.8] = 11.61$; $P = 0.009$) and interaction between time and group ($F[7.56] = 5.47$; $P < 0.001$). Thus, there were statistically significant differences in T_{gi} between the SCI and AB divers measured post-dive. Figure 2 shows the mean T_{gi} for both groups through the measured time. In the AB group, mean T_{gi} after 120 min is only 0.3°C less than the pre-dive mean, whereas in the SCI group, it is 1.0°C lower (pre-dive: 37.6°C (0.27), 120 min post-dive: 36.6°C (0.14)).

COLD SENSATION

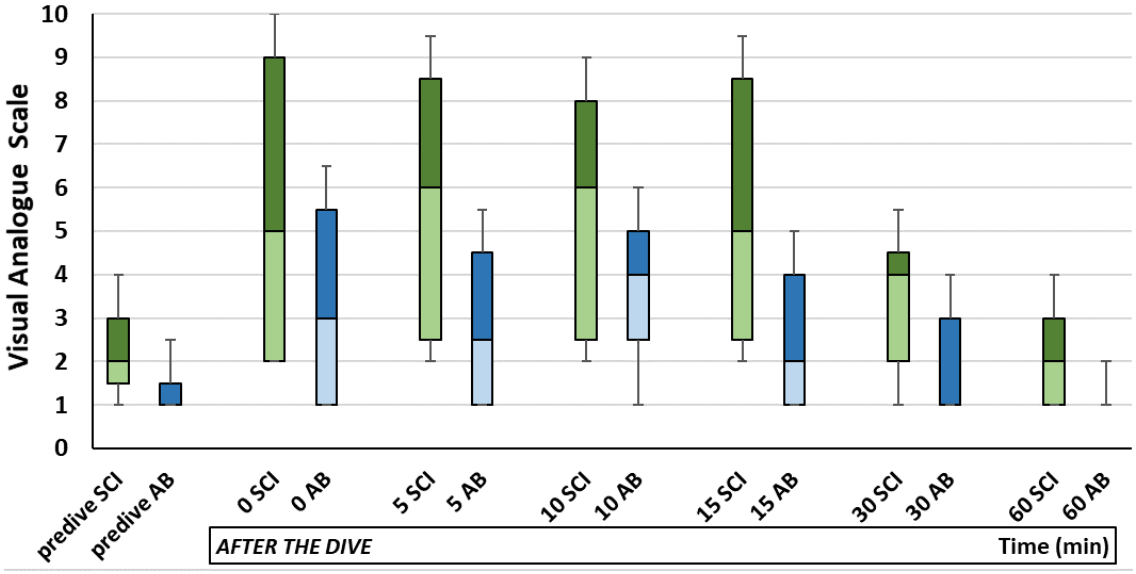
There were no statistically significant differences in the perception of temperature between the AB and SCI divers either before, immediately after or at any time post-dive (Figure 3). SCI divers started to report increasing perceptions of cold immediately after to 15 min post-dive. Thereafter, despite a continued reduction in T_{gi}, the SCI divers did not perceive any further increases in cold perception after 15 min post-dive (Figure 3). Only SCI divers 3 and 4 (one T6 and the other T12 injury levels) exhibited shivering around the torso and sternocleidomastoid muscles lasting about 25 min post dive.

Discussion

The principal finding of the present study is that SCI divers experienced a significant but variable fall in T_{gi} during and after a 30–35-min, shallow, cold-water (6°C) dive. In contrast, AB divers exhibited uniformly only a minimal change in deep body temperature during the dive and no 'afterdrop' post dive.

Figure 3

Assessment of cold with a visual analogue scale (0 – no cold and 10 – severe cold) in five SCI divers (green) and five AB divers (blue) prior to the dive and at 0, 5, 10, 15, 30 and 60 min post-dive (median [IQR] as box and whisker plots)



Since a given dive pair, comprising an AB and SCI diver, conducted the dive together, the activity of the two divers should be similar. Before the dive, all divers were instructed to swim leisurely. However, the pattern of swimming between groups was different. For SCI divers, propulsion is provided by using the arms and non-SCI divers the legs. Core temperature falls significantly more when exercise is performed by the arms when compared with the legs in cold water.[24] The larger surface area-to-body mass ratio will also contribute to this accelerated heat loss. According to the values for surface-to-mass ratio, the ratio for arms is almost twice that for legs.[25] SCI have a higher convective coefficient[26] and, therefore, higher absolute heat flux to cold water. On the other hand, the muscle temperature depends on exercise intensity, which might be higher in the SCI group. Also, blood flow would be greater in higher intensity exercise, so a greater proportion of heat is transferred to the core. It can be postulated that the SCI group were exercising at a higher intensity than the AB divers whilst swimming side-by-side at the same pace and, therefore, had a higher metabolic rate. However, the peripheral location of the working, perfused muscles indicates that most of this heat would be lost to the cold water. It is assumed that SCI divers have greater conductive and convective heat transfer at the neoprene-water interface than AB divers.

It is also likely that there are differences in the pattern and magnitude of heat loss from different body regions. Despite the thermal insulation provided by the neoprene suit, the skin temperature during the dive most likely decreased to a level where vasoconstriction was initiated to retain heat. Whereas, for the regions above the level of the spinal cord injury the level of vasoconstriction was most likely similar to or possibly greater than that in the AB divers, regulation of perfusion of regions below the injury would be substantially impaired in the SCI divers.[27] The difference in perfusion of peripheral regions within the two diver groups using skin temperatures was not monitored in the present study. Nevertheless, the greater cooling of the core region of the SCI divers can be attributed to impairment in the regulation of peripheral perfusion and, thus, impairment of the heat retention response.

The 'afterdrop' in core temperature is a characteristic response observed during rewarming of individuals after exposure to a high heat loss environment inducing core temperature cooling.[28] The aetiology of the core temperature afterdrop is suggested to be the thermal inertia in heating the cooled peripheral regions.[29,30] The SCI group experienced greater continued cooling during the post-dive 120 min observation time. The heat production created as a by-product from exercise stopped after the dive. Divers stayed in an air environment in which further cooling via convection (light wind (1 $m \cdot s^{-1}$)) and evaporation (before changing into dry clothes) can occur. SCI divers needed longer (up to 10 min) to change and, therefore, were more exposed to heat loss. The absence of a significant T_{gi} after-drop in the AB divers suggests that they remained in or close to thermal balance during and after the dive. However, the difference in afterdrop was so prominent that longer changing time can only contribute to cooling without being a main reason for it.

COLD PERCEPTION

The perception of body temperature was similar in the two groups despite the lower T_{gi} in the SCI group. A rating of temperature perception is a result of cortical integration of thermo-afferent information. The lack of sensory information from the regions below the injury would not appear to contribute significantly to the overall perception of

cold in the SCI divers. Consequently, within the framework of the dives conducted and the cold exposure experienced by the SCI divers, it would appear that their behavioral regulation of body temperature would be appropriate but is altered, which might present safety issues.

SHIVERING

Despite the decrease in core temperature in the SCI divers during the dive, shivering was observed in only two of them post dive. Shivering in humans with an intact spinal cord is initiated by thermo-afferent information from temperature sensors in the skin, as well as direct thermal stimulation (temperature of blood perfusing the region) of hypothalamic temperature neurons. Whereas, the former is probably substantially reduced in SCI, it is the latter that provides the stimulus for shivering in muscles with intact innervation.[31] Previous studies suggest that the muscles of the lower extremities are not activated during the initiation of shivering; activation of the shivering proceeds caudally starting with the mastoid and sternocleidomastoids muscles.[32,33] The impaired activation of the leg muscles in SCI divers is likely less important than the impairment of heat retention in this situation. Because both groups remained seated during the observation time, the activity level of the divers post-dive did not account for the slower rewarming in the SCI divers.

LIMITATIONS

The small number of divers is the main limitation of the study. However, it is difficult to find SCI divers with similar levels of injury who also perform cold-water dives. This is likely why there have been no previous studies of SCI vs. AB divers. All the SCI divers were classified as ASIA score A (no sensory or motor function is preserved in the sacral segments S4–S5) and had an injury at the thoracic level with the complete absence of motor function below the level of injury. The variability of the cooling pattern could be the result of the different levels of thoracic injury (T6 to T12), but the small numbers prevent any worthwhile analysis of this relationship.

Matching divers by BMI could be a possible limitation of the study. BMI does not reflect location or amount of body fat but it is internationally recognised as a marker of obesity and adiposity.[34,35] A higher BMI, whether reflecting greater body fat, increased muscle mass or simply overall size, is associated with a slower drop in core temperature during cold-water swimming.[36,37] Therefore, it was assumed that BMI matching was appropriate for the given conditions. Also, the two groups were shown to be matched for the body surface area-to-mass ratio, a factor that contributes to heat loss (Table 1).[38]

Using gastrointestinal temperature as a marker of core temperature could be a limitation because the location of the radio pills is dependent upon gastrointestinal motility. Despite that, the radio pill is widely accepted and used in field-based exercise studies as an indicator of core temperature.[39] Finally, for real-time detailed core temperature changes, continuous temperature monitoring during the dive is essential. Unfortunately, no commercial measurement system has been available until recently.

Conclusions

In a shallow dive in 6°C water, the deep body temperature of SCI divers fell significantly, whereas in AB divers it remained stable. However, the decrease in T_{gi} approached levels considered hypothermic (35°C) in only one (with a T12 injury) of the five SCI divers. The subjective perception of cold by SCI divers did not reflect the reduction in T_{gi} observed. Further studies are needed with larger numbers, various water temperatures, dive times and injury levels, preferably in controlled laboratory conditions and with in-dive monitoring.

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Review article

Monoplace chamber treatment of decompression illness: Review and commentary

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Key words
Cerebral arterial gas embolism; Decompression sickness; Diving medicine; Patient monitoring; Recompression; Pressure chambers

Abstract

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This paper summarises the history and capabilities of monoplace chambers in treatment of decompression illness (DCI); both in support of diving operations and in the hospital setting. In the field, monoplace hyperbaric chambers provide victims of DCI immediate access to recompression in settings where traditional multiplace chambers are not available. Alternatively, they may facilitate pressurised transport to a multiplace chamber for continued management. Recently, collapsible lightweight versions have improved suitability for field deployment aboard small vessels in remote settings, and for use by less technically capable military, occupational and civilian operators. The resulting elimination of treatment delays may prove lifesaving and central nervous system sparing, and avoid subsequent diving fitness disqualification. Monoplace chambers thus facilitate diving operations that would otherwise be difficult to condone on health and safety grounds. The 1960s saw the introduction of multiplace hyperbaric chambers into the hospital setting, as a number of non-diving conditions appeared to benefit from hyperbaric oxygen. This coincided with interest in hyperbaric oxygen as a solid tumour radiation sensitiser. Development of a novel acrylic-hulled single occupancy chamber enabled patients to undergo radiotherapy while pressurised within its oxygen atmosphere. Increasing numbers of health care facilities adopted this chamber type as a more economical, less complex alternative to the multiplace chamber. Incorporation of relevant biomedical technologies have allowed monoplace chambers to support increasingly complex patients in a safe, effective manner. Despite these advances, criticism of medical centre-based monoplace chamber treatment of DCI exists. This paper evaluates this controversy and presents relevant counter-arguments.

Introduction

Therapeutic recompression has long represented standard care for those suffering decompression illness (DCI). It was proposed in 1854,[1] then first employed in a systematic fashion during excavation of the Hudson River tunnel.[2,3] By the early twentieth century, an on-site recompression or hyperbaric chamber was increasingly considered essential support for compressed air operations.[4,5] Constructed of steel, it was large enough to accommodate several occupants. This afforded immediate and simultaneous treatment of multiple patients, given large 'at risk' populations employed within pressurised bridge caissons and mass transit tunnels. Connected to the chamber was an entry compartment, or air lock, to facilitate transfer of patients and support personnel into and from the main compartment while it remained pressurised. The chamber's principal therapeutic basis was that of Boyle's Law; namely, air pressure increases proportionally decrease the volume of gas emboli.

On-site recompression chambers were soon associated with naval and civilian diving operations. In contrast to referenced fixed location civil engineering projects, diving worksites involved fewer at-risk personnel, occasionally operated from water-borne platforms, and were increasingly remote. To support this form of compressed air work, traditional large permanently emplaced multiplace chamber design evolved to one that was smaller and relatively transportable. Subsequent use of aluminum alloy further decreased weight and enhanced transportability.

Growth in demand for underwater work, occasional space limitations, increasingly remote worksites and certain economic constraints led to the introduction of a single occupancy 'monoplace' chamber. Air remained the compression gas and operating pressures of early models equalled many of their multiplace counterparts.[6,7] In due course, oxygen, delivered by facemask, became an important therapeutic adjunct, as it did for the multiplace chamber.

Monoplace chamber support of diving operations commonly involves two distinct strategies. One is to effect treatment while the chamber remains at the dive site, thereby ensuring its continued availability during ongoing underwater activities. The alternative is a transportable system, although chamber designs are not mutually exclusive. In this second example, and upon initiation of on-site recompression, transfer of the pressurised diver to a regional multiplace chamber occurs by whatever expedited means planned or available. Upon arrival, the patient relocates to the multiplace chamber. Several factors dictate how this is accomplished. If the monoplace is equipped with a flange coupling compatible with the multiplace, transfer under pressure takes place by physical connection of the two chambers.[8] Once attached, compression of the multiplace chamber to equal monoplace pressure occurs, at which point multiplace inside attendants (IAs) open interconnecting hatches and assist the patient into the multiplace compartment for continued treatment. Monoplace chamber design tends to be narrower and lighter in order to accommodate this transfer method. In the absence of a physical connection capability, and size constraints permitting, support personnel place the monoplace into the unpressurized multiplace chamber.[9] The multiplace is then compressed to monoplace pressure and the patient relocated as before. Failing the ability to effect either option, one may elect to complete treatment in the monoplace or decompress it and promptly recompress the patient in the multiplace. Various clinical circumstances, operational constraints and environmental factors dictate which of these decisions would best apply.

In the 1980s a fabric-hulled compressed air monoplace chamber was introduced.[10] Exceptionally lightweight, it is readily transportable in carrying cases. Although it lacks the degree of pressurisation inherent in earlier monoplace designs, it has an oxygen delivery system so is capable of providing US Navy Treatment Table 6 (USN TT6); an essential DCI standard of care.[11] Such chambers presently support unique military needs,[12] civilian professional, marine science[13] and recreational diving communities,[14] and have proven an effective on-site option to reduce inherent treatment delays.[13,14]

There has been little criticism of monoplace chamber support of a wide range of diving activities and its transfer under pressure capability.[15–18] On the contrary, its increased acquisition in recent years appears testament to its perceived lifesaving and central nervous system-sparing potential, and avoidance of outcomes that result in career-ending diving medical disqualification. Monoplace chambers support diving operations that would otherwise be difficult or impossible to condone on health and safety grounds. This is certainly the case in remote settings where injured diver retrieval can be complex, lengthy, hazardous and expensive. Cocos Island, essentially a rocky outcrop off Costa Rica and popular for cage diving among great white sharks, is one example. It is only accessible by boat so injured divers must endure the 30-hour return trip to the mainland for care. Fixed wing aircraft cannot land and the island is well beyond helicopter range. 'Fast boat' recovery attempts are dangerous and may be thwarted by unpredictable weather conditions that far into the Pacific. The author was recently involved in a 28-hour retrieval of a diver from San Benitos Island, off Baja California, Mexico.

The 1960s saw the introduction of hospital-based multiplace hyperbaric chambers as several other conditions appeared to benefit from their use. This same period coincided with considerable interest in hyperbaric oxygen (HBO) as a solid tumour radio-sensitizer.[19,20] A newly designed acrylic-hulled monoplace chamber facilitated this novel approach; one that enabled patients to undergo radiotherapy while pressurised within its oxygen atmosphere.[21] Hospitals increasingly adopted this chamber type as a more economical and operationally less complex alternative to the multiplace chamber for provision of HBO treatment (HBOT). Over the ensuing years, several biomedical technologies have been developed or adapted to allow monoplace chamber support of increasingly complex cases.[22–28] Today, it affords safe and effective therapy across the full range of patient states, from ambulatory cases to those critically ill and dependent upon mechanical ventilation.

In contrast to its on-site support role, the hospital based monoplace chamber enjoys a practice setting readily supported by advanced diagnostic capabilities, complementary therapies and multidisciplinary expertise. Given this optimal clinical environment, given that the monoplace meets US Navy minimum hardware capabilities for recompression therapy,[29] given that it has been successfully employed over several decades,[30–34] and given that it is considered appropriate for treatment of DCI in authoritative reviews,[11,35] it is surprising that there has been criticism of its use for this purpose in the hospital setting.[32,36,37]

Monoplace chamber perceived limitations addressed

Criticisms of the monoplace chamber in general and its use to treat DCI in particular can be summarised as patient isolation, lack of an air break delivery system, inability to support critically ill patients, limited pressurisation capability, heightened fire risk, impact of decompression on an existing pneumothorax and management of excreta. What follows is a review of the monoplace chamber's current capabilities in the context of these criticisms. It should enlighten those not familiar with, or have a dated understanding of the scope of the monoplace hyperbaric delivery system's technical arrangements and operational standards, the sum of which should serve to dispel much negative dogmatism.

PATIENT ISOLATION

The principal advantage of the multiplace chamber is the ability to accompany patients during treatment. For the

injured diver, this affords objective assessment of treatment response to guide subsequent management decisions. Typically today, this will centre on whether to extend the USN TT6. Historically, treatment decisions based upon clinical response were more impactful given a multitude of recompression approaches, therapeutic gas choices, saturation storage options and subsequent decompression table selection.

Should the diver exit the multiplace chamber incompletely recovered, follow-up treatments using USN TT5, 6, or 9, are commonly rendered until resolution, or no sustained improvement following two consecutive treatments.[38] There is no credible evidence to indicate that this 'simplified' approach is any less effective than deeper, longer and operationally more complex options, most of which have since been abandoned.

Appropriately equipped and knowledgably staffed monoplace chambers readily support a USN TT 6. The key 'monoplace' decision is how to address the issue of medically necessary extensions in the absence of objective guidance. For more than three decades, the hyperbaric medicine programme at the author's institution has successfully employed one such approach. It involves table extension determination at the beginning rather than the end of the third oxygen breathing cycle at 284 kPa (2.8 atmospheres absolute [atm abs]). If the patient reports being asymptomatic at this time point, the USN TT6 is continued without extension(s). Should a residual undetected deficit exist, it has the therapeutic benefit of oxygen for 20 more minutes at 284 kPa and several additional hours during delivery of the remainder of the table. Should a post-treatment assessment determine incomplete relief, serial follow-up treatments occur consistent with multiplace operations. If the patient remains symptomatic at the beginning of the third oxygen cycle at 284 kPa, extension(s) occurs at that pressure. Assessment also takes place at 192 kPa (1.9 atm abs), in determining any additional extensions. Employment of all four extensions resulting in an 8-hour chamber exposure has been uneventfully administered.

Evaluating the patient's oxygen delivery system for good fit, and its management should central nervous system (CNS) oxygen toxicity develop is another important multiplace chamber inside attendant function. During monoplace operations, the patient breathes directly from the chamber's oxygen atmosphere so the potential multiplace chamber mismatch between chamber pressure and oxygen pressure when using an oral nasal mask does not exist.[39,40] As patients are readily visible from the monoplace control panel, any suggestion of oxygen intolerance prompts instruction to the patient to begin breathing air. Air delivery systems for the full range of patient states and are discussed in the next section.

If the chamber operator misses premonitory events and/or seizure occurs, chamber pressure remains unchanged until seizure activity has ceased. Even though the patient remains in a pressurized oxygen atmosphere, seizure latency is similar to patients immediately converted to air breathing, as would occur in a multiplace chamber. This has been a consistent observation during many decades of monoplace operations; namely, it is not necessary to interrupt oxygen breathing to halt an oxygen-induced seizure. Some may find this counter-intuitive as in their experience removal of the multiplace patient oxygen delivery system was associated with seizure cessation. The seizure would have ceased if mask/hood oxygen breathing continued, as oxygen is metabolized to sub toxic levels. For a patient to remain exposed to hyperbaric oxygen following a seizure, however, invites a second episode. Therefore, once all seizure activity has ended and the patient appears to be ventilating spontaneously, the monoplace chamber is decompressed.

Should a sudden change in patient status take precedence, decompression of the monoplace chamber from its highest operating pressure will take no more than approximately 120 seconds. This represents a distinct advantage over multiplace operations in terms of time of access to advanced care and related inside attendant decompression risk. Several cases of inside attendant DCI secondary to such multiplace chamber 'aborts' were included in a recent attendant DCI review.[41] One involved treatment of a complex CAGE patient who developed ventricular fibrillation during the latter stages of a USN TT6A. Defibrillation was urgently required and considered inherently dangerous under hyperbaric conditions,[42] and chambers 'must be surfaced to perform defibrillation' in accordance with US Navy policy.[38] Subsequent accelerated decompression resulted in a significant inside attendant decompression injury, with permanent and career-ending sequelae. Conceivably, a replacement IA could rapidly enter the chamber as it is decompressed in order to assume patient management, thereby allowing the original IA to undergo scheduled decompression in a separate lock. Time and resources did not permit such substitution in the referenced cases. In cases of multiplace chamber cardiopulmonary arrest, CPR can and is likely to be administered during emergent decompression, something not, of course, available in a monoplace chamber.

LACK OF AIR BREAKS

The original design of the monoplace chamber was to deliver 100% oxygen in a continuous manner. As such, there was no consideration to equip it with an intermittent air breathing capability, thus preventing treatment of DCI in accordance with typical US Navy protocols. This prompted development of a monoplace-specific treatment table that did not require air breaks, since proven effective for both acute and delayed DCI presentations.[31,32,34]

In 1984, the author incorporated the aviator type oro-nasal mask in common use during multiplace chamber use at the time into monoplace operations, thereby allowing administration of USN TT5 and 6. It eventually became the position of the National Board of Diving and Hyperbaric

Medical Technology that all hyperbaric chambers, regardless of type, should be equipped with an air break capability.[43] This position has more to do with CNS oxygen toxicity prophylaxis and management of any premonitory events than treatment of DCI, *per se,* but serves both purposes.

Options now exist to enable provision of air breaks for all patient states. In those fully alert and orientated, the referenced oro-nasal mask was the early choice. As it is relatively costly, requires some maintenance and has decontamination needs, an inexpensive disposable non-rebreather oro-nasal mask (Vyaire Medical, Mettawa (IL), USA, ref 001203) evolved as an alternative and is increasingly in use today. This author has confirmed its air break effectiveness via in-chamber transcutaneous PO_2 monitoring. Careful regulation of air delivery to this free flow device is required to avoid overt dilution of the chamber's oxygen atmosphere. Observing the rebreather bag to collapse slightly upon inspiration suggests an appropriate rate of flow. Restrained patients, and others unable to manipulate a face mask (bulky burn dressing covering the hands, for example), are fitted with a face tent (CareFusion, Yorba Linda (CA), USA, Ref: 001220) prior to entry into the chamber. Oxygen flows to the device during active treatment to avoid CO_2 accumulation. To initiate an air break, the chamber operator switches the face tent's oxygen supply to air and reverts to oxygen upon completion of the air break. For patients with a tracheostomy, attachment of a trach collar (CareFusion, Yorba Linda (CA), USA, Ref: 001225) serves this same purpose. Control of oxygen and air to the collar occurs in the same manner as the face tent. For mechanically ventilated patients, adaption of the external control module and in-chamber anaesthesia bag readily permits intermittent air breathing.[24] Switching the chamber compression gas from oxygen to air to attempt provision of air breaks is ineffective and should be avoided.[44]

SUPPORT OF SERIOUSLY ILL PATIENTS

Intravenous fluid and drug administration is commonplace. The infusion pump remains outside the chamber and a through-hull assembly within the chamber door allows as many as six separate infusion lines to connect into the chamber, depending on make and model.

ECG monitoring during monoplace operations is likewise commonplace and can involve either three or five leads. Each connects to a 19-pin through-hull electrical penetrator, so capacity exists to include central arterial and central venous pressure measurements (five lines each) in those so monitored. Manually operated pressure infusers with oxygen-compatible lubricant support the heparinized solution (Ethox Medical, Buffalo (NY), USA). A monoplace-specific non-invasive blood pressure monitor became available in the 1990s (CAS Medical Systems, Branford (CT), USA). It proved particularly useful in that it avoided the need for arterial line placement in patients who require close monitoring otherwise occurring non-invasively. Unfortunately, it is no longer available due to a lack of commercial viability and there may be an occasional need for arterial line placement.

Removal of the vacuum drainage assembly and attachment of a Heimlich chest drain valve (Bard Medical, Franklin Lakes (NJ), USA. Ref 373460) accommodates patients with a chest tube(s). Urinary catheter management involves emptying the drainage bag and rolling it up with the vent open to expel residual air, then resealing, prior to treatment. This helps promote drainage, as the bag will not hang very far below the level of the bladder.

A monoplace-specific ventilator has been available since 1978 (Sechrist Industries, Anaheim (CA), USA) with PEEP and CPAP capabilities. As noted, it is readily adaptable for provision of air breaks.[24] One aspect of ventilator-patient airway management not presently available is suctioning. Attempts to do so by using the internal to external chamber pressure differential have not yet evolved to standard practice because of technical and safety considerations. Suctioning prior to treatment has proven effective enough not to interrupt treatment in the three-decade Prisma Health Richland Hospital experience.

In summary, an appropriately equipped monoplace chamber managed by a knowledgeable team is capable of supporting the full range of patient states.

LIMITED PRESSURISATION CAPABILITY

This was more a shortcoming when treatment pressures greater than 284 kPa were commonplace. Animal studies of cerebral arterial air embolism (CAGE) failed to demonstrate any advantage to preceding 304 kPa (3.0 atm abs) with compression to 608 kPa (6.0 atm abs) in terms of recovery of cortical evoked potentials and cerebral blood flow.[45] These same authors suggested that there might be advantages to confining treatment to 284 kPa. An open-skull animal model of cerebral air embolism using air as the compression and breathing gas reported elimination of arterial bubbles at pressures of 284 kPa (2.8 atm abs) (one animal), 344 kPa (3.4 atm abs) (three animals) and 405 kPa (4.0 atm abs) (two animals).[46] In every instance, there was evidence of change in bubble size and partial restoration of circulation just beyond 203 kPa (2.0 atm abs). Recent authoritative reviews have concluded that there is no conclusive evidence that higher pressures offer any advantage over 284 kPa for both decompression sickness (DCS)[11] and CAGE[47] (these clinical entities being collectively referred to as DCI). US Navy clinical experience comparing CAGE treated on USN TT6A (which includes an initial exposure to 608 kPa) and USN TT6 found no difference in rate of symptom resolution.[48] Recurrence (an ominous prognostic sign[49]) occurred in 19% of those treated on TT6A, and none when using TT6. Onsite monoplace chambers may not always be adequate for the very rare instance of a diver who experiences a rapid uncontrolled ascent after a provocative depth-time exposure.

Such instances have been associated with the use of dry suits during military and civilian diving operations.

Current recommendations centre on the 284 kPa pressure associated with USN TT5 and 6.[11,47] These same recommendations acknowledge the role of monoplace chambers "*under the direction of a diving medicine specialist*".[35] In a 20-year retrospective, Weaver reported encouraging outcomes and tolerance to monoplace chamber use for USN TT6, involving 72 cases of DCI.[33]

HEIGHTENED FIRE RISK

There is no greater threat to hyperbaric operations than fire, where the enclosed pressurised space and use of oxygen serve to compound its effects. Regrettably, monoplace and multiplace chamber fires continue to occur with disturbing frequency. In recent decades, they have largely resulted from the most fundamental of lapses in development and/or execution of a fire safety plan. This allowed, *inter alia*, patients and inside attendants to enter chambers with inadequate screening, resulting in the introduction of otherwise prohibited flame-producing, heat-generating and battery-powered items. Therefore, it is strongly recommended that a key component of any hyperbaric fire safety plan is a strictly enforced 'no pockets' policy for all chamber occupants. As biometric sensors are sufficiently miniaturized to be worn as finger rings and finger jewelry a no-pockets policy would not serve to eliminate these battery powered products. Taping over a traditional finger ring not readily removed prevents damage to the chamber's acrylic tube. This would not be appropriate for biometric sensors, which must be removed.

Oxygen concentration influences burning rates and flame spread, so an oxygen-filled monoplace chamber, indeed, involves greater consequential risk should fire occur than its multiplace counterpart. One might view this somewhat differently than fire risk, which is largely identical regardless of chamber type.

Clinically based multiplace chambers are invariably equipped with one or more fire extinguishing options, the most effective of which is water deluge. When successfully activated it prevented loss of life and serious injury.[50] On two occasions when it failed to operate, 15 occupants succumbed.[51,52] A fire suppression system has not been integral to monoplace design and manufacture. However, a recent standard within Australia and New Zealand requires all monoplace chambers operating within its jurisdiction to be equipped with a fire extinguishing system that "*continuously soaks the patient during depressurization*".[53]

It is critical that comprehensive fire safety precautions exist within every hyperbaric medicine service, regardless of chamber type. They begin with chamber design and construction compliance with authoritative codes and standards, and extend to adherence with manufacturer-recommended operational practice and periodic servicing. Such compliance and adherence renders monoplace and multiplace chambers inherently and intrinsically safe. Failure to follow recognised design codes resulted in one fatal monoplace fire.[54] Shortcomings included installation of an unapproved intercom system, which proved to be the cause of the fire. Failure to follow the most fundamental of manufacturer-recommended servicing expectations was the cause of another fatal monoplace chamber fire.[55]

Complementing manufacturer responsibilities must be an end-user hyperbaric fire safety plan that is strictly enforced. It should centre on preventing prohibited 'No Go' items from entering the chamber secondary to an unwavering screening process. Avoidance of hydrocarbon/oil-based hair and skin grooming products and use of 100% hospital provided cotton clothing (absent pockets) and linen are important additional strategies. Confirmation of patient grounding prior to every monoplace treatment is mandatory. Low relative humidity (RH) promotes static electricity accumulation, the discharge of which could be problematic in the presence of a hydrocarbon-containing atmosphere.

Manipulation of monoplace oxygen flow serves, *inter alia*, to control humidity levels. RH will decrease at high flow rates, as incoming oxygen is dry and there is little accumulation of patient insensible moisture loss. RH will increase with low flow rates as insensible moisture loss more readily accumulates. This desired effect on RH has the added benefit of limiting pulmonary irritation associated with prolonged breathing of dry oxygen. Low flow rates elevate RH from the 20s to greater than 60% in this author's experience. If monoplace patients complain of being cold, it is most likely the result of too high a flow rate. Rather than provision of an extra blanket (representing an additional 'fuel' source in the event of fire), one should slow the flow rate. Except for a high initial flow rate at the beginning of the treatment to hasten conversion of the monoplace chamber's atmosphere from air to oxygen, subsequent lowered rates contribute to patient safety and oxygen conservation.

The 100% oxygen environment restricts use of some specialised critical care equipment that otherwise could be used in the multiplace chamber.

The sum of all of this is that monoplace chambers are inherently and intrinsically safe. The onus is firmly on each programme and its operations personnel to maintain strict compliance with their respective hyperbaric safety plan.

IMPACT OF CHAMBER DECOMPRESSION ON AN EXISTING PNEUMOTHORAX

Another commonly expressed concern relates to the presence of a pneumothorax, either missed prior to compression, developed while at pressure or arising from decompression-induced pulmonary barotrauma. Residual pleural air not eliminated by compression and inherent unsaturation during

oxygen breathing will certainly expand as the chamber ascends, the degree of which is a function of initial volume and degree of pressure reduction. If the volume is significant, it could result in tension pneumothorax.[56] Should observation of the patient during decompression suggest the presence of a pneumothorax, the monoplace operator must immediately halt further ascent. Depending upon the symptomatic state, one might elect to recompress the chamber slightly prior to assembly of appropriate personnel, supplies and portable X-ray. Once they are in place, decompression begins, with the emerging clinical picture serving to dictate rate. Historic multiplace chamber precedent suggests that this process is likely to result in successful management of the patient. On several occasions, a pneumothorax was missed by multiplace IAs and decompression continued unknowingly.[57–59] Each case resulted in tension pneumothorax, one bilateral, and all successfully managed by conventional means, following removal from the chamber. Risk of in-chamber pulmonary barotrauma-induced pneumothorax is low given diver health screening requirements. In the clinical hyperbaric medicine setting, this is an important risk assessment. Patients with underlying pulmonary pathologies for which the risk-benefit analysis favours HBOT require certain precautions. They include provision of an in-chamber bronchodilator assembly, constant ECG monitoring, a reminder to report any change in status, particularly close observation during decompression and slowed ascent rates.

For treatment of CAGE secondary to pulmonary barotrauma, one author suggests a prophylactic chest tube be considered during multiplace operations and recommends it for monoplace practice.[47]

MANAGEMENT OF EXCRETA

Injured divers are encouraged to void and defaecate if possible prior to entering the chamber. For alert and orientated males, a urinal(s) accompanies the patient for use as necessary. Generally, one reserves catheterisation for the more seriously injured. For females, bladder catheterisation is more common given difficulties associated with use of a bedpan, although production of larger diameter monoplace chambers in recent years has made a bedpan somewhat more manageable. During 34 years of monoplace chamber practice at Prisma Health Richland Hospital has there been interruption of recompression for DCI in this regard.

'Short' vs. 'long' DCI treatment tables

An abbreviated approach to DCI treatment presenting at monoplace-based facilities became available in the 1970s.[30] It served to overcome the absence of the intermittent air breathing capability necessary to employ US Navy minimal recompression oxygen breathing treatment tables. Several years later a slight variant was introduced.[60] Reported as effective in acute and delayed DCS and CAGE presentations,[30,31,34,61] these tables compared favorably to standard 'long' USN treatment tables for both minor and serious forms of DCI, based upon review of 2,800 Divers Alert Network (DAN) database patients.[32]

As today's monoplace chamber is likely to be equipped with an air break capability[43] it could be argued that short tables are no longer necessary. However, some are likely to prefer their use. These tables continue to be 'highly effective' in the experience of some and appear to reduce health care costs.[34]

Summary and referral guidance

While there is tacit approval of its on-site role, controversy exists regarding hospital-based monoplace chamber treatment of DCI.[32,36,37] This chamber type's early 'minimalist' configuration and capabilities were suited for use dominated by stable, electively referred outpatients. Design criteria did not account for acutely injured divers, particularly in the era of 'deeper' recompression, alternative breathing gases, extended periods at pressure and lengthy decompressions. It was also uncommon for monoplace supervising physicians to have sought out sufficiently robust training in the diving medical aspects of HBOT. All of this would suggest that early criticism of monoplace recompression was well founded.

Over the ensuing decades, however, biomedical support capabilities have evolved to the extent that hospitalised, critically ill patients routinely undergo monoplace chamber treatment for a number of indications. A more standardised approach to decompression injury management occurred during this same period. These events sufficiently altered the dynamic to a point where today's monoplace chamber can successfully support a majority of DCI cases, when overseen by a knowledgeable physician.

In the USA, only a small minority of the many hospital-based monoplace programmes have adopted these capabilities in order to manage acute severe conditions, including DCI.[62] That few physicians responsible for monoplace delivery of HBOT elect to undergo training specific to injured diver care, and are prepared to remain available beyond normal working hours, is also problematic. Therefore, where to refer injured divers and others for whom immediate hyperbaric oxygenation is imperative is challenging.[62] Given that monoplace chambers dominate the practice of hyperbaric medicine in the USA, and are frequently in closer proximity to a dive site than a multiplace chamber, one would hope that more such programmes will commit to 24/7 availability. This 24/7 shortcoming may exist to some extent elsewhere. While this paper argues for recognition of the monoplace hyperbaric delivery system as a viable option for treatment of DCI, it does so while noting these important caveats.

When an injured diver presents to a medical facility housing a multiplace chamber there is no controversy concerning its advantages over a monoplace chamber. Controversy exists when this same patient presents at a facility equipped with a monoplace chamber. The issue has centred on whether to

Table 1
Diving accident destination planning and prioritising tool

KEY LOGISTICS	CHAMBER 1		CHAMBER 2		CHAMBER 3		CHAMBER 4	
Road distance *(miles)*								
Air distance *(miles)*								
Helicopter pad	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No
Pad altitude *(feet)**								
Chamber 24/7 available	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No
Chamber hospital based	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No
Chamber type	☐ multi	☐ mono	☐multi	☐ mono	☐ multi	☐ mono	☐ multi	☐ mono
Facility type‡	☐A ☐B ☐C ☐D		☐A ☐B ☐C ☐D		☐A ☐B ☐C ☐D		☐A ☐B ☐C ☐D	

ADVANCED CARE	CHAMBER 1		CHAMBER 2		CHAMBER 3		CHAMBER 4	
- IV fluids/drugs	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No
- ECG monitor	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No
- BP monitor	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No
- Invasive pressure	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No
- Ventilator	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No

CONTACT DETAILS	CHAMBER 1	CHAMBER 2	CHAMBER 3	CHAMBER 4
24/7 phone				
Back-up phone				
Landing pad coordinates				
Administrative email				
Administrative website				

HYPERBARIC PHYSICIAN	CHAMBER 1		CHAMBER 2		CHAMBER 3		CHAMBER 4	
i. Dr.								
DMOtrained**	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No
UHM*board certified**	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No
UHM fellowship trained	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No

HYPERBARIC PHYSICIAN	CHAMBER 1		CHAMBER 2		CHAMBER 3		CHAMBER 4	
ii. Dr.								
DMOtrained**	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No
UHM*board certified**	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No
UHM fellowship trained	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No	☐ Yes	☐ No

*** Determine increase or decrease in altitude from scene**
**** Diving Medical Officer**
***** Undersea and Hyperbaric Medicine**

‡ A Wound Center
B Civilian Hospital
C Military Facility
D Commercial Diving

effect patient transfer to a multiplace facility while accepting risks inherent with treatment delay, or to use the monoplace chamber, assuming it is deemed operationally and clinically capable. Kindwall and colleagues in Milwaukee (who felt their recommendations were objective enough as they operated both chamber types) have long held that the monoplace is adequate to treat DCI 'in most cases'.[32] In a related editorial, Moon at Duke agreed, and added that referral of a DCI case to a distant multiplace rather than immediate local treatment in a monoplace "*would be erroneous*".[63] If one accepts that these authoritative authors rendered opinions prior to several technical advancements and treatment table 'streamlining' noted herein, there should be little reason to question their current validity.

If both chamber types are equidistant from an injury site or referring hospital, the question becomes one of appropriate referral guidelines. Intuitively, the multiplace chamber would appear the logical choice and commonly is. However, some issues would need reconciliation. Table 1 represents a sample destination-planning template to guide those responsible for safe conduct of diving activities. It affords

direct comparison of key logistical (including any elevation changes), operational and clinical expertise characteristics, in order to identify which facility would be most appropriate for a given case. For example, one might argue that a severe decompression insult requiring mechanical ventilation might be better served in a capable monoplace chamber within a comprehensive medical centre, rather than a multiplace chamber housed on a military base without clinical facilities and reliant on hand-operated bag mask ventilation.

The medical department of the Divers Alert Network (DAN) classifies referral chambers based upon overall capabilities, regardless of type. In doing so, they place greater emphasis on clinical and operational quality. Comparison is made between a multiplace chamber that has few technical limitations but is 'operationally' challenged and a monoplace programme with technical limitations which are circumvented with operational skill and clinical acumen (DAN America, personal communication, 2019). This decision-making approach appears consistent with the intent of Table 1, as it is with the above example.

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Guidelines

South Pacific Underwater Medicine Society guidelines for cardiovascular risk assessment of divers

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Abstract

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The South Pacific Underwater Medicine Society (SPUMS) diving medical for recreational scuba divers was last reviewed in 2011. From 2011 to 2019, considerable advancements have occurred in cardiovascular risk assessment relevant to divers. The SPUMS 48th (2019) Annual Scientific Meeting theme was cardiovascular risk assessment in diving. The meeting had multiple presentations updating scientific information about assessing cardiovascular risk. These were distilled into a new set of guidelines at the final conference workshop. SPUMS guidelines for medical risk assessment in recreational diving have subsequently been updated and modified including a new Appendix C: *Suggested evaluation of the cardiovascular system for divers*. The revised evaluation of the cardiovascular system for divers covers the following topics:
1. Background information on the relevance of cardiovascular risk and diving;
2. Defining which divers with cardiovascular problems should not dive, or whom require treatment interventions before further review;
3. Recommended screening procedures (flowchart) for divers aged 45 and over;
4. Assessment of divers with known or symptomatic cardiovascular disease, including guidance on assessing divers with specific diagnoses such as hypertension, atrial fibrillation, cardiac pacemaker, immersion pulmonary oedema, takotsubo cardiomyopathy, hypertrophic cardiomyopathy and persistent (patent) foramen ovale;
5. Additional cardiovascular health questions included in the SPUMS guidelines for medical risk assessment in recreational diving;
6. Updated general cardiovascular medical risk assessment advice;
7. Referencing of relevant literature.
The essential elements of this guideline are presented in this paper.

Introduction

Diving in all forms places increased demands on the cardiovascular system. Immersion itself causes an increase in cardiac preload (increased venous return) and at the same time, peripheral vasoconstriction, causing an increase in blood pressure and afterload. These changes are typically accompanied by sustained mild to moderate exercise and occasional requirements for peak exercise in challenging circumstances. Given all this and the increasing age of the 'average' diver, it is not surprising that 39% of recreational diving fatalities in divers aged 45 or over in our region have a cardiac event as the disabling injury.[1]

The primary goals of evaluating the cardiovascular system in a diving candidate are to:

- Identify those who appear to be at increased risk of myocardial ischaemic events, heart failure, dysrhythmias and other cardiac pathology that might disable a diver underwater; and
- Establish that the candidate has an adequate exercise capacity for diving.

Methods

The South Pacific Underwater Medicine Society (SPUMS) 48th (2019) Annual Scientific Meeting theme was cardiovascular risk assessment in diving. The meeting had multiple presentations updating scientific information about assessing cardiovascular risk. These were distilled (with audience participation) into a new set of guidelines applicable to prospective or established divers at the final conference workshop. Conference presentations and workshop discussions were led by a committee (represented by the authorship of this paper) that included three cardiologists (NJ, RR, MT) and three diving physicians (DS, MHB, SJM). This committee subsequently refined and finalised the following guideline.

Guideline

All diving candidates and established divers aged 45 years and over should undergo a medical assessment with a focus on cardiovascular evaluation, preferably by a doctor with training in diving medicine. This recommendation is based on commonly used age criteria accepted as risk thresholds in cardiovascular risk calculators.[2,3]

WHICH DIVERS WITH CARDIOVASCULAR PROBLEMS SHOULD NOT DIVE?

Diagnoses usually considered to render an individual unsuitable for diving include:

- Untreated and/or symptomatic coronary artery disease;
- Left ventricular dysfunction of any cause. Divers with well treated or recovered left ventricular dysfunction with good ejection fraction (especially with ejection fraction (EF) > 50%) would usually be acceptable if there was good exercise capacity and the underlying causes treated. All such divers require cardiology review;
- Hypertrophic cardiomyopathy would usually preclude diving. Cardiology review is required in all cases;
- Congestive heart failure;
- Pulmonary hypertension;
- Long QT syndrome or other arrhythmia-inducing ion channelopathies;
- Paroxysmal arrhythmias causing unconsciousness or impairment of exercise capacity;
- Poor exercise capacity of apparent cardiac origin;
- Moderate to severe valvular lesions;
- Complex congenital cardiac disease. (Note that an atrial septal defect (ASD) is not included here – ASD patients are at increased risk of neurological decompression sickness (DCS) and should be assessed by a diving doctor and a cardiologist before being cleared for diving);
- The presence of an implanted cardiac defibrillator;
- Recurrent syncope;
- Anticoagulation – including warfarin, direct thrombin inhibitors (e.g., dabigatran), and factor Xa inhibitors (e.g., rivaroxaban, apixaban) or similar – for whatever reason; this does not include single antiplatelet therapy (e.g., aspirin).] Some experts allow single anticoagulant therapy under selected circumstances. This remains a controversial area and the committee acknowledges the lack of reliable evidence to support either position.

The successful treatment of some of these disorders may result in a candidate becoming suitable for diving. In particular, a candidate with coronary artery disease who has been successfully revascularized may be suitable for diving if inducible ischaemia can be excluded and adequate exercise capacity demonstrated (see below). Another example is a candidate with a history of paroxysmal arrhythmia who has undergone successful pathway ablation.

Following successful cardiac intervention, candidates may require some recovery time before commencing or resuming diving. Many cardiologists and diving physicians would not allow diving while on dual antiplatelet therapy. The precise period of diving abstinence should be determined by the cardiologist and diving physician.

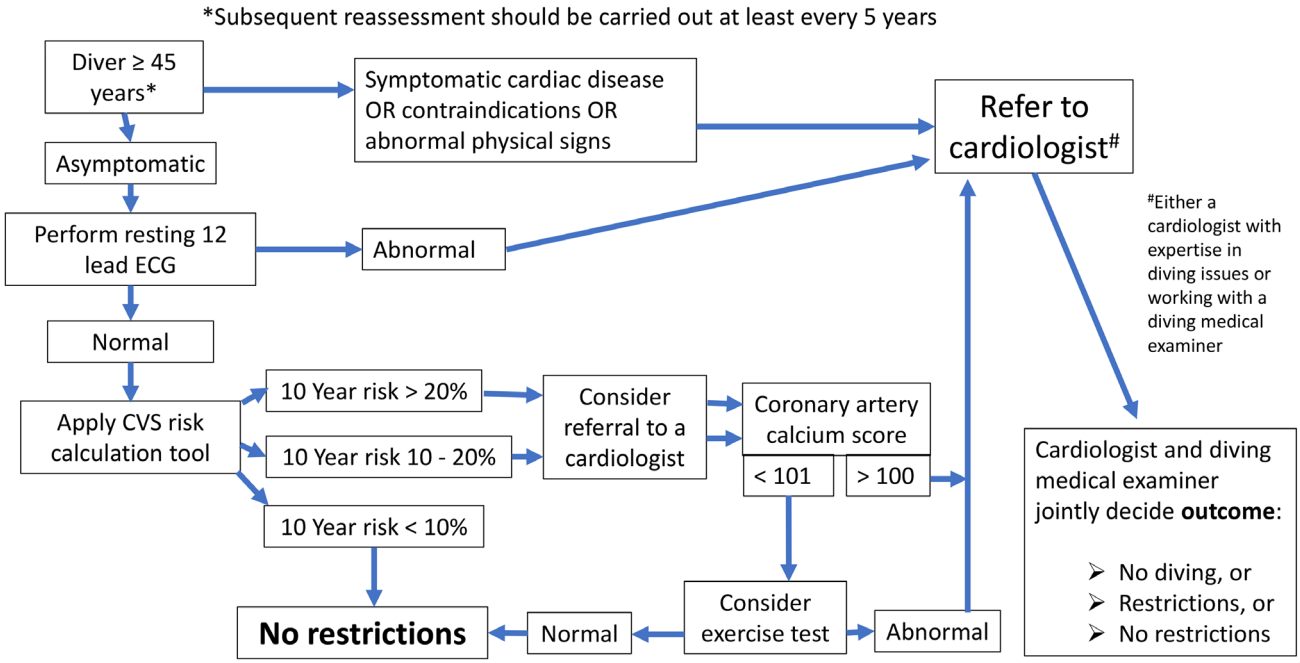
Candidates with any of the above diagnoses who wish to consider diving after appropriate treatment should be referred to a physician with training in diving medicine for evaluation.

RECOMMENDED SCREENING PROCEDURES FOR DIVERS AGED 45 AND OVER

Figure 1 proposes a screening algorithm for diving candidates or divers aged 45 years and over.

- All symptomatic candidates should be referred to a cardiologist for investigation.
- Candidates with a positive cardiovascular history (including younger diving candidates or established divers < 45 years) should undergo a focused medical assessment; initially by a doctor with training in diving medicine. Cardiology referral should be considered.
- All asymptomatic divers or candidates ≥ 45 years should have a resting electrocardiogram (ECG) performed and any significant abnormalities should prompt referral to a cardiologist.
- Asymptomatic candidates or divers ≥ 45 years should be assessed with a standard, validated, cardiovascular risk assessment tool (e.g., the National Vascular Disease Prevention Alliance in Australia).[2] The specific tool used may vary.

Figure 1
Screening algorithm for cardiovascular disease in diving candidates or divers aged 45 years and over



- Candidates with an estimated 10 year risk < 10% may proceed to diving with no further assessment. Some diving doctors would also perform a standard exercise test (with ECG monitoring). The diving medical may also prompt a discussion of life-style modification.
- Candidates with a higher risk should have a coronary calcium score and those at > 20% (and possibly those > 10%) 10-year risk should have a computed tomography (CT) angiogram and/or functional stress test. Such testing may be best organised by a cardiologist.
- A normal CT angiogram or a functional stress test negative for ischaemia suggests that the candidate should be able to dive without important excess risk.
- A plan (including review frequency) for follow-up cardiac health surveillance tailored to the diver's risk profile should be established at the time of the initial evaluation.

ASSESSMENT OF DIVERS WITH KNOWN OR SYMPTOMATIC CARDIOVASCULAR DISEASE

All candidates for diving, or seeking ongoing monitoring of their suitability to continue diving should complete the full questionnaire that forms part of the SPUMS guidelines for medical risk assessment in recreational diving.

Candidates who have responded indicating they may have known or symptomatic cardiovascular disease need further specialist investigation by an appropriate physician. This may include myocardial perfusion scan, stress echocardiography or stress exercise ECG ("stress test"). Although an exercise ECG is relatively insensitive to early coronary disease, it has the advantage of demonstrating exercise capacity and can be modified to test sustained exercise at 6 MET. Sustained exercise at a minimum of 6 MET is a pragmatic expectation for a recreational diver but there may be an occasional need to exercise transiently at higher levels during diving.

NOTES ON SPECIFIC DIAGNOSES

Treated hypertension with adequate control and in the absence of other risk factors that would indicate screening for coronary artery disease is acceptable for diving. Although local practices may vary in some details, hypertension should always be investigated and treated according to contemporary evidence-based guidelines.[4,5] Hypertension above 160/100 mmHg is a contraindication until investigated and treated.

For divers taking antihypertensive drugs, certain antihypertensive drugs may be preferred to others in the context of scuba diving, and participation in scuba diving may be of consequence for antihypertensive treatment choices. Expert opinion should be sought. It is recommended that subjects with hypertension be assessed for signs of cardiac ischaemia and/or dysfunction and be referred to a vascular specialist or cardiologist for cardiovascular screening when deemed appropriate. Divers with hypertension should be informed about the symptoms of immersion pulmonary oedema and receive specific instructions to immediately abort a dive in case of these symptoms.[5]

Atrial fibrillation where the rate is adequately controlled in a candidate without inducible myocardial ischaemia and

who exhibits adequate exercise capacity is acceptable in diving. However, many such patients are anticoagulated and anticoagulation is itself a contraindication for diving (see above). All patients with atrial fibrillation should have an echocardiogram to exclude structural heart disease and to assess for diastolic dysfunction.

Successful aberrant pathway ablation in case of Wolff Parkinson White (WPW) syndrome and atrio-ventricular nodal re-entry tachycardia (AVNRT), or pulmonary vein isolation in case of atrial fibrillation may also render the candidate acceptable for diving, however these individuals should have a bubble-contrast echo to ensure no persistent hole remains through the inter-atrial septum.

A cardiac pacemaker is not an absolute contraindication to continued diving, but the underlying pathology is important to consider, as is the proven ability of the device to function at depth. Pressure capability of a device can usually be obtained from the manufacturer.

A previous episode of *immersion pulmonary oedema, Takotsubo cardiomyopathy* or a diagnosis of *obstructive cardiomyopathy* should contra-indicate further diving until appropriately assessed. A diver or new diving candidate with such a history should be referred to a physician with training in diving medicine for discussion of the relevant issues.

Persistent (patent) foramen ovale (PFO). SPUMS does not advise routine testing for the presence of a PFO.[6]

A PFO that exhibits right-to-left shunting with no or minimal provocation is a risk factor for serious neurological DCS. In established divers, such lesions are usually discovered by bubble contrast echocardiography conducted after a relevant episode of DCS or the development of a suspicious rash after diving.

These divers are usually advised to cease diving, modify their diving to reduce venous bubble formation or to have the PFO repaired. There are some data to suggest the incidence of DCS remains higher in those who elect to modify their diving, and this option is less often recommended than previously.[7,8] When this option is taken for whatever reason, it would be reasonable to advise diving more conservatively in order to minimise venous bubbles. There are various strategies that might be employed to reduce the risk of significant venous bubble formation after diving, or the subsequent right-to-left shunting of such bubbles across a PFO. The appropriateness of this approach, and the strategies chosen, need to be considered on an individual basis and in discussion with a diving medicine expert. Examples include: reducing dive times to well inside accepted no-decompression limits; restricting dive depths to less than 15 m; performing only one dive per day; use of nitrox with air dive planning tools; intentional lengthening of a safety stop or decompression time at shallow stops and avoidance of heavy exercise and unnecessary lifting or straining for at least three hours after diving.[6–9]

Occasionally new diver candidates have a previously discovered PFO; in such cases an objective assessment of the shunting behaviour of the lesion is required in order to adequately counsel the candidate about the implied risks in diving. If not already done, this is best achieved using a bubble contrast echocardiogram and provocative manoeuvres. It is strongly recommended the results of such tests are discussed with a physician who has training in diving medicine.

ADDITIONS TO THE SPUMS DIVER HEALTH RISK ASSESSMENT QUESTIONNAIRE

As a result of the workshop, it was identified that a number of additional questions needed to be added to the SPUMS guidelines for medical risk assessment in recreational diving questionnaire, covering lifestyle and specific cardiovascular risk.

The general advice preceding the questions was also revised to state:

- If you have never heard of the condition or had the diagnosis applied to you – then reply no; and
- If you are not confident that you understand the question, then leave this blank and discuss with the doctor.

The new questions are documented next.

Lifestyle questions:

- How often do you exercise and at what estimated level of intensity of that exercise (minutes per week, high/ moderate/low intensity)?
- Are you currently smoking?
- Did you smoke in the past?
- How many cigarettes per day do/did you smoke and for how many years?
- If other forms of tobacco, please detail.

Specific cardiovascular risk questions:

- Do you have any known heart disease, or have you ever consulted a cardiologist (specialist heart doctor)?
- Is there any family history of heart disease or diabetes?
- Is there any family history of sudden death at a young age?
- Are you ever aware of a racing or irregularly beating heart, or any other known problem with your heart beat?
- Have you ever had giddiness, light-headedness or periods of unconsciousness, whether or not associated with exercise?
- Do you ever get discomfort in your chest on exertion (angina)?
- Do you get very short of breath on exertion (out of proportion to the exercise, or before your legs get tired)?
- Have you ever been short of breath lying down or woken from sleep with breathlessness?

- Do you have a pacemaker or implanted defibrillator?
- Have you ever had an operation on the heart including any placement of stents?

Have you ever had a diagnosis of the following:
a) High blood pressure?
b) Rheumatic fever or problems with your heart valves?
c) High cholesterol?
d) Immersion pulmonary oedema?
e) Heart failure, or a problem with your heart muscle including cardiomyopathy or obstructive coronary heart disease?
f) A 'hole in the heart' (patent foramen ovale, atrial septal defect, ventricular septal defect) or other congenital heart disease?
g) Blood clots in the legs or lungs?
h) A stroke?
Have you ever failed or had a significant medical issue with a diving medical in the past?

Modifications to section A 4.10 – Cardiovascular system

Twenty-eight percent (28%) of recreational diving fatalities have a cardiac event as the disabling injury. It follows that the primary goals of evaluating the cardiovascular system in a diving candidate are to identify those at risk of myocardial ischaemic events, myocardial insufficiency, or other cardiac events (such as arrhythmias) that might be disabling underwater.

All divers or diving candidates aged 45 and over are at higher risk of cardiac disease even when asymptomatic. Therefore, all should be assessed according to the guidelines documented according to the algorithm above.

The SPUMS guidelines for medical risk assessment in recreational diving (Appendix C) also provide guidance for assessment of younger candidates or other high-risk groups who have a history indicating increased cardiac risk or in whom physical examination reveals cardiovascular abnormalities.

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Professor Mitchell, an author on this paper, is the Editor of *Diving and Hyperbaric Medicine* journal. However, there were no review management process conflicts in respect of this article. As an adopted societal guideline arising from an iterative process of consensus generation by international experts, it was not subject to peer review.

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Diving after SARS-CoV-2 (COVID-19) infection: Fitness to dive assessment and medical guidance

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Key words
Diving medicine; Health surveillance; Medicals-diving; Occupational health; Pulmonary barotrauma; Exercise; Cardiovascular

Abstract

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Scuba diving is a critical activity for commercial industry, military activities, research, and public safety, as well as a passion for many recreational divers. Physicians are expected to provide return-to-diving recommendations after SARS-CoV-2 (COVID-19) infection based upon the best available evidence, often drawn from experience with other, similar diseases. Scuba diving presents unique physiologic challenges to the body secondary to immersion, increased pressure and increased work of breathing. The long-term sequelae of COVID-19 are still unknown, but if they are proven to be similar to other coronaviruses (such as Middle East respiratory syndrome or SARS-CoV-1) they may result in long-term pulmonary and cardiac sequelae that impact divers' ability to safely return to scuba diving. This review considers available literature and the pathophysiology of COVID-19 as it relates to diving fitness, including current recommendations for similar illnesses, and proposes guidelines for evaluation of divers after COVID-19. The guidelines are based upon best available evidence about COVID-19, as well as past experience with determination of diving fitness. It is likely that all divers who have contracted COVID-19 will require a medical evaluation prior to return to diving with emphasis upon pulmonary and cardiac function as well as exercise capacity.

Introduction

Scuba diving is a passion for many recreational divers, but also represents a critical component of the commercial diving industry, scientific research, military operations and public safety diving. Severe acute respiratory syndrome coronavirus two (SARS-CoV-2 hereafter referred to as 'COVID-19') affects the lungs with potential sequelae in the lung parenchyma.[1] During descent to depth gas-containing anatomic spaces will be compressed, and during ascent, any compressed gas introduced to these spaces will expand. These potential gas volume changes create a risk of barotrauma, hence the general consensus that healthy lungs are a requirement for diving.[2,3] Diving also poses significant stressors on the cardiovascular system resulting from increases in systemic blood pressure, centralisation of blood volume, thermal stress from water immersion and increases in systemic oxygen consumption.[4] Physical challenges similar to those of other sports would require similar evaluation while keeping in mind the added physiologic changes from immersion.[5] There are currently widespread concerns in the diving and medical communities on fitness to return to dive post COVID-19. Research examining the origins and structure of the virus, its pathogenesis, and the clinical features of its acute presentation is growing, creating a foundation of evidence from which to draw while evaluating divers. However, the long-term sequelae are still unknown.

University of California San Diego diving medicine practitioners released guidelines for return to diving based upon decades of diving medical experience and the currently available literature about COVID-19.[6] Our objective here is to review the available literature considered when generating these guidelines, as well as to discuss the potential implications of COVID-19 sequelae in divers and draw relevance from fitness to dive implications of similar diseases.

Background

In a pre-COVID-19 underwater world, decompression sickness (DCS) and pulmonary barotrauma with resultant arterial gas embolism (AGE) were the dominant diving-related injuries originating from breathing compressed air under pressure and requiring treatment with hyperbaric oxygen treatment in a pressurised chamber. The overall per capita DCS rate among recreational divers has been reported as 20.5 per 10,000 person years.[7] Although DCS is a rare and usually self-limiting injury, permanent disability can occur. The risk of dying from recreational diving activities is small with Divers Alert Network (DAN) reporting 1.8 deaths per 100,000 divers per year.[8] Common factors associated with diving fatalities include insufficient breathing gas, panic, rapid ascent and equipment malfunction/problems.[8] In the 2018 DAN Annual Diving Report, the leading cause of death was drowning while the leading 'disabling injury' that led to death was cardiovascular-related problems.[8] COVID-19 exhibits a pathophysiology acutely relevant to diving with potentially increased susceptibility to DCS, AGE, or immersion pulmonary oedema from cardiac, pulmonary and haematological events. This pandemic may precipitate a much larger demand for diving medical examinations.

Magnitude of the problem

Gathering reliable data to assess the number of active scuba divers has eluded the diving industry since its inception. There are no available tools to track diver participation in this sport. Further, a case definition of what constitutes an active scuba diver remains subject to diverse interpretations.

The generally accepted range of 'active' scuba divers in the US by the Diving Equipment and Marketing Association (DEMA) is from 2.5 to 3.5 million.[9] The Sports and Fitness Industry Association (SFIA) reports 2,351,000 million casual participants in scuba diving and 823,000 core participants.[10] DAN, a diving safety organisation, likely has a representative active segment of the recreational scuba diving community with members who regularly participate in scuba diving activities and, therefore, procure DAN membership and diving accident insurance. With the caveat that DAN represents recreational divers and a large market share in the USA and Europe, DAN reports the following 2019 membership numbers worldwide: DAN US/Canada, 274,708; DAN Europe, 123,680; DAN Japan, 18,137; DAN World Asia Pacific, 12,163; DAN World Latin America/ Brazil, 8,008; DAN South Africa, 5,894.

The true active segment of the US diving population could be fewer than 1,000,000, possibly as low as 500,000, depending on the definition of 'active'. SFIA does not track global numbers and there is no equivalent organisation outside the US.

Entry of non-divers into the sport through certification courses also introduces a level of uncertainty when attempting to assess the magnitude of the diving medical examination need. Before COVID-19, three reporting scuba training/certification organisations (Professional Association of Diving Instructors PADI, Scuba Diving International, Scuba Schools International) submitted to DEMA a combined average of 22,325 entry-level certifications per quarter (personal communication, Tom Ingram, CEO DEMA, YEAR). At the leadership level, deriving the number of active scuba instructors in the US and internationally is equally difficult. Thirteen US organisations train and certify scuba instructors. Globally, over 300 individual certifying organisations train and certify divers and instructors. The Professional Association of Diving Instructors (PADI) reported 137,000 professional members worldwide in 2019. If PADI represents 70% market share, then the number of instructors globally reasonably approximates 195,000.

Scientific divers reported by the American Academy of Underwater Sciences (AAUS) number 4,500 at 150 organisational member scientific diving programmes.[11] The Centers for Disease Control and Prevention (CDC) and Bureau of Labor Statistics reported 3,380 commercial divers in the US.[12] The true number of active public safety divers is similarly unknown, but estimated to be between 3,000 and 5,000 in the US (personal communication, R Sadler, Medical Director of Dive Rescue International, 2019).

Recreational diver medical certification by a physician before undertaking diver training is a requirement in some countries, driven by insurance regulations. In the US, evaluation before recreational and governmental certification courses is done in the form of a screening medical questionnaire (the World Recreational Scuba Training Council (WRSTC) diver medical participant questionnaire). An evaluation by a physician is required for those who have positive responses on the questionnaire or for those engaged in any professional diving leadership training or certification programme (i.e., dive guide, divemaster, assistant instructor or instructor).[13] Diving medical certification for scientific divers and commercial divers in the US is required and regulated by the Occupational Safety and Health Administration (OSHA).[14] These divers are subject to routine diving medical examination intervals as mandated by their organisations and the AAUS. Most scientific divers are exempt from OSHA, but still have required exams regulated by AAUS.

Development of fitness to dive guidelines in the COVID-19 pandemic

There has been much recent discussion in the diving and medical communities on the evaluation of new diving candidates or return to diving by existing divers who have had COVID-19. Segments of the diving community (e.g., recreational instructors, commercial, military, scientific

and public safety divers) may be required under certain circumstances by employers, or if they have tested positive for COVID-19, to undergo diving medical (re)certification to identify or mitigate potential risks from COVID-19 sequelae.

The diving medicine community is presented with the challenge of performing fitness to dive evaluations in the context of a disease in which the natural history is currently unknown. In what is known of its pathophysiology the pulmonary, cardiac, and thromboembolic/hypercoagulable effects seem to be very relevant to divers. Potential long-term sequelae will primarily relate to structural changes of the lung parenchyma such as the fibrosis reported in severe acute respiratory syndrome (SARS-CoV-1) and Middle East respiratory syndrome (MERS) potentially increasing risk of barotrauma, and also decreased exercise tolerance, increased susceptibility to cardiac events such as heart failure, pulmonary oedema, and arrhythmias.[1] Effects of COVID-19 on other organ systems are less specific or less relevant for the specific challenges of diving.

The recommendations promulgated here are based on knowledge of similar conditions and the limited specific knowledge of COVID-19. It should be noted some of the conditions discussed below may be automatically disqualifying in the more conservative proscriptive approach to evaluating for commercial, military, public safety or scientific divers, and some of these recommendations may only apply to recreational divers. It is not within the scope of this paper to dissect the nuances between these organisations.

Pulmonary

PATHOPHYSIOLOGY OF COVID-19 AND RELEVANCE TO DIVING

Although COVID-19 has many clinical manifestations, the primary mechanism of infection, morbidity and mortality has been in the form of a respiratory illness, ranging from mild to severe. The mechanism of pulmonary damage from the virus is thought to be related to a cytokine-mediated inflammatory response, possibly compounded by iatrogenic injury from mechanical ventilation, high airway pressures and exposure to hyperoxia.[1]

There have been many reports describing some of the characteristic acute changes seen on lung imaging in these patients. In a retrospective case series,[15] the changes seen on computerised tomography (CT) scans in patients with COVID-19 and mild to moderate disease were most commonly ground glass opacities (GGO), crazy-paving pattern, and consolidations, with subpleural (peripheral) abnormalities being more common than central. The majority of patients had bilateral disease. These radiographic abnormalities seemed to peak around day 10 of the illness and then gradually resolved. At the time follow-up ended (approximately 26 days after onset of illness), the crazy-paving pattern had resolved, but there were still extensive GGO present.[15] Similar illnesses from other members of the coronavirus family have been seen, specifically in SARS-CoV-1and MERS.[16] Similar to these other diseases, COVID-19 patients may initially have normal chest radiographs (15–20%) and then go on to develop peripheral, multifocal airspace disease (GGO and/or consolidation), cavitation, and lymphadenopathy. Spontaneous pneumothorax is rare. COVID-19 is more often bilateral (similar to MERS) and follow-up imaging (as noted above) will often show progressive opacities.[16]

The chronic phase of COVID-19 has not been described yet; but one may hypothesise it will behave similarly to the other coronaviruses. In a small case series, follow-up chest radiographs showed pulmonary fibrosis in one in three MERS patients (median time of follow-up 43 days).[17] Persistent radiographic findings were associated with a history of a more severe acute clinical course. The follow-up of SARS-CoV-1 patients has been slightly better described. Two-hundred and fifty-eight patients were followed for three months after discharge and all had serial chest imaging and pulmonary function tests (PFTs) performed.[18] If either were abnormal, the patients proceeded to CT imaging. Fifty patients had abnormal diffusion capacity for carbon monoxide (D_{LCO}) (defined as < 80% of predicted value) and 48 patients had residual abnormalities on chest radiography (CXR). The initial follow-up was done at approximately one month and both PFTs and imaging continued to improve at repeat examinations. It should also be noted these patients were symptomatic at the time of follow-up, describing generalised weakness and decreased exercise tolerance due to shortness of breath. Abnormalities of pulmonary diffusion testing were found to be more sensitive than CT scans and imaging seemed to lag behind the clinical course.

Persistent abnormalities were also seen in SARS-CoV-1 patients on CT with paired inspiration-expiration views at follow-up.[19] Imaging done at an average of 140 days post onset of symptoms showed persistent abnormalities, including GGO, reticulations, and air trapping in 16 out of 20 patients. A 15 year follow-up study of SARS-CoV-1 patients showed a rapid improvement in radiographic lesions over the first year followed by a plateau and relatively stable PFTs.[20]

The above described sequelae of other coronaviruses are certainly worrisome and if COVID-19 behaves similarly, it may have potential implications for divers. First is the potential for decreased exercise capacity because of residual lung disease. Pulmonary demands of diving are distinct from those of exercise on land, being affected by increased breathing resistance from diving equipment, immersion, and gas density.[21] This is discussed in further detail in the cardiac section below, but an appropriate exercise capacity will be required for return to diving. Second, there is the possibility that these residual changes would potentially expose the diver to a higher risk of barotrauma. The

increased risk of pulmonary barotrauma, potentially because of decreased lung compliance from residual scarring or fibrosis, structural abnormalities such as bullae or blebs or residual air trapping may be a significant issue for divers. Additionally, residual lung disease from COVID-19 could interfere with the pulmonary vasculature enough to allow asymptomatic venous gas emboli to more easily cross the pulmonary capillary filter leading to their arterialisation.

FITNESS TO DIVE IN OTHER PULMONARY CONDITIONS

Pneumonia (lobar, viral). A CXR should be performed after six weeks to ensure no underlying structural abnormality is present. Exercise tolerance should be back to the patient's baseline and oxygen saturation with exercise should be normal. PFTs should be back to baseline; this usually occurs within three to five weeks following a pulmonary infection.[22]

Organising pneumonia (OP). COVID-19 bears resemblance to OP on chest CT, which is an inflammatory process rather than an infectious one.[23,24] OP is usually non-necrotizing but often takes longer to resolve than infectious pneumonias. However, there is no known distinction or prior data separating OP from other forms of pneumonia related to diving. Thus, a CXR is indicated after the clinical resolution of disease.

Pulmonary fibrosis. Pulmonary fibrosis is considered a contraindication for scuba diving because of the increased risk of barotrauma due to decreased lung compliance. There is also concern that the fibrotic lung tissue will interfere with gas exchange. However, this is based on expert opinion and not evidence, as noted in the British Thoracic Society guidelines.[25]

Blebs and bullae. Traditional teaching has been blebs and bullae should be contraindications to scuba diving because of increased risk of overexpansion injury and subsequent barotrauma, including the potential for pneumothorax and arterial gas embolism. Indeed, there have been case reports of individuals who have suffered barotrauma and subsequently were found to have pulmonary bullae.[26,27]

Many of the lesions described in these case reports are relatively large (> 20 mm) and it seems reasonable that patients with large bullae and blebs should be excluded from diving.[27] It is a disqualifying condition for military, commercial, and scientific divers. However, the guidance on small blebs and bullae in recreational divers is less clear. Asymptomatic blebs and bullae have been found in a large portion of the population. In a case series of autopsies performed on otherwise healthy individuals without lung disease, the incidence was approximately 33%.[28] Some describe it as so frequent a finding that "*radiologists in my institution do not routinely report on small blebs on CT scans as they are so common as to be considered normal findings in the patient population seen by a major hospital radiology department*".[29] Given the large number of active divers, the high incidence of blebs and bullae in the population and the relatively low incidence of pulmonary barotrauma and arterial gas embolism in divers, it seems reasonable to conclude there are many individuals scuba diving with blebs and bullae without injury. Indeed, this is the basis for not recommending routine screening CT scans in professional divers, as it would unnecessarily disqualify many divers.[29] Thus, it has been the practice at our institution for recreational divers with incidentally discovered small blebs and bullae to continue diving, though with a discussion of potential risks and risk mitigation.

Pneumothorax. While a history of spontaneous pneumothorax remains a contraindication to diving because of the increased risk of recurrent pneumothorax, patients with a history of post-traumatic and uncomplicated iatrogenic pneumothoraces may be candidates to dive if they have normal PFTs and follow-up imaging.[25] For individuals with a history of significant lung injury or surgery, in which there is concern for air trapping, a high-resolution chest CT scan with inspiratory and expiratory views can confirm or rule out air trapping.

Asthma. Controversy exists in the diving community with regard to whether asthmatics should dive.[25] Theoretical risks to the asthmatic diver include increased risk of barotrauma, decreased exercise tolerance and potentially cold/exercised induced bronchospasm while diving.[21] However, review of retrospective data shows there are many asthmatics diving without significantly increased risk of morbidity. Current recommendations suggest asthmatics with normal PFTs are candidates for diving. With exercise and cold-induced asthma, they require appropriate exercise/cold challenged PFTs.[21,30–32]

Cardiac

PATHOPHYSIOLOGY OF COVID-19 AND RELEVANCE TO DIVING

COVID-19 manifests primarily as atypical pneumonia, but cardiac involvement is increasingly recognised as a prominent feature. There are no uniform patterns of cardiovascular manifestations in patients with COVID-19. They constitute a spectrum with significant clinical overlap that includes isolated elevations in cardiac biomarkers, acute coronary syndrome (ACS), arrhythmias, and myo- or myopericarditis.[33] Published autopsy series have shown myocyte necrosis, lymphocytic myocardial infiltrates and dilated right ventricles.[34] Despite the virus's cardiotropic potential, direct myocardial invasion has not been proven, suggesting that most of the myocardial damage is secondary to systemic inflammation or a general hypercoagulable state.[35] Direct invasion of the virus has been shown, however, in the endothelial cells.[36] This may have significant long-

term implications, particularly for divers, because DCS appears to cause endothelial dysfunction. Troponin elevation is reported to occur in 8–28% of patients with COVID-19 and is associated with a higher mortality risk.[37] Isolated elevations in brain natriuretic peptide (BNP) have also been reported in patients with increased mortality.[38] The mechanisms underlying cardiac injury in these patients are complex and the following have been proposed:

- COVID-19 uses the angiotensin converting enzyme II (ACE-2) receptor as a port of entry to human cells and in turn leads to its down-regulation. This results in an increase of circulating angiotensin-II levels leading to vasoconstriction, inflammation and a prothrombotic state.[39]
- Systemic hyperinflammation resulting from uncontrolled amplification of cytokine production following the initial immunologic response against viral replication may lead to direct myocardial injury, microvascular dysfunction or atherosclerotic plaque rupture.[35]
- Cardiac damage may result from direct invasion of the virus into the myocardium or the endothelium.[36]
- Myocardial damage may simply result from increased metabolic demands coupled with systemic hypoxia due to respiratory failure.

Our knowledge of these manifestations derives mostly from reports of hospitalised patients with higher disease severity.[40,41] The incidence of cardiac involvement in patients with mild or moderate illness managed in ambulatory settings is largely unknown. Attention should be paid to the potential lack of work-up patients may have received during the acute phase of their illness. In this pandemic, resources are strained and patients convalesce at home in circumstances where they may otherwise be hospitalised. If there is a history of potential cardiac involvement, the physician should attempt to clarify what type of cardiac manifestations were present and to what degree they were evaluated. Examples of potential cardiac manifestations include myocarditis, myocardial injury (evidenced by elevated biomarkers), cardiomyopathy, arrhythmias, thromboembolic disease, and acute coronary syndrome.[42]

This will help answer important questions such as:

- What was the right and left ventricular function?
- Was there evidence of myocardial inflammation on MRI or other testing modalities? and
- Was epicardial coronary disease diagnosed with coronary angiography and/or treated with percutaneous coronary intervention (PCI)?[33] Establishing the severity of the viral illness and the nature of cardiac involvement will be paramount in guiding the clearance to dive process.

Recreational and commercial diving to a greater degree, may demand a work-rate of 6–7 MET (approximately 21 $ml \cdot kg^{-1} \cdot min^{-1}$ oxygen consumption) or higher when managing common contingencies. Hence, adequate cardiac reserve needs to be established before diving. Current guidelines recommend at least 10 MET (approximately 35 $ml \cdot kg^{-1} \cdot min^{-1}$ oxygen) for commercial divers and 6 MET for recreational divers.[13,43] Thorough cardiac evaluation should be done in patients recovering from a disease with complex pathophysiology such as COVID-19.[33] Silent residual cardiac inflammation could be unmasked by the stresses of the underwater environment resulting in decompensated heart failure or cardiac arrhythmias, among others.

FITNESS TO DIVE IN OTHER CARDIAC CONDITIONS

Coronary artery disease

Coronary artery disease and hypertension are well known risk factors for fatalities associated with diving.[44] Clearance for future diving will depend on the overall burden of disease and, if a coronary intervention was performed, on the burden of residual disease. Some modality of exercise stress testing should be performed before returning to dive to rule out ongoing ischaemia and demonstrate adequate exercise capacity.[45]

Myocarditis/pericarditis/congestive heart failure

In general, heart failure and cardiomyopathies are considered a contraindication to diving because of the increased cardiovascular demands placed on the heart from diving.[44] Clearing patients to dive after pericarditis or myocarditis has not been comprehensively discussed in the literature, but one would presumably need a normal echocardiogram and likely a normal stress echo. Diving is known to be high risk for precipitating arrhythmias, and cases of Takotsubo cardiomyopathy have been reported.[43,46,47] The effects of immersion and centralised shunting of blood also place the diver at risk for immersion pulmonary oedema.[48,49] A depressed left ventricular ejection fraction or impaired exercise tolerance during an exercise stress test limited by symptoms or arrhythmias should disqualify a candidate from diving.

Thromboembolic disease

PATHOPHYSIOLOGY OF COVID-19 AND RELEVANCE TO DIVING

The incidence of venous thromboembolism (VTE) has been reported to be significantly increased in COVID-19 compared to other similar illnesses, but the true incidence is unknown.[50] COVID-19 is thought to induce a hypercoagulable state and although the mechanism remains to be elucidated (potentially via ACE-2 receptors and increased levels of Angiotensin II), there have been reports of elevated D-dimers and troponins, which are associated with worse outcomes.[50] In the acute phase, this predisposes the patient to multiple complications, including VTE and ACS. The question has

been raised whether or not this would predispose a diver who had COVID-19 to DCS, which is also thought to be an inflammatory, prothrombotic state. The chronic effects are unknown, but it seems unlikely the hypercoagulable state would remain after the acute phase of the illness is over. The more likely consequence to divers would be the complication of ACS (see above in cardiac section) or pulmonary embolism.

FITNESS TO DIVE IN OTHER THROMBOEMBOLIC CONDITIONS

Pulmonary embolism. Pulmonary embolism is not an absolute contraindication to diving. An echocardiogram and exercise tolerance test could be performed to evaluate for any residual right heart strain or development of chronic thromboembolic pulmonary hypertension. Caution should be taken when diving on anticoagulant medication due to the increased risk of bleeding from even minor trauma.

Current recommendations for evaluation of divers or diving candidates after COVID-19

We have developed working guidelines based on the limited evidence of sequelae of COVID-19 available and experience with other diseases which share similar features (see above). We have categorised divers based on the history and severity of their illness and determined their return to dive evaluation accordingly. As with any illness, ultimately the work-up is left to the discretion of the evaluating physician. The guidelines which follow explicitly pertain to divers who are asymptomatic after their illness, including normal exercise tolerance (see exercise tolerance section). We currently recommend following CDC guidelines for screening of an employee for any diver prior to diving.[51] Measuring vital signs or oxygen saturation routinely before diving is not practical, but no diver should dive if they currently have or within 14 days have had any cough, shortness of breath or difficulty breathing, fever, chills, myalgias or new loss of smell or taste.

DEFINITIONS OF TERMS USED IN GUIDELINES

COVID-19 suspected illness

We define a COVID-19-suspected illness as symptoms consistent with COVID-19 with or without a positive polymerase chain reaction (PCR) or antibody test, as testing is currently unreliable. As more accurate antibody testing is developed and becomes widely available it will likely be useful in guiding these evaluations. In defining 'symptoms consistent with COVID-19' we are currently using the CDC case definition of COVID-19 for those patients who did not have PCR or antibody confirmed illness.[51]

Thus, a COVID-19 illness is suspected when there have been at least two of the following symptoms:

- fever (measured or subjective), chills, rigors, myalgia, headache, sore throat, new olfactory and taste disorder(s);
- OR at least one of the following symptoms: cough, shortness of breath, or difficulty breathing;
- OR severe respiratory illness with at least one of clinical or radiographic evidence of pneumonia, or acute respiratory distress syndrome (ARDS);
- AND no alternative more likely diagnosis.

Exercise tolerance

Exercise tolerance is the most important definition used in our guidelines and it is vital physicians evaluate it carefully. It has been our experience that a diver with significant cardiac or pulmonary pathophysiology will most likely not have normal exercise tolerance. However, the definition of the word normal is critical. First, the diver must have returned to his or her baseline level of exercise tolerance. Even minor deviations from their baseline ('getting more winded', longer recovery times, etc) warrant further testing and investigation. Second, the physician must be satisfied the diver's exercise regimen reflects an appropriate level of equivalence for the predictable demands of diving. As mentioned above, current guidelines recommend at least 10 MET for commercial divers and 6 MET for recreational divers.[13,43] If the physician is not assured the diver's self-reported exercise level meets appropriate criteria or is concerned it would not reveal underlying cardiac or pulmonary disease, further testing is warranted.

GUIDELINES FOR DIVER EVALUATION

The following guidelines are intended for the evaluation of divers who had a COVID-19 suspected illness, are currently asymptomatic, and have subjectively returned to their baseline exercise tolerance. The tables are intended to be used in sequential fashion (i.e., Table 1, then Table 2). Table 1 requires a thorough history of the diver's illness in order to appropriately categorise them. A diver should be placed in the highest category where they meet any (not all) of the criteria. For example, any patient who was hospitalised or required the use of supplemental oxygen is automatically categorised as moderate or severe. Any patient who required intensive care unit (ICU) admission or any assisted ventilation, such as bi-level positive airway pressure (BiPAP), continuous positive airway pressure (CPAP) or intubation, is categorised as severe. If a patient was hospitalised and there is no record of a cardiac work up, they are also placed in the severe category.

After a patient has been categorised based on their initial illness severity, Table 2 is then used to guide their work up before returning to dive. The initial imaging recommended is a chest X-ray and a CT performed if the X-ray is abnormal. A CT scan (potentially with inspiration/expiration views) would be more sensitive than a radiograph for detecting abnormalities but it is our position that a CT scan is not

Table 1
Classification of divers or diving candidates based on severity of COVID-19 suspected illness. The categories of divers are based upon presenting symptoms and severity of disease, including oxygen requirement, imaging, need for and level of hospitalisation, and cardiac involvement. A diver should be placed in the highest category where they meet any (not all) of the criteria. BIPAP = bilevel positive airway pressure support; BNP = brain natriuretic peptide; CK-MB = creatine kinase MB fraction; CPAP = continuous positive airway pressure support; CT = computed tomography; DVT = deep venous thrombosis; ECG = electrocardiogram; ICU = intensive care unit

Category 0 *NO history of COVID-19-suspected illness*	**Category 1** ***MILD*** *COVID-19-suspected illness*	**Category 2** ***MODERATE*** *COVID-19-suspected illness*	**Category 3** ***SEVERE*** *COVID-19-suspected illness*
Definition: Divers who have no history of COVID-19 suspected illness should proceed with normal evaluations. Additionally, we would use these criteria in those who may have had a positive screening PCR or antibody test, but without any history of illness or symptoms consistent with COVID-19.	Definition: • Did not seek health care or received outpatient treatment only without evidence of hypoxaemia. • Did not require supplemental oxygen. • Imaging was normal or not required.	Definition: • Required supplemental oxygen or was hypoxic. • Had abnormal chest imaging (chest radiograph or CT scan). • Admitted to the hospital but did NOT require mechanical (intubation) or assisted ventilation (BIPAP, CPAP) or ICU level of care. • If admitted, had documentation of a normal cardiac work up including normal ECG and cardiac biomarkers e.g., troponin or CK-MB and BNP.	Definition: • Required mechanical (intubation) or assisted ventilation (BIPAP, CPAP) or ICU level of care. • Cardiac involvement defined as abnormal ECG or echocardiogram, or elevated cardiac biomarkers e.g., troponin or CK-MB and BNP (or absence of documented work up). • Thromboembolic complications (such as pulmonary embolism, DVT, or other coagulopathy).

indicated if a patient has a normal radiograph, PFTs, and exercise tolerance. CT may be overly sensitive for clinically insignificant lesions, as well as cause unnecessary radiation exposure and cost.

The guidelines detailed above require a more rigorous and conservative workup than would traditionally be required after a viral respiratory illness. However, this disease has proven itself to be atypical in a number of ways, including multi-organ system involvement and potential long-term effects on the pulmonary and cardiovascular systems. It is impossible to provide an algorithm that encompasses all combinations of the nature and severity of apparent COVID-19 sequelae, but that decision-making is likely to be based on similar principles to those applied in evaluating similar respiratory and cardiovascular problems arising from other causes. Because of the potential risks (including barotrauma, decreased exercise tolerance, cardiomyopathy, and arrhythmia), it is prudent to do a thorough evaluation of divers who have recovered from COVID-19.

Symptomatic divers or those with abnormal test results

Symptomatic individuals or those who have abnormal testing per the guidelines above should be advised not to dive (though each will need to be evaluated on a case by case basis and exceptions are to be expected). For example, those with persistent parenchymal damage, evidence of air trapping, cardiac injury, or inadequate exercise capacity should be advised not to dive. However, there may be more subtle abnormalities, such as borderline PFTs or a subtle radiographic abnormality in an otherwise asymptomatic healthy diver. These may not necessarily represent a lifetime ban on diving as many of the sequelae which are currently disqualifying may resolve over time and re-testing may be indicated. It is beyond the scope of this paper to provide detailed recommendations for all of these possibilities and we strongly recommend that the interpretation of the results of investigation, ongoing health re-evaluation or surveillance, and related fitness to dive decisions involve a physician with training in diving medicine.

Limitations

As evidenced by the above discussion, there is relatively little fitness to dive literature in general and what is available consists of case series and reports, as well as workshop proceedings. Most recommendations are the results of consensus and opinion. The long-term effects of COVID-19 are even less well understood and the recommendations above are made with the expectation that they will be revised as more evidence becomes available.

Conclusions

The potential implications of COVID-19 on fitness for diving are real, though the chronic sequelae of this disease are not yet known. Diving physicians are mandated to proceed

Table 2

Recommendations for evaluations of divers or diving candidates. Recommendations for evaluation are based upon the divers' severity of COVID-19 suspected illness (see Table 1). If results are unknown or unavailable, recommendations are for more extensive cardiac and pulmonary evaluations. BNP = brain natriuretic peptide; CK-MB = creatine kinase MB fraction; CT = computed tomography; ECG = electrocardiogram; PA = posterior-anterior; RSTC = Recreational Scuba Training Council. * If there is doubt that the diver's self-reported exercise level meets appropriate criteria or concern it would not reveal underlying cardiac or pulmonary disease, further testing is warranted

Category 0 *NO history of COVID-19-suspected illness*	**Category 1** *MILD COVID-19-suspected illness*	**Category 2** *MODERATE COVID-19-suspected illness*	**Category 3** *SEVERE COVID-19-suspected illness*
• Initial/periodic exam per professional group or RSTC guidelines. • Chest radiograph only if required per professional group or RSTC guidelines. • No additional testing required.	• Initial/periodic exam per professional group or RSTC guidelines. • Spirometry. • Chest radiograph (PA and lateral); if abnormal, obtain chest CT. • If unknown (or unsatisfactory) exercise tolerance*, perform exercise tolerance test with oxygen saturation.	• Initial/periodic exam per professional group or RSTC guidelines. • Spirometry. • Chest radiograph (PA and lateral); if abnormal, obtain chest CT. • ECG. • Echocardiogram (if no work up was done as an inpatient. Can forgo if had negative work up). • If unknown (or unsatisfactory) exercise tolerance*, perform exercise tolerance test with oxygen saturation. • Investigation and management of any other complications or symptoms per provider and professional group or RSTC guidelines.	• Initial/periodic exam per professional group or RSTC guidelines. • Spirometry • Chest radiograph (PA and lateral); if abnormal, obtain chest CT. • ECG. • Repeat cardiac troponin or CK-MB and BNP to ensure normalization. • Echocardiogram. • Exercise Echocardiogram with oxygen saturation. • Investigation and management of any other complications or symptoms per provider and professional group or RSTC guidelines.

with fitness to dive evaluations despite this lack of evidence and thus must draw upon past experience with similar conditions. The above guidelines represent our current opinion on best practice and will continue to be updated as a better understanding of the novel COVID-19 is gained. The updated guidelines will be available at: https://health.ucsd.edu/coronavirus/Documents/UC%20San%20Diego%20Guidelines%20for%20Evaluation%20of%20Divers%20during%20COVID-19%20pandemic.pdf.

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Short communication

Pre-hydration strongly reduces decompression sickness occurrence after a simulated dive in the rat

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Key words
Hydration; Animal model; Rat; Diving deaths

Abstract
(Wang Q, Guerrero F, Theron M. Pre-hydration strongly reduces decompression sickness occurrence after a simulated dive in rat. Diving and Hyperbaric Medicine. 2020 September 30;50(3):288–291. doi: 10.28920/dhm50.3.288-291. PMID: 32957132.)
Introduction: Hydration status is considered a parameter likely to influence the risk of decompression sickness (DCS), but scientific evidence is scarce and conflicting. This experiment aimed to analyse the influence of pre-hydration on DCS occurrence in a rat model.
Methods: Intra-peritoneal injections of saline solution were administered to rats (NaCl 0.9% 0 ml (Control), 0.1 ml (Group 1), or 1 ml·100g^{-1} body mass (Group 2) at each of 24 h, 12 h, and 30 min prior to simulated air dives (45 min at 1,010 kPa; compression and decompression rates 101 kPa·min^{-1}; stops 5 min at 202 kPa, 5 min at 160 kPa, 10 min at 130 kPa). Evaluation of DCS occurrence and severity was made after decompression.
Results: Pre-dive hydration reduced severe DCS from 47% (Control) to 29% (Group 1) and 0% (Group 2), and increased the proportion of animals without any signs of DCS from 40 (Control) to 57% (Group 1) and 93% (Group 2); Chi^2 $P = 0.041$.
Conclusions: This experiment demonstrated that pre-hydration can drastically reduce the DCS occurrence in an animal model. In the context of scuba diving, this result highlights the importance of elucidating the mechanisms linking hydration status and DCS risk.

Introduction

During scuba diving, the tissues of divers are progressively loaded with inert gases and during the fall in ambient pressure when ascending and reaching the surface, tissue gas supersaturation may lead to bubble formation. Tissue and circulating bubbles are considered to be the primary trigger of decompression sickness (DCS). Circulating bubbles are nevertheless a poor predictor of DCS[1] and the mechanisms of DCS are far from being fully understood. During the last decade, preconditioning strategies (exercise, sauna, preoxygenation, vibration, chocolate or hydration) have been investigated as potential means of reducing the risk of DCS in divers.[2,3]

The question of the link between the hydration status and the risk of DCS was raised 70 years ago in the context of altitude DCS[4], but the literature on this issue is still scarce and conflicting. Dehydration has been proposed as a risk factor in few DCS case reports.[5,6] However, in others, it has been proposed as a possible cause of bubble reduction after diving, both in the case of pre-dive exercise[7] and in experimental dehydration in rats.[8] On this point, animal studies find either no effect of dehydration on DCS in a murine model,[8–10] or an increased DCS occurrence in dehydrated rabbits and swine.[11,12] Interestingly, in a human study using infrared-ray dry sauna a reduction of circulating bubbles was associated with a moderate dehydration after a simulated dive.[13] There are, as far as we know, only three papers concerning pre-hydration. Two were performed on swine. One study failed to reduce neurological DCS after crystalloid infusion.[14] The other study was designed to evaluate the effect of methylprenidsolone on DCS, not to assess the effect of prehydration.[15] In that study an intravenous infusion of saline appeared to strongly reduce the occurrence of DCS and death, but only after comparison with a historical control group without a saline infusion. In humans, a study established that pre-hydration could reduce circulating bubbles after diving.[16]

In this context, there is a clear need to assess the influence of the hydration level on DCS occurrence after a dive. Consequently, in order to investigate the effect of pre-hydration on DCS occurrence intra-peritoneal (IP) injections of saline solution were adminstered to ratsprior to simulated air dives and we evaluated the occurrence and severity of DCS.

Table 1
Characteristics of the studied 15 male Sprague-Dawley rats. IP = intra-peritoneal

Parameter	Control	Group 1 IP injection 0.1 mL.100g^{-1}	Group 2 IP injection 1 mL.100g^{-1}	*P*-value
n	15	14	14	–
Age (weeks)	12	12	12	–
Weight (g) mean (SD)	410 (48)	442 (51)	423 (55)	> 0.05

Methods

ANIMALS

Animal experiments were conducted in accordance with the Guide for the Care and Use of Laboratory Animals published by the US National Institutes of Health (NIH Publication No. 85-23, revised 1996) and with the approval of the Université de Bretagne Occidentale Ethics Committee for Animal Experimentation (approval no. 01462.02). This study complies with recognized ethical standards and national/international laws.

Forty-three male Sprague-Dawley rats, 12 weeks old on the day of the experiment, were obtained from Janvier SAS (Le Genest St Isle, France). Animals were housed individually in a cage in an environmentally controlled room (temperature 21°C (SD 1), 12–12 h light-dark cycle) and were fed daily with 20–25 g of standard rat chow and water *ad libitum*. The animals were randomly assigned into three groups of 14 to 15 animals: air diving with no hydration (the animals were exposed to the simulated dive without treatment, Control); air diving with low hydration treatment (intra-peritoneal injection of NaCl 0.9% 0.1ml·100g^{-1} body mass at each of 24 h, 12 h, and 30 min before the simulated dive, Group 1); and air diving with high hydration treatment (intraperitoneal injection of NaCl 0.9% 1 ml·100g^{-1} body mass at each of 24 h, 12 h, and 30 min before the simulation, Group 2). Number, age, sex and weight of the rats in the three groups are given in Table 1. There was no significant difference in the weight of rats between groups.

DIVING PROTOCOL

The dive protocol applied in the present study is routinely used in the lab and is known to induce DCS in 63% (SD 4) of cases[17] (in rats of identical strain, age, sex and weight). Each rat was positioned in a 130 L steel hyperbaric chamber, always at the same hour to avoid interference by biological rhythms. Air was used as the breathing mixture. The animals were compressed at a rate of 100 kPa·min^{-1} to 1,000 kPa absolute pressure (90 metres' seawater [msw] equivalent) and remained at that pressure for 45 min (Figure 1). Decompression then followed at a rate of 101 kPa·min^{-1} with three decompression stops: 5 min at 202 kPa (10 msw), 5 min at 160 kPa (6 msw) and 10 min at 130 kPa (3 msw). Total hyperbaric exposure duration was 83 min.

DCS ASSESSMENT

Following hyperbaric exposure and decompression, the rats were observed for two hours for the appearance of four standard DCS symptoms: respiratory distress; walking difficulty; paralysis and/or convulsions. Animals were scored as having DCS only when one or more of these four symptoms appeared. The trinary classification of no DCS, mild DCS (one or more of the four symptoms but without death) and death was applied.

STATISTICS

The weight of rats was presented as mean (SD). The difference in the weight of rats between groups was evaluated with one way-ANOVA (Statistica 13.3) and considered significant when $P < 0.05$. The difference between the ratios of DCS morbidity among groups was analysed with a Chi-Square test (Statistica 13.3) and considered significant when $P < 0.05$.

Results

After the dive without pre-hydration, the percentage of severe and mild DCS occurrence were 47% and 13% respectively. In the same condition 40 % of the animal experienced no DCS after decompression. Hydration before the dive significantly reduced the proportion of severe DCS (to 29% and 0% in the groups receiving injections of 0.1 ml·100g^{-1} and 1 ml·100g^{-1} of saline respectively) and significantly increased the proportion of animals without any signs of DCS (to 57% and 93% in the same groups respectively; Chi^2 $P = 0.041$) (Figure 2).

Discussion

The goal of this study was to analyse the link between pre-hydration and DCS occurrence in a murine model. The results clearly show that in our conditions, intraperitoneal injection of saline solution of either 0.1 or 1 ml·100g^{-1} body mass 24 h, 12 h and 30 minutes before a simulated dive significantly reduced DCS occurrence from 60% to 7%. This

Figure 1
Dive protocol for *in vivo* diving simulations of rats

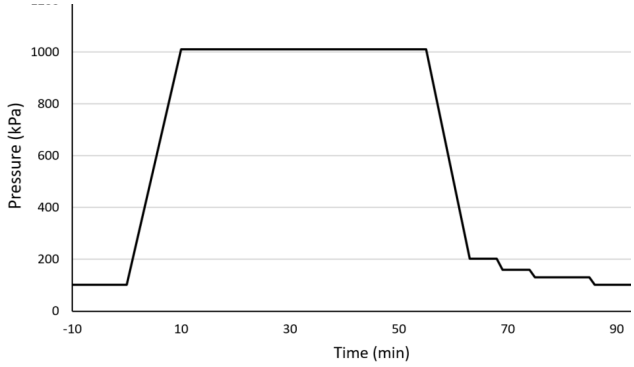
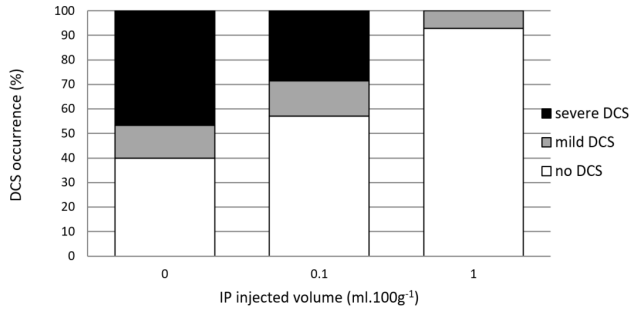


Figure 2
Morbidity of DCS in rats after *in vivo* simulated dives



result is consistent with the study in swine that suffered from the absence of a proper control group[15] and with the study in humans that did not assess DCS but used circulating bubbles as a proxy of decompression stress.[16] In that study, where decompression bubbles were measured after a 30-min dive at 30 msw, oral prehydration (with 1,300 ml of isosmotic saline-glucose beverage) significantly reduced venous bubbles when compared to a control group.[16]

Three main mechanisms are proposed in the literature to explain the effect of the hydration status on the risk of DCS.

1. *Surface tension (ST):* low ST is known to facilitate the formation of bubbles and dehydration may decrease ST. But in the case of pre-dive hydration, no change in plasmatic ST was observed by Gempp et al.[16] while circulating bubbles were reduced.

2. *Vasoconstriction:* large fluid intake can lead to gastric distention, peripheral sympathetic mediated vasoconstriction and consequently reduce the inert gas intake during the dive.[2] However, in the present experiment pre-hydration was performed via intraperitoneal injection; therefore this hypothesis is unlikely.

3. *Prevention of hypovolaemia:* scuba diving is known to induce hypovolaemia, and potentially, reduction of tissue microperfusion, inert gas removal and an increased risk of DCS.[11] The hypovolaemia is a consequence of immersion and not of hyperbaric exposure. Since the animals were exposed to pressure in a hyperbaric chamber, this hypothesis is an inadequate explanation of results in the present experiment.

Unfortunately, this experiment was designed only to evaluate DCS occurrence and mortality after the dive. The high percentage of dead animals in the control group prevented any biological analyses from being performed and did not allow exploration of the mechanism of a reduction of DCS after pre-hydration. Nevertheless, in the human experiment,[16] pre-dive hydration caused reduction in circulating bubble formation, and it is plausible that a reduction in bubble formation could explain the protective effect of pre-hydration on rats. It must be acknowledged that notwithstanding these results, there has been no demonstration of an effect of pre-hydration on DCS risk *per se* in human subjects. Furthermore, the question of the scalability of the fluid loading in humans is relevant since excessive fluid loading in divers must be avoided as it could lead to an increased risk of immersion pulmonary oedema. However, from an experimental point of view, the modulation of DCS occurrence via the manipulation of the hydration status can be a very interesting tool to develop our understanding of this multifactorial disease.

Regarding the assessment of DCS, animals were observed by two operators with significant experience, and only unambiguous symptoms (i.e., respiratory distress, walking difficulty, paralysis, convulsions or death) were considered for determination of the presence of DCS. More ambiguous signs such as, for example, paresthesia, prostration or agitation were not used because of the risk of subjectivity. This method was used in our previous studies.[18,19] Although it may probably underestimate the incidence of DCS in our model, we think that it helps prevent biases resulting from subjectivity and, therefore, allows comparison between groups.

Conclusions

Pre-hydration significantly reduced DCS occurrence in a rat model. The result can only be extrapolated to human diving with caution due to uncertainty over scalability and since hyper-hydration in the latter setting could lead to an increased risk of immersion pulmonary oedema. This finding nevertheless identifies the potential importance of hydration status in DCS risk and highlights the need for further experiments to explore the underlying mechanisms of this effect.

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Case reports

Arterial gas embolism breathing compressed air in 1.2 metres of water

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Key words
Air embolism; Cerebral arterial gas embolism (CAGE); Diving; Pulmonary barotrauma

Abstract

(Hampson NB, Moon RE. Arterial gas embolism breathing compressed air in 1.2 metres of water. Diving and Hyperbaric Medicine. 2020 September 30;50(3):292–294. doi: 10.28920/dhm50.3.292-294. PMID: 32957133.)
Arterial gas embolism (AGE) may result when diving while breathing compressed gas and ascending rapidly or with a closed glottis. Pulmonary over-pressurisation can result in lung stretch injury with entry of bubbles into the pulmonary venous circulation and subsequently the systemic arterial circulation. We present the case of an individual who suffered AGE while breathing compressed air at 1.2 metres' fresh water (mfw) in a swimming pool and discuss the factors determining the depth at which this form of injury may occur. This case serves to underscore the fact that risk of AGE exists at shallow depths.

Introduction

A recognised complication of diving while breathing compressed gas is pulmonary barotrauma.[1] Pulmonary over-pressurisation and barotrauma takes many forms, ranging from pulmonary interstitial, mediastinal or subcutaneous emphysema to pneumothorax and arterial gas embolism (AGE).[2] AGE is believed to result from disruption of the pulmonary venous circulation and entry of gas into the vasculature.[1] Divers are often unaware of the depth at which such problems may occur, believing that limiting excursions to a relatively shallow depth removes all risk. The exact depth required for AGE to occur remains undefined. The purpose of this report is to present the case of an individual who developed AGE following a brief surface-supplied dive at 1.2 metres' fresh water (mfw) in the shallow area of a swimming pool. Additionally, we provide a discussion of the potential factors that contribute to the pathophysiology of pulmonary barotrauma (e.g., AGE) at shallow depths.

Case report

The patient was a 25-year old healthy, fit military aviator referred for treatment of AGE. On the day of his injury, he participated in a training exercise in a swimming pool. While wearing his flight suit, he first sat on the floor in the shallow area of the pool (120 cm water depth) and breathed compressed air for about one minute. The breathing gas was supplied by a compressor located on the pool deck which was connected by a hose to a regulator and then three additional feet of hose to his mouthpiece. When sitting on the bottom, the top of his head was just below the water's surface. This exercise was accomplished uneventfully. He then exited the pool and re-entered it, this time hanging upside down by his knees from the pool's edge, again breathing from his mouthpiece. He was head-down in this fashion for a total of approximately three minutes. To demonstrate how a regulator works, he was asked to move it vertically in the water, sensing less pressure when moved toward the surface and more pressure when it was moved deeper. He recalled moving the regulator to the bottom of the pool, disengaging his knees from the pool's edge and standing up. Total time in the water was estimated at four minutes. He felt entirely well during exit from the pool, then experienced the onset of vertigo while stowing his gear. This was associated with left temporal headache which progressed rapidly to bitemporal pain. He estimated onset of symptoms within five to ten minutes after exiting the pool. He was transported immediately to the base medical clinic where evaluation demonstrated "*unsteady Romberg test and mild difficulty with finger-to-nose testing*". High flow oxygen administration, intravenous fluids and 25-degree head-down positioning were initiated by the on-site medical staff, and the patient transported by ground to the emergency room of a hospital with a multiplace hyperbaric facility.

Upon arrival, history and physical examination were notable for complaints of headache over the top of his head self-rated at 7–8/10 and pressure in the left ear without evidence of otic barotrauma on examination, as well as "*positive Romberg testing with falling to the right*". With a working diagnosis of AGE, he was treated on a US Navy Treatment Table 6A

with full extensions. Upon completion, headache was rated at 1/10, sensation of ear pressure improved and dizziness described as only slight. Neurological examination was normal. The patient had a very slight sensation of dizziness for 48 hours, then experienced resolution and felt entirely well.

One day prior to the event, he had participated in an aircraft decompression exercise. He began breathing 100% oxygen by face mask on the runway, flew to 15,000 feet altitude decompressed his aircraft to ambient, re-pressurized the aircraft to 8,000 feet, and ascended to 40,000 feet. He remained on oxygen throughout the flight and felt well afterward. The following morning, he awoke with the sensation of pressure in both ears, cleared them with a Valsalva manoeuvre and felt normal.

Subsequent evaluation included a normal chest radiograph and pulmonary function testing. The latter included plethysmographic measurement of lung volumes that demonstrated total lung capacity of 8.94 L (119% predicted), vital capacity 7.10 L (120% predicted), residual volume 1.85 L (105% predicted) and normal airways resistance. Other measurements included the distance from the patient's knees to his mouth (81 cm), pool deck to the surface of the water (20 cm) and pool deck to the bottom of the pool (140 cm).

Discussion

This individual is believed to have suffered cerebral AGE while surface-supplied diving at a depth of 1.2 mfw. The diagnosis is supported by his history, temporal onset of symptoms after emerging from the water, physical examination findings and response to recompression therapy. Other diagnoses to consider in the diver with acute neurological symptoms and signs can be effectively excluded on clinical grounds in this case. This was not decompression sickness due to the brief duration and shallow nature of the diving exposure. Inner ear barotrauma could cause dizziness and imbalance but would not have been expected to respond to recompression therapy. A transient ischaemic attack due to a thromboembolic event would be extremely unlikely in a healthy, fit military aviator of this age and, again, would not temporally respond to hyperbaric oxygen treatment.

His case is remarkably similar to the one reported by Benton in 1996, also a military aviator who developed AGE while undergoing training in a swimming pool.[3] In that case, the diver was limited in depth to one metre and was not inverted. He suffered multiple neurological symptoms immediately upon exiting he water, including upper extremity paraesthesias, subjective diplopia, and objective memory loss upon testing. He required repetitive hyperbaric treatment but eventually all symptoms and signs resolved. Subsequent pulmonary function testing and thoracic imaging were normal and he was cleared to return to flying.

Whether lung rupture occurs during diving with compressed gas breathing depends upon several factors, including lung compliance, transpulmonary pressure and lung volume.[4] Some small degree of pulmonary over-pressurisation can be accommodated by lung expansion, diaphragmatic inversion and compression of the heart and intrathoracic veins.[5] Lung rupture occurs when pulmonary parenchyma is stretched beyond its limits and is subsequently torn by over-pressurisation.

In experiments involving decompression of dogs from 100–200 feet' seawater over 60–90 seconds with the trachea closed, the animals developed pulmonary interstitial emphysema and AGE when the intratracheal pressure reached a critical value of approximately 10.7 kPa (80 mmHg) or a transpulmonic (intratracheal minus intrapleural) pressure of 8.0–9.3 kPa (60–70 mmHg).[6] In a classic 1961 article, two fresh human cadavers aged 47 and 64 years were demonstrated to develop pulmonary barotrauma when the lungs were pressurised to intratracheal pressures of 9.7 and 10.7 kPa (73 and 80 mmHg) while the thorax was unbound.[2]

What is required to achieve these pressures while diving? If the intra-alveolar pressure were 0 mmHg and the respiratory system compliance 0 ml·mmHg^{-1} at total lung capacity (TLC), this pressure could be achieved at sea level by adding one-tenth of an atmosphere absolute (10.1 kPa [76 mmHg]) to the system, an equivalent depth underwater of one metre. As such, this is the depth commonly proposed as the lower limit at which AGE can occur.[1,4,7]

However, during breath holding with closed glottis at TLC, intra-alveolar pressure is already elevated due to elastic recoil of the lungs and chest wall. When measured in 14 subjects, the average intra-airway pressure was 2.8 kPa (21 mmHg) above ambient pressure at TLC.[8] The same measurement was made in a group of ten healthy young adults and an average of approximately 4.3 kPa (32 mmHg) was seen.[9] If pressure was the only determinant of pulmonary rupture, these data suggest it may be possible for AGE to occur at a minimal depth; even less than one metre in the case of a diver who ascends with full inspiration and closed glottis.

The volume of gas necessary to consistently cause demonstrable lung stretch injury has been assessed in a group of breath-hold divers. Evidence was found of pulmonary barotrauma (mediastinal emphysema on computerised tomography) in each of five divers proficient at adding gas volume to their vital capacity (VC) through gastroesophageal insufflation (GI).[10] Prior to imaging they used GI to add an average of 1,400 ml (26%) to their VC. As the VC represents approximately 80% of TLC, it is apparent that a 20% increase in TLC (0.8 x 26%) may be sufficient to cause lung stretch injury, suggesting a maximal depth limit of two meters for predictable lung injury to occur.

These calculations assume that the lung is a homogeneous structure with uniform compliance throughout. However, studies of regional ventilation show significant heterogeneity which most likely indicates variability in lung compliance and airway resistance.[11] Such heterogeneity could be caused by prior local infection or exposure to external irritants or toxins. There are instances of similar changes in transpulmonary pressure causing AGE, often in individuals with pre-existing lung pathology and changing altitude.[12]

In this case, the diver was breathing from the regulator while inverted and head-down with his head near the bottom of the pool. Although he recalls moving the regulator to the pool floor before surfacing, it is not known whether he inspired to TLC prior to disengaging his knees from the pool edge and standing up. If he did, it is possible that immediately before surfacing his lungs were 'pre-stretched' due to increased airway pressure resulting from elastic recoil, as well as a positive static lung load resulting from his regulator being situated at a deeper depth than his lungs.

In summary, the depth at which a diver breathing compressed gas is at risk for pulmonary barotrauma is somewhat individual and not simply based upon intrapulmonary pressure. It also depends upon the degree of inspiration prior to breath-hold ascent, as well as heterogeneity of the lung and how comparable a healthy diver's lung tissues are to those of canine models and middle-aged cadavers. Under certain circumstances, the minimum depth at which there is the possibility of pulmonary barotrauma resulting in AGE may even be less than the one metre quoted.

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Dysbaric osteonecrosis in technical divers: The new 'at-risk' group?

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Abstract
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Introduction: Dysbaric osteonecrosis (DON) in people working under increased atmospheric pressure is well documented. It is generally less common in military and commercial divers than in caisson workers, except in some high-risk groups, such as in many indigenous diving industries where workers have little or no understanding of decompression principles. With the increasing popularity within the recreational diving community of deep air and mixed-gas decompression diving ('technical diving'), it is likely that diving physicians may see an increase in the prevalence of DON in this group in the future.
Methods: The case report is presented of a technical diving instructor, with a 30-year history of deep diving, who developed bilateral humeral head DON and required a right shoulder hemi-arthroplasty. A focused literature search was also undertaken to identify published cases of DON in recreational divers.
Results: The frequency, duration and depth of exposure to pressure, inadequate decompression, the occurrence of DCS and increasing age have been common features associated with DON in both divers and caisson workers. Many of these features were present in this technical diver.
Conclusions: Whilst DON is uncommon in recreational air scuba divers, all the above risk factors are present to a greater degree in technical diving. It is suggested that medical review for DON is merited from time to time in this potentially high-risk group of recreational divers.

Introduction

Aseptic bone necrosis (AVN) is the final common pathway of various conditions leading to bone death,[1] most commonly long-term, high-dose steroid use and alcoholism. Other contributing conditions include pancreatitis, lupus, sickle cell disease, Gaucher's disease, radiotherapy, trauma, pregnancy and thrombotic conditions.[1,2] A recent review paper discusses the diagnosis and staging of AVN of the femoral head.[3] AVN in caisson workers and divers, termed dysbaric osteonecrosis (DON), is well documented. In the 1970s, the Medical Research Council (MRC) Decompression Sickness Panel reported an incidence of 19% in caisson workers and reported a link to decompression sickness (DCS).[4,5] It is reported to be less common in military[6] and commercial divers[7,8] except in some high-risk groups[9] and those with little or no understanding of decompression principles.[10] Readers are referred to a useful review of DON in professional divers.[11] Lesions may be either juxta-articular (Type A, Table 1) or in the shaft of long bones (Type B), the former being far more likely to produce symptoms.

DON is considered rare in recreational divers, though individual cases have been reported.[12–15] With the increasing popularity within the recreational diving community of deep mixed-gas decompression diving ('technical diving'), it is likely that diving physicians may see an increase in the prevalence of DON in this group in the future because of the nature of their diving activities. For this reason, we present an illustrative case of a technical diving instructor with DON and briefly review the relevant literature.

Literature review

PubMed, the Rubicon Foundation Research Repository (http://archive.rubicon-foundation.org/xmlui/), the complete collection of the *South Pacific Underwater Medicine Society Journal* and *Diving and Hyperbaric Medicine* journal and major textbooks on diving medicine were searched using the terms avascular necrosis, dysbaric osteonecrosis, and caisson disease and in combination with the terms diving, decompression sickness, scuba and technical diving. Further articles of potential interest from reference lists were also reviewed. The intention was not to perform a comprehensive

Table 1
Classification of dysbaric osteonecrosis lesions (after the United Kingdon Medical Research Council). * = classification of the present case

Lesion	Subtype	Comments
A type lesions Juxta-articular	A1 Dense area with intact articular cortex	Prevalence of A lesions: Tunnellers and saturation divers Femur > Humerus Other divers Humerus > Femur
	A2 Spherical opacities	
	A3 Linear opacities	
	A4 Structural failures - Translucent subcortical bands - Collapse of articular cortex - Sequestration of cortex*	
	A5 Secondary degenerative osteoarthritis	
B type lesions Shaft	B1 Dense areas	n/a
	B2 Irregular calcified areas	
	B3 Translucent and cystic areas	

literature search, but to focus on published evidence relevant to recreational and technical diving.

Case report

A man in his 40s had been actively involved in recreational diving since 1991. He qualified as a dive instructor in 1994, teaching a mix of recreational and technical scuba diving around the world, doing 200 or more hours a year in the water. During this period, technical mixed gas diving took up about a quarter of his diving hours, but no detailed records of his diving were kept. Between 2010 and 2018, he recorded on his annual medical questionnaires over 1,800 dives of which 80% were to depths greater than 30 metres' sea water (msw) and over 1,500 were on mixed gas/trimix using closed-circuit rebreathers.

In 2002, he did a wreck dive to 115 msw using trimix, followed by a gas switch to air at 30 msw during the ascent. At the 9 msw decompression stop, he developed severe vertigo which persisted post dive. Audiometry was normal. A diagnosis of inner-ear decompression sickness (DCS) was made and he underwent a RNZN heliox Table 1A (similar to a Comex 30 treatment table) with a good response. Following two further short hyperbaric oxygen treatments he was symptom-free. Two years later in 2004, he dived on the same wreck to a depth of 120 msw. Again, a gas switch to air was made, this time at 40 msw. On reaching the 15 msw stop, he developed severe vertigo, vomiting and tinnitus. Because symptoms had improved at 6 msw after switching to 100% oxygen, he did not present for assessment until the following day when examination revealed a fine right nystagmus, very poor sharpened Romberg test and a 70 dB hearing loss at 8 KHz in the right ear. The diagnostic difficulty of differentiating between inner-ear DCS and barotrauma necessitated a cautious approach to recompression on a Royal Navy Treatment Table 62; however, his symptoms responded well. Again, after two further short hyperbaric oxygen treatments, he was symptom-free.

In 2011, aged 42 years, he developed pain in the left shoulder. Given his diving history, he underwent a long-bone X-ray survey which showed "*a localised lucency in the left humeral head with a surrounding sclerotic rim, the right shoulder was normal in appearance and there were faint, slightly serpiginous-appearing sclerotic lesions in the proximal left and distal right femoral shafts.*" In 2013, he was seen by an orthopaedic surgeon (BC) for assessment of the left shoulder lesion, which was managed conservatively at that time. No other risk factors for DON other than diving were identified.

In late 2018, now 49-years old, he presented following minor trauma to his right shoulder, with significant pain and limitation of movement associated with crepitus. Plain X-ray (Figure 1) and magnetic resonance imaging (MRI) (Figure 2) of the right shoulder showed a juxta-articular DON lesion of the humeral head measuring 8 mm by 24 mm with fragmentation of the articular surface and disruption of the articular cartilage. Given his symptoms were impeding his ability to work, he proceeded to surgery, at which time the articular surface of the right humerus had a 25 mm by 20 mm unstable osteochondral fragment in the central head. A right shoulder pyrocarbon hemiarthroplasty of the humeral head was inserted.

Recovery was uneventful with steady return of strength and range of motion such that he was cleared to return to work as a diving instructor four months post-operatively. For the hemiarthroplasty, there is little increased risk of aseptic loosening above what is already expected for patients under the age of 55. These patients have approximately double the failure rate of over 55-year olds.[16] The prognosis for the opposite shoulder is for gradual deterioration in symptoms over time due to the presence of DON in the humeral head with subchondral fracturing and collapse of the joint surface. This will eventually lead to arthritis or displacement of the fragmentation of the osteonecrotic segment, resulting in arthroplasty when the symptoms warrant.

Figure 1
Plain X-ray of the right shoulder of a recreational and technical diving instructor showing an avascular lesion of the humeral head

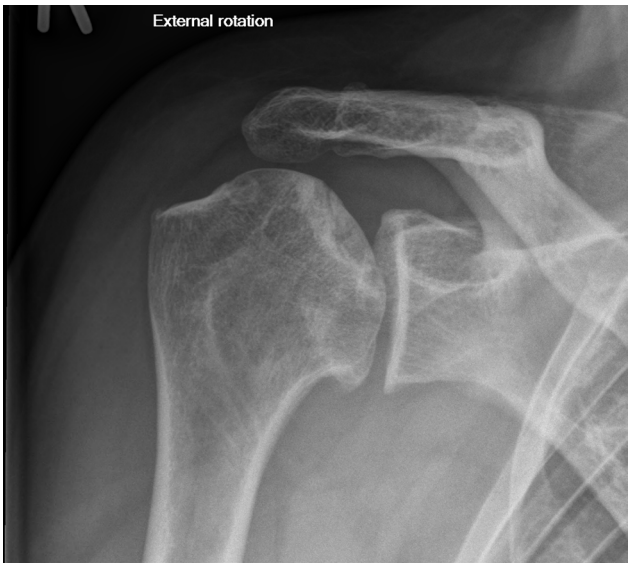
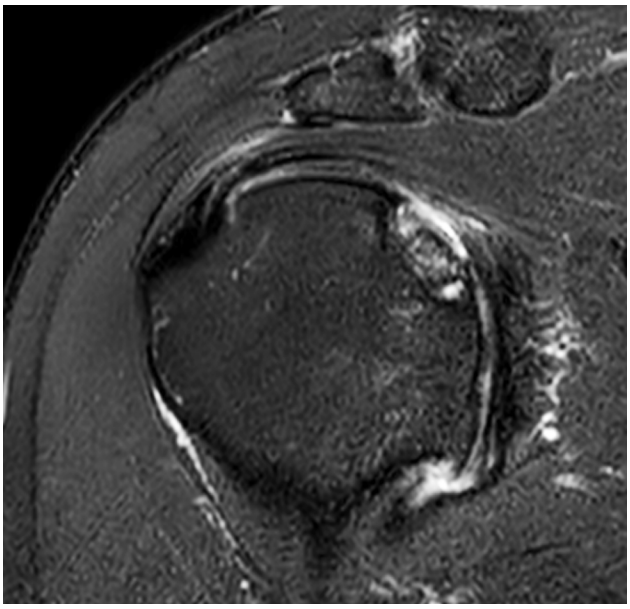


Figure 2
Coronal t2-weighted magnetic resonance image of the right shoulder of a recreational and technical diving instructor showing an avascular lesion of the humeral head



Discussion

AVN secondary to DCS was first noted in the late nineteenth century, culminating in work such as that of the MRC Decompression Sickness Panel in the 1960s and 1970s,[4,5,7] and later studies in China[17] and elsewhere. A high incidence of DON has been confirmed in high-risk diving groups such as long-term Japanese commercial divers,[9] and Hawaii's diving fishermen.[10] The United Kingdom DCS Central Registry and Radiological Panel determined that the risk of DON in caisson workers and commercial divers was directly related to the degree of pressure and the number of exposures,[5,7] whilst in Japanese commercial divers, diving exposure over 17–25 years was associated with DON, particularly in those diving to 35 msw or deeper and where there was a history of DCS.[9] In the majority of reports, juxta-articular lesions appear to be more common in the humeral head than in the femoral head of divers,[7,8] though this was not so in one study of 450 'hard-hat' Japanese divers in which the distribution was similar for both sites.[9] The prevalence of DON in divers in these various studies shows widely differing rates.

Thus, the frequency, duration and (deeper) depth of pressure exposure, inadequate decompression, the occurrence of DCS (and possibly delayed recompression) and increasing age have been common features associated with DON in both divers and caisson workers.[4–9,17] Whether age is, in fact, an independent risk factor or secondary to the length of the pressurisation/diving career is debatable. Although inert gas embolism is thought to be a mechanism linked to DON,[18] there is no clear evidence of a cause-and-effect relationship,[19,20] this more likely being a gradual process over many dives. Other mechanisms, such as a hypercoagulable state and fat emboli, also may be involved.[21] No certain aetiology for DON has been established.

DON is uncommon in typical recreational diving. The first report of dysbaric osteonecrosis lesions in sports divers was in an Australian study of 110 navy, professional and sport divers.[22] There were three lesions (one juxta-articular) in the 19 sport divers in the study. In the single cases reported,[12–15] in one,[15] subsequent enquiry from the author (Laden G, personal communication, 2019) revealed that this diver was a technical diver with several hundred deep and mixed-gas dives; in another,[13] the diver had performed 190 dives over six years, over 100 having been to depths greater than 30 msw. By contrast, in a third,[12] only no-decompression air diving had been undertaken, mostly to less than 18 msw. None had had symptoms suggesting DCS. Nevertheless, single events of DCS, especially if there was a delay to recompression treatment, appear to be associated with an increased risk of DON even in recreational divers.[23,24]

In a long-bone survey of 56 Turkish dive instructors each of whom had performed in excess of 500 dives, DON lesions were detected in 14, with only one juxta-articular lesion in the humeral head.[25] On univariate analysis of a wide range of factors, only increasing age was associated with DON and this, in itself, was correlated with diving experience and the total numbers of dives performed.

For occupational divers, long bone surveys are "*optional and as medically indicated*" in most jurisdictions.[26] In the Australian/New Zealand Standard,[27] juxta-articular DON is not considered to be a contraindication to continued

diving. Under these regulations, this dive instructor with known DON was cleared to dive following the condition and risks associated with continued diving having been fully discussed and understood, and a document signed by the diver to that effect.

Several features of the present case – many years of deep and decompression diving resulting in a higher likelihood of DCS and the possible role of gas switches in inducing a gas phase in sensitive tissues such as the inner ear[28] or bone – illustrate why technical divers are more likely to be at risk of DON than the average recreational diver. Therefore, we believe that recreational technical divers need greater regular medical screening than open-circuit air scuba divers. Early radiological referral in the presence of joint symptoms which might suggest a juxta-articular DON lesion is warranted, as in this case.

Reporting an amateur scuba diver who developed DON, the authors state "*avascular bone necrosis … may lead to joint dysfunction and lifelong disability*".[15] Modern orthopaedic surgery for shoulder and hip joint pathology now allows a range of treatment options, including hemi- and total arthroplasty for patients with juxta-articular DON.[29] Joint preserving surgery for osteonecrosis of the shoulder includes core decompression, bone grafting or autologous bone marrow grafting. Core decompression can be effective in early stages of the disease when there is no collapse of the humeral head but is less effective once the humeral head shows signs of collapse.[30] Bone grafting, either simple autologous grafting, strut bone graft or vascularised bone graft are complex procedures with increased morbidity and variable results.[31,32] It remains unclear as to the most effective joint sparing treatment of osteonecrosis. The natural history of conservatively treated advanced osteonecrosis of the humeral head is poor. Once the condition results in structural damage (equivalent of stage A4, Table 1), almost half will require shoulder arthroplasty.[33]

Hyperbaric oxygen treatment (HBOT) has been reported in several studies to show long-term benefit for early (equivalent to stages A1–A3; Table 1) femoral head AVN. In one double-blind, randomised study of 20 patients, the HBOT group showed a significant reduction in pain at the end of 30 treatments ($P < 0.001$) compared with the sham air group who were then offered HBOT, which they all accepted.[34] At seven years' follow up of 17 of the 20, "*all patients remained substantially pain-free … with none requiring hip arthroplasty. Substantial radiographic healing … was observed in seven of nine hips* [on MRI]."[34]

The medicolegal aspects of DON in working divers in the Tasmanian fish farming and abalone industries were discussed at a conference in Hobart in 1988 with reference to a new Workers' Compensation Act that came into force that year in Australia.[35] This Act recognised DON in Schedule 4 as "*compressed air illness including avascular necrosis caused by any work involving exposure to increased or reduced atmospheric pressure from working underground or underwater or from working at high altitudes*". Similarly, this diving instructor's DON was approved for treatment under the Accident Compensation Corporation (ACC) in New Zealand. Whether the ACC would recognise DON in a recreational diver who was not an 'employed' diver remains a moot point.

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Cerebral arterial gas embolism proven by computed tomography following transthoracic echocardiography using bubble contrast

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Key words
Central nervous system; Stroke; Doppler; Persistent (patent) foramen ovale (PFO); Radiological imaging; Hyperbaric oxygen treatment; Case reports

Abstract

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A 75 year-old male developed features of an acute stroke following bubble contrast echocardiography, which was shown on emergent computed tomography scanning to be a result of cerebral arterial gas embolism (CAGE) to the left middle cerebral artery. Ischaemic stroke symptoms have previously been reported as a rare complication of bubble contrast echocardiography. Radiologically proven CAGE from bubble contrast echocardiography had not been reported at the time this case occurred. Immediate provision of 100% oxygen and administration of hyperbaric oxygen are recommended treatments for CAGE and were associated with a substantial recovery for this patient.

Introduction

Bubble contrast echocardiography (BCE) is a common investigation performed to determine the presence of a persistent (patent) foramen ovale (PFO) in patients with cryptogenic stroke, decompression sickness (cutaneous, neurological and inner ear) and platypnoea-orthodeoxia syndrome.[1] Complications are rare, and radiologically proven cerebral arterial gas embolism (CAGE) from BCE had not been reported at the time this case occurred.

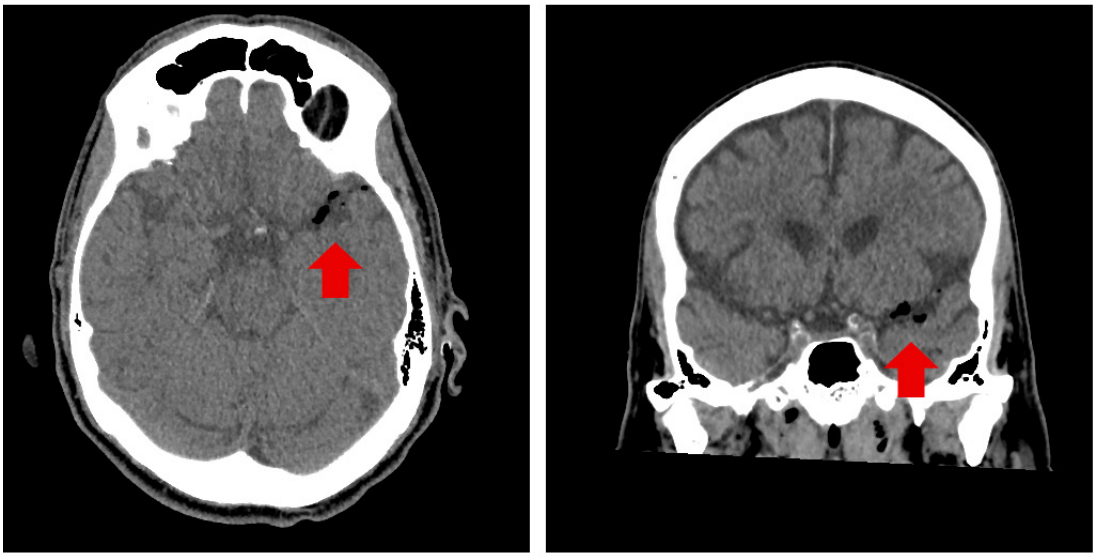
Case report

In January 2016, a 75-year-old male developed sudden onset right hemiparesis, dysarthria and dysphasia 50 minutes after a transthoracic echocardiogram (TTE) study using agitated saline contrast. The procedure had been requested by his general practitioner to investigate a small symptomatic right frontal cortical infarct. The procedure was performed as per the usual protocol in a private cardiology testing practice which had performed this procedure many thousands of times without incident over more than 30 years. Intravenous access in this case was via a vein on the dorsum of the hand rather than the antecubital fossa. The BCE confirmed a persistent (patent) foramen ovale (PFO), with trivial bubbles at rest (1–2 per frame) and minor shunting (~20 bubbles) post Valsalva release. Only four injections were administered via a 3-way tap with a total of 2 ml of air agitated into saline.

Approximately 50 minutes post procedure, the patient stood up while being connected to Holter monitoring and immediately became aphasic with right-sided weakness. An urgent computed tomography (CT) scan of the brain was performed at Sir Charles Gairdner Hospital, a nearby tertiary stroke referral centre, which demonstrated intravascular gas along the length of his left middle cerebral artery (MCA) as well as its cortical branches (Figure 1). His initial NIH Stroke scale was 18 and he had a modified Rankin score of 4. He was transferred to Fiona Stanley Hospital (FSH) for emergency hyperbaric oxygen treatment (HBOT) according to a US Navy Table 6 which was commenced within 4.5 hours of symptom onset. A repeat CT brain and CT angiogram an hour post HBOT showed resolution of air emboli with interval loss of the grey white differentiation in the left MCA territory in keeping with developing infarction. He continued to have some residual speech impairment and motor weakness.

The patient sustained moderate aural barotrauma during his first session of HBOT and hence bilateral myingotomies and grommet insertions were performed. Following this he received a further eight sessions of HBOT until there was a plateau of symptom recovery.

Figure 1
Non-contrast CT head (axial and coronal views) demonstrating extensive gas in the left MCA vessels consistent with air embolism



Following an inpatient stay in the acute stroke unit the patient was discharged to rehabilitation and recovered aside from mild language deficits.

Discussion

CAGE is an uncommon and probably under-recognised complication of invasive medical procedures. Although ischaemic neurological events have been reported post BCE, none of these cases demonstrated intravascular air on imaging.[2,3] It is well recognised that not all cases of CAGE show gas on brain imaging.[4,5]

Although microbubbles entering the arterial circulation almost inevitably pass to the cerebral circulation during BCE, they rarely cause symptoms because of their small size. This contrasts with the likely growth of similar sized arterial bubbles entering tissues supersaturated with inert gas in recently surfaced divers.[6] In this case, it is possible that usually harmless microbubbles generated in preparation of bubble contrast may have coalesced into a large bubble in forearm veins post-procedure and migrated to the central circulation, across the PFO and then to the brain when the patient stood up; possibly augmented by upper limb muscle contraction when used to assist this change in position. Presumably, aggregation of microbubbles is less likely to occur if the injection is into a proximal (antecubital) vein, which is not always accessible. This aggregation and sequestration of microbubbles, with fewer therefore available to proceed centrally and be seen during the BCE study itself, suggests that the PFO was actually larger than the contrast echocardiogram demonstrated.

Improved clinical outcomes after CAGE may correlate with reduced time to HBOT.[7–9] This patient had some delay to HBOT as ambulance transfers were required; initially to Sir Charles Gairdner Hospital, and then (when CAGE was diagnosed as the cause of his acute stroke) to Fiona Stanley Hospital, the only centre providing HBOT in Western Australia.

This case was initially presented at the World Stroke Congress in 2016 and published as an abstract in the related Proceedings.[10] Since that time a further case has been reported; that of an 89 year old woman who had a large right-sided CT-proven CAGE with symptom onset 20 minutes post procedure. This patient succumbed to her illness the day after without active treatment. HBOT was not administered.[11] The role of investigation for PFO in cryptogenic stroke in patients over 60 years of age is still to be defined.[12]

Current CAGE treatment guidelines recommend immediate cessation of further entry of gas, 100% oxygen and appropriate resuscitation of the patient as CAGE can lead to haemodynamic instability and cardiac arrest.[4] Early HBOT should be provided in a centre equipped to manage potentially unstable patients.

Conclusion

Iatrogenic CAGE causing stroke is a very rare complication of BCE. It should be considered where there is onset of neurological symptoms following this procedure, or any other where the possibility of introduction of intravascular gas exists. Emergent HBOT is indicated.

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Hyperbaric oxygen treatment in a patient with Guillain-Barré syndrome receiving mechanical ventilation

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Key words
Spinal cord injury; Neurology; Intensive care medicine; Case reports

Abstract

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The mortality rate of patients with Guillain-Barré syndrome (GBS) who develop respiratory muscle paralysis and need mechanical ventilation is increased. Though an unestablished indication, hyperbaric oxygen treatment (HBOT) has been used to treat patients with mild GBS who do not have respiratory muscle paralysis. The use of HBOT in severe cases has not been reported. We present a patient with severe GBS who received HBOT while ventilated in a multiplace hyperbaric chamber. Three courses of HBOT (one session per day, 10 sessions per course) were administered with a 2-day rest period between each course. The HBOT protocol was 40 minutes at 220 kPa with 25 minutes of compression and decompression. Following weeks of gradual deterioration, motor function improved after the first HBOT session. After eight HBOT sessions, the patient was successfully discontinued from mechanical ventilation and after 10 sessions the patient's muscle strength was significantly improved. After 30 HBOT sessions, the patient had normal breathing and speech, and did not cough when eating. Upper limb muscle strength was graded as 4 on the Medical Research Council (MRC) scale, lower limb muscle strength was graded as MRC 3. The patient was successfully discharged. Mechanically ventilated GBS patients may benefit from HBOT but studies are required to separate spontaneous recovery rates from treatment benefit.

Introduction

Guillain-Barré syndrome (GBS) is a monophasic, autoimmune polyneuropathy causing demyelination of the spinal nerve roots and peripheral nerves. It is characterised by progressive symmetrical muscle weakness of acute or subacute onset; commonly precipitated by an infection.[1,2] Patients with severe GBS may develop cranial nerve palsies, respiratory muscle paralysis, dysphagia, dysphonia and respiratory failure. There is no definitive therapy for GBS; while most patients have a favourable prognosis after active treatment, severe cases may die from respiratory muscle paralysis complicated by pneumonia. The use of hyperbaric oxygen treatment (HBOT) in patients with severe GBS with respiratory muscle paralysis requiring mechanical ventilation has not been reported. We report a woman with severe ventilator-dependent GBS who received HBOT with contemporaneous improvement of her symptoms such that mechanical ventilation could be discontinued, and she could be discharged.

Case report

A 24-year-old female was hospitalised because of limb weakness and numbness for two months and difficulty breathing for one month. The patient visited several hospitals without a diagnosis being made. Her symptoms had worsened, with muscle strength progressively decreasing, her voice becoming weak, onset of dysphagia and difficulty walking.

She initially presented to another local hospital where cerebrospinal fluid examination showed a protein of 0.59 $g \cdot L^{-1}$ (normal range 0.15–0.45 $g \cdot L^{-1}$), glucose of 3.85 $mmol \cdot L^{-1}$ (normal range 2.8–4.5 $mmol \cdot L^{-1}$), positive Pandy's reaction, white blood cell count of $2 \times 10^{6} \cdot L^{-1}$, and no red blood cells. After excluding other diseases, the patient was diagnosed with GBS and pneumonia, receiving a tracheotomy, mechanical ventilation and continuous intravenous infusion of gamma globulin for five days. She was also treated with hormone pulse therapy and administrated neurotrophic, anti-inflammatory and anti-tuberculosis medications. However, the patient's condition failed to improve, requiring continued mechanical ventilation from which she was unable to be weaned. After one month of treatment in the neurology department of the local hospital she was transferred to the Department of Neurology, the First Hospital of Jilin University, Peoples' Republic of China for further treatment.

On admission, the patient's temperature was 36.8°C, heart rate 112 beats·min^{-1}, and blood pressure 80/60 mmHg. The patient was alert but with no spontaneous breathing, and had received a tracheostomy. Scattered moist rales were heard in both lungs. Neurological examination showed that the patient was conscious, her tongue was midline when extended, bilateral pain and temperature sensation were normal and there were no signs of meningeal irritation. Her limb muscle strength was graded as zero on the Medical Research Council (MRC) scale, and tendon reflexes were absent. A lung CT scan revealed tuberculosis of the right upper lobe and both lower lobes, several calcified mediastinal lymph nodes and multiple enlarged lymph nodes in both axillae. Her admission diagnosis included GBS and pneumonia, with shock. After admission, the patient received continuous mechanical ventilation, and neurotrophic, anti-inflammatory, low-dose hormone and immunosuppressive medication. However, there was no significant improvement in limb muscle strength and she could not be weaned from mechanical ventilation.

On the tenth day of admission, the patient underwent HBOT with mechanical ventilation, accompanied by a hyperbaric physician. Three courses of HBOT were performed (one session per day, 10 HBOT sessions per course) with a two-day rest period between each course. Each session lasted 90 minutes with a treatment pressure of 220 kPa with 25 minutes of compression and decompression and inhalation of 100% oxygen for 40 minutes. After the first HBOT session, the patient was improved, with muscle strength in her upper limbs graded as one, and in the lower limbs graded as two. After five sessions, the patient was able to move her fingers and slightly move her neck and shoulder muscles; proximal lower limb muscle strength was graded as three, distal muscle strength was graded as two, and tendon reflexes were restored. After eight HBOT sessions, the patient was successfully discontinued from mechanical ventilation and after 10 sessions, the patient's muscle strength was significantly improved. As the patient's condition was significantly improved, she was able to be discharged. After discharge she had two further courses of HBOT as an outpatient. After the third course of HBOT, the patient had spontaneous normal breathing, normal voice, and did not cough when eating. Upper limb muscle strength was graded as four and lower limb muscle strength was graded as three. At one-month follow-up, the patient was able to take care of herself.

Discussion

Plasma exchange and immunoglobulin therapy are the proven effective treatments for GBS, no other treatments have been shown to be effective.[3,4] In our case, the patient had received treatment with plasma exchange, hormone pulse therapy, intravenous infusion of high-dose gamma globulin and administration of neurotrophic, anti-inflammatory drugs without improvement after one month of treatment, and mechanical ventilation could not be discontinued. The patient then received HBOT whilst ventilated with immediate signs of improvement; her respiratory and limb muscle strength began to recover gradually, enabling the patient to be discontinued from ventilatory support and eventually to be able to take care of herself.

HBOT increases blood oxygen concentration, produces vasoconstriction and inhibits neuro-oedema.[5] HBOT increases the diffusion distance of oxygen through the tissues, increasing tissue oxygen supply, improving the hypoxic state of nerves which may create a healing environment to improve the repair of injured nerves. Studies have shown that HBOT can promote axonal regeneration and facilitate nerve repair.[6–8] HBOT combined with methylprednisolone can promote facial nerve regeneration.[9]

This report shows that patients with severe GBS can receive HBOT while ventilated. There was a precise temporal relationship between commencement of HBOT and improvement in her clinical condition after one month of no improvement with other therapies. However, GBS has not been recognised as a proven indication for HBOT[10] and can only be considered an experimental indication as previously defined.[11] Many patients with GBS may recover spontaneously, so whether the improvement in patients with GBS is attributable to HBOT requires further investigation.

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Letters to the Editor

Central nervous system oxygen toxicity during 100% oxygen breathing at normobaric pressure

I read with interest the report of symptoms suggesting central nervous system (CNS) oxygen toxicity while breathing carbon dioxide (CO_2)/oxygen (O_2) mixtures at ambient pressure,[1] which Dr Eynan and colleagues concluded may have been due to normobaric CNS O_2 toxicity. While perhaps plausible, it should be noted that similar symptoms also consistent with O_2 toxicity have been reported from hypercapnia alone. These have included muscle twitching, facial tremors, myoclonus, extremity paralysis, hyporeflexia, flaccid paralysis, impaired consciousness and generalized convulsions.[2,3] In a study of normoxic, normal volunteers (inspired PO_2 21.3 kPa [0.21 atmospheres (atm)]) breathing CO_2 at 6.6–8.6 kPa (0.065–0.085 atm) in a dry hyperbaric chamber at 1.46 atm absolute, other symptoms typical of CNS oxygen toxicity were reported: tunnel vision, vision loss, dizziness and near-syncope.[4]

In their report, Eynan and colleagues point out the extreme sensitivity of a diver to CO_2. However, it is not clear that he is sensitive to O_2. Notwithstanding the rarity of this man's symptoms, rather than an uncommon manifestation of CNS O_2 toxicity, the diver's symptoms at sea level pressure may instead be a rare manifestation of CO_2 narcosis at relatively low PCO_2. The authors should test their hypothesis by exposing the diver in a blinded manner to CO_2 at low and high PO_2.

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Reply

I should like to thank Dr Richard Moon for his comments on our article.[1] I read with interest his letter regarding the possibility that the symptoms we reported may have been due to hypercapnia alone, because our subject was breathing 100% oxygen at normobaric pressure. He reached this conclusion on the basis of the article by Gill and colleagues,[2] in which symptoms akin to those associated with central nervous system (CNS) oxygen toxicity were detected when the inspired PCO_2 was between 6.6–8.6 kPa (0.065–0.085 atm).

I should like to raise two points, which may demonstrate that the diver in question is in fact highly sensitive to oxygen.

1. During his interview before the test, the subject complained of dizziness, headaches and nausea he had experienced during the series of dives using closed-circuit apparatus commenced two weeks previously. It is unlikely that he would have suffered from severe hypercapnia during his dives with the oxygen rebreather. It is more plausible that with no elevation at all of CO_2 in his inspired gas thanks to the CO_2 absorbent, the only aspect we have to consider in our attempt to determine the reason for his symptoms may be the hyperbaric oxygen he inspired during the dive.

2. When the CO_2 in the subject's inspired gas reached a level of 2 kPa during the CO_2 detection test, he complained of severe dizziness and headache. With CO_2 in excess of 3 kPa, he also reported twitching of his facial muscles, especially around the mouth. However, this level of inspired CO_2 is much lower than that reported by Gill and colleagues.[2]

These two points would indicate that our diver may have been extremely sensitive not only to CO_2, but also to hyperbaric oxygen.

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Annual update on the database of Randomised Controlled Trials in Diving and Hyperbaric Medicine

What is HBO Evidence?
http://hboevidence.wikis.unsw.edu.au/Home

A database of short critical appraisals (CATs) of all randomised controlled trials and systematic reviews in both diving and hyperbaric medicine. These appraisals are classified into disease categories and present the evidence in a standard way to enable rapid and meaningful comparisons between trials. This site is freely available to the public and professionals alike.

All enquiries: Professor Michael Bennett, Prince of Wales Clinical School, Sydney m.bennett@unsw.edu.au or follow the latest news on Twitter @gordoben

Current numbers: At the time of writing we have 317 completed CATs and 86 trials awaiting appraisal. Completed CATs:

• Neurology	61
• Radiation enhancement	43
• Problem chronic wounds	34
• Radiation tissue injury	27
• Acute ischaemia	25
• Diving medicine	21
• Audiovestibular	18
• Carbon monoxide poisoning	17
• Sports injuries	17
• Physiology and pharmacology	09
• Ophthalmology	08
• Thermal burns	07
• Fractures	07
• Necrotising infections	01
• Miscellaneous conditions	22

A selection of our latest CATs

- New evidence that HBO is effective for radiation cystitis (Oscarsson 2019).
- Further evidence that HBOT is effective in diabetic foot ulcer (Salama 2019).
- Pooled analysis suggests there is some justification for further studies into the use of HBOT for PTSD after mild head trauma (Hart 2019).
- Critical flicker fusion frequency testing may indicate cognitive decline in IGN (Lafere 2019).
- New evidence that unresolving athletic injuries may improve with HBOT (Chen 2019).

Things you can do: We need volunteers! I can train anyone to make new CATs for us, so why not have a go? Alternatively, we are always on the lookout for new trials or those we may have missed. If you know of any (human) RCTs in this area please contact us.

Michael Bennett, Prince of Wales Clinical School, University of New South Wales, Sydney, Australia
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Key words
Hyperbaric research; Diving research; Data; General interest

Notices and news

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

EUBS President's report

Ole Hyldegaard

EUBS meeting 2020

Since the first inaugural meeting of the European *Undersea Bio-medical* Society in London on 30 September 1971 (with the later change of our name to the European Underwater and Baromedical Society), there has been a regular Society annual scientific meeting over the last four decades since the meeting in Newcastle, UK in 1979. Sadly, and for the first time in many years, this year's meeting had to be postponed due to the COVID-19 pandemic, a health care crisis many of us are very acutely aware of. This year's EUBS 2020 Annual Scientific Meeting No. 46, was scheduled for Prague in the Czech Republic with Dr Michal Hàjek, President of the Czech Society of Hyperbaric and Aviation Medicine and Director of the Center of Hyperbaric Medicine, Municipal Hospital Ostrava, as Secretary General for the meeting.

In April/May this year the EUBS ExCom and the local organizers of the 2020 EUBS meeting were aware of the uncertain situation both, with respect to the development of the pandemic in the different regions of the European area as well as dynamically changing travel restrictions and area lock-downs caused by the ever fluctuating COVID-19 outbreaks. Accordingly, as was previously announced at the EUBS website – we agreed with the organizers to postpone the Prague meeting until 2021 and also postpone the scheduled EUBS 2021 meeting to be held in Portugal in the city of Porto and organized by Dr Oscar Camacho until 2022. This situation has obviously caused the need for the local organizers to change plans, rebook meeting facilities, change the arrangements of several other required meetings etc. On behalf of the EUBS ExCom and all members of the EUBS, I wish to express my sincere and heart-felt thanks for the local organizers of this years postponed Prague meeting and especially to Dr Hájek for being able to reorganize and reschedule his arrangements with a short notice. I also want to express my sincere appreciation for the flexibility shown by Dr Camacho and his team in Porto for being willing to reschedule until 2022 smoothly. Let us all hope that all countries will achieve a level of epidemic/pandemic control that will allow us to meet in Prague 2021. The EUBS society is not a large organization and therefore, our annual scientific meetings are very dependent on the personal interaction and contacts. Social interaction is important and the cancellation of this year's meeting was not an easy decision on more than just a scientific level.

Other great cities of the European continent are, luckily, also signed up for future meetings. The EUBS ExCom will interact and plan accordingly for EUBS meetings from 2023 and beyond, trying to fulfill as many of the meeting requests we have received as possible. It is comforting to know that many good colleagues are happy, willing and capable of holding the annual EUBS scientific meetings to come in the future.

The EUBS general assembly and 2020 arrangements

At the time of writing, our usual Annual Scientific pre-meeting for the EUBS ExCom is being organised. Although, we will not have an Annual Scientific Meeting this year we are obliged to ensure our proposal for changes, the Annual Fiscal Foundation report, costs for the Society and the costs for the Journal of *Diving and Hyperbaric Medicine* are all presented and voted for by EUBS members. At the moment we will have to decide on how this will take place because of the current situation and cancellation of the physical annual meeting. The EUBS ExCom will do what is necessary to display the required information, and its likely this will be via online voting present suggestions and our yearly reports for acceptance by the General Assembly EUBS members.

New EUBS Member-at-Large

As stated in the online EUBS news section, a new Member-at-Large for the EUBS ExCom has been elected. Dr med Rodrigue Pignel will leave office as Member-at-Large elected in 2017. Dr Óscar Ferraz Camacho, Medical Director of the Hyperbaric Medical Unit at the Hospital Pedro Hispano in Matosinhos, Portugal, will take his place in ExCom as the new Member-at-Large 2020. The EUBS ExCom extends their thanks to Rodrigue for his time and work performed in the ExCom, and we are happy he will continue this work for the EUBS including the ongoing and important updates of the Oxynet Database. Dr Pignel is applauded for his great work in the EUBS ExCom and his continued scientific contributions to field of high-pressure physiology and hyperbaric medicine is well known and highly acknowledged. Thank you Rod! Also thank you to Dr Óscar Ferraz Camacho for signing up and again a heart felt congratulations to Óscar and welcome aboard the EUBS ExCom.

Looking forward to normalized times and a great EUBS meeting in Prague 2021.

Ole Hyldegaard

EUBS Member-at-Large elections

Each year between June and August, EUBS membership elects a new Member-at-Large. Dr Med Rodrigue Pignel will leave office as Member-at-Large 2017 and Dr Óscar Ferraz Camacho, Medical Director of the Hyperbaric Medical Unit at the Hospital Pedro Hispano in Matosinhos, Portugal, will take his place in ExCom as the new Member-at-Large 2020. ExCom extends their thanks to Rodrigue for his work in the ExCom and we all hope (know) he will remain active in the society.

The new online voting process was well used with 55% of our members voting. However, further feedback is appreciated, send an email to secretary@eubs.org.

EUBS 2020 – postponed to 2021

Due to the COVID-19 pandemic, our 2020 Annual Scientific Meeting could not take place as planned in September 2020. It was decided by ExCom to postpone it and thus, our next years' EUBS Annual Scientific Meeting will take place in Prague, Czech Republic in September 2021 (exact dates to be confirmed).

The meeting will be organised by a local organising committee chaired by Michal Hajek, MD, Ph.D, a longtime member of EUBS, and member of Executive Board of ECHM; in collaboration with the Czech Society of Hyperbaric and Aviation Medicine, the City Hospital of Ostrava, the Faculty of Medicine of Ostrava University, the Faculty of Medicine of Charles University in Hradec Kralove, the Cochrane Institute Czech Republic, The Czech Republic (Middle European) Centre for Evidence-Based Healthcare: The Joanna Briggs Institute Centre of Excellence, the Masaryk University GRADE Centre, DAN Europe, and others.

Hyperbaric medicine has a long tradition in Czech Republic, in 2020 it was 55 years since that field of medicine in this country was established.

Prague is the capital and largest city in the Czech Republic, the forteenth largest city in the EU and the historical capital of Bohemia. The city is home to about 1.3 million people, while its metropolitan area is estimated to have a population of 2.6 million. Prague has been a political, cultural and economic centre of central Europe complete with a rich history. It was founded during the Romanesque and flourishing by the Gothic, Renaissance and Baroque eras. Prague was the capital of the kingdom of Bohemia and the main residence of several Holy Roman Emperors, most notably of Charles IV (1346–1378). It is located in the centre of the European continent, with direct air links with most European capitals and direct air connection from Frankfurt am Main, Germany, for connecting to overseas flights to other continents.

It is hoped and expected that by September 2021, 'real life meetings' will again be possible, as they provide the 'salt and pepper' of scientific work and allow direct, informal contacts in a relaxed atmosphere. So please keep September 2021 free for Prague.

EUBS 2020 Annual General Meeting

Normally, during the EUBS Annual Scientific Meeting, we also have our Annual General Meeting, where EUBS ExCom presents a report of their activities to the members and asks for approval of their decisions/proposals from the EUBS members. Unfortunately, this will not be possible to organise in 'live' conditions, because of the travel and meeting restrictions imposed by all European and world countries to combat the COVID-19 pandemic spread.

However, some decisions need to be approved by the EUBS members and therefore, ExCom is planning an 'online' General Assembly, to be organised by the end of September or beginning of October 2020. The exact format is (at the time of writing this text) still being organised, but it will allow online voting with respect to the proper procedures. A formal invitation with instructions will be sent to all EUBS members through the EUBS website newsletter and via social media (Twitter, Instagram, Facebook).

EUBS website

As always, please visit the EUBS website (http://www.eubs.org/) for the latest news and updates. Please renew your membership annually – each member will receive a personal renewal invitation one month before expiry; even if your membership has expired, you can easily renew it when trying to login again. Do not hesitate to contact the EUBS secretary if you have any difficulties secretary@eubs.org.

EUBS and OXYNET

Thanks to the efforts of Dr Rodrigue Pignel, our 2017 Member-at-Large, the OXYNET database of contact information for hyperbaric centres in Europe has been updated and will be made available through the EUBS and ECHM websites. This is an enormous amount of work, which is never finished as local/regional situations may change and so a mechanism for ensuring the information remains valid will be developed. This will of course require the cooperation of at least one EUBS member per country, so a call for volunteers will be issued in order to help us periodically verify addresses, telephone numbers and treatment capabilities of hyperbaric centres in your country.

Other

Occasionally, we can use the EUBS website newsletter as a tool to seek help for our members, as it is a perfect way to reach all of the EUBS members and because communication, networking and interaction are prime goals of our Society. A new page on our EUBS website has been created (EUBS Members Help Requests, under the 'Activities' menu on the homepage). Please check this page and try to help out. If you require help also and would like to use this service, please contact the webmaster (webmaster@eubs.org).You should also consult the page where research projects seeking collaborators and international participation are presented.

Level II Master course in Diving and Hyperbaric Medicine

Hyperbaric School of Padua, Italy (Director Prof Gerardo Bosco)

This Master course is aimed to provide theoretical and practical training to medical doctors and surgeons who want to deepen their knowledge in the field of hyperbaric medicine. The course offers a high level of specialist training: formal lessons, seminars and workshops given by international specialists and practical stages in hyperbaric medicine centres. The practical part also provides exercises in laboratories and research fields of physiopathology and diving medicine, with an accurate description of the instruments used. The Master's training objective follows the ECHM (European Committee for Hyperbaric Medicine) standards.

Career opportunities: Medical doctor in healthcare management and management of a hyperbaric oxygen therapeutic centre and/or a diving medicine centre.

For more information (Italian): http://tiny.cc/PadovaMaster

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 email. Once registered, watch on line, or download to your iPhone, iPad or computer for later viewing.

For further information contact via email:
folke.lind@karolinska.se

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

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

The Science of Diving

Support EUBS by buying the PHYPODE book 'The science of diving'. Written for anyone with an interest in the latest research in diving physiology and pathology. The royalties from this book are being donated to the EUBS.
Available from: Morebooks
https://www.morebooks.de/store/gb/book/the-science-of-diving/isbn/978-3-659-66233-1

The EUBS website is at
https://www.eubs.org/

Members are encouraged to log in and keep their personal details up to date.

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

EUBS Facebook page

Follow us for updates at:
https://www.facebook.com/European-Underwater-and-Baromedical-Society-283981285037017

Notices and news

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

SPUMS President's report

Neil Banham

This is my first report as the new President of SPUMS. I have big shoes to fill, following in the footsteps of our Immediate Past President Clinical Professor David Smart, and our outgoing Immediate Past President, Professor Mike Bennett. I am reassured that I will have David to guide me in this challenging role, and that I have an experienced and enthusiastic ExCom to manage SPUMS business in the next three years of my tenure. Mike Bennett has just finished serving for many years on the ExCom and for recognition of his service to SPUMS he was awarded Life-membership of our Society at our recent (virtual) AGM. Congratulations Mike!

Despite the recent challenges of Covid-19, SPUMS continues to remain a strong organisation with a sound financial position. The Impact Factor of our journal *Diving and Hyperbaric Medicine* continues to grow (now 1.5), remaining the pre-eminent journal in our field. I thank our hard working Editor Professor Simon Mitchell as well as contributors and reviewers for this excellent result. The work of Nicky Telles should not be forgotten, both for her work with the journal and for SPUMS itself.

Covid-19 has affected us all, and in particular causing conferences to be cancelled. It was most disappointing that our Annual Scientific Meeting in Tutukaka had to be cancelled. Many thanks to Convenor Dr Greg van der Hulst and his team for their fantastic effort in organising as well as their efforts post cancellation enabling most monies paid to be returned to registrants. Tutukaka will hopefully be able to host our ASM in 2021 – Covid-19 restrictions permitting. Covid-19 also led to the cancellation of the UHMS ASM in San Diego in June as well as the EUBS ASM to be held in September in Prague, now deferred until September next year. I had planned to attend all of these meetings in my role as SPUMS President but this will have to wait until at least 2021. Planning is also underway for options for the 50th SPUMS ASM in 2022, the venue to be determined by any Covid-19 related travel restrictions at the time and the availability of a vaccine.

Besides the journal and the opportunity to attend our ASM, SPUMS membership also entitles appropriately qualified members to be listed on the SPUMS Diving Doctors List and to be awarded the Diploma in Diving and Hyperbaric Medicine (DipDHM). The SPUMS website (https://www.spums.org.au) contains information regarding these, as well as the 5th edition of the SPUMS Diving Medical which contains updated guidelines on cardiovascular fitness for diving as discussed and agreed at the 2019 SPUMS ASM in Honiara, Solomon Islands. The SPUMS ExCom and in particular the Webmaster Joel Hissink are exploring ways to update this website and make it easier to renew membership. Any suggestions can be emailed to webmaster@spums.org.au. I encourage you to recommend SPUMS membership to your colleagues with an interest in diving and/or diving medicine.

Tasks for the next three years include documenting the history of the first 50 years of SPUMS, updating the website and continuing to promote diving and hyperbaric medicine.

In closing, I would again like to thank my friend and colleague David Smart for his efforts over the six years of his Presidency to maintain and grow our Society. We all owe David a great debt.

Stay safe!

Dr Neil Banham
President SPUMS

The SPUMS website is at **https://spums.org.au/**

Members are encouraged to log in and keep their personal details up to date.

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

SPUMS Facebook page

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

ANZHMG Chair report 2020

It is with great sadness that we heard of the recent passing of Dr John Orton. John was Medical Director of the Hyperbaric Medicine Unit in Townsville from 2011until his retirement in 2017. John was Chair of ANZHMG from 2014–2017 and Course Convenor of the ANZHMG Introductory Course in Diving and Hyperbaric Medicine in 2016–2017, as well as assisting in this role in 2015. John had happily retired to his vineyard in New Zealand.

The 2020 edition of the two week ANZHMG course was again held in Fremantle, Western Australia at a new venue, the Hougoumont Hotel between 24 February – 06 March. The course Convenor Dr Ian Gawthrope organised a highly successful course with 24 participants. Course activities outside of the classroom included a visit to the submarine escape facility at HMAS Stirling, a day focussed on diver retrieval at the Jandakot RFDS 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 Professor Simon Mitchell, Clinical Professor David Smart, Professor Mike Bennett, Dr Andrew Fock, Dr Ken Thistlethwaite, Dr John Lippmann, Dr David Wilkinson, Dr Peter Buzzacott and Dr Iestyn Lewis – many thanks to all who contributed.

The course prize was awarded to Dr Zach Tappenden.

The course returned a small surplus to SPUMS which will be in part used to fund deposits for the 2021 course, tentatively planned for 15–26 February 2021, again at the Hougoumont Hotel in Fremantle.

Dr Ken Thistlethwaite was instrumental in developing a process such that Amron hoods could be continued to be used to deliver HBOT to patients in Australia despite them not being TGA approved, this now simply involves getting consent from each patient and then advising the TGA of the total number each six months.

Dr David Cooper developed the ANZHMG/HTNA COVID-19 Guidelines, pertinent to hyperbaric medicine practice in Australasia and circulated them to all Australasian hyperbaric facilities. As this was developed as a 'living document', it has not been posted on the SPUMS website.

This will be my last report as Chair of the ANZHMG. I would like to thank the ANZHMG Secretary Dr Ken Thistlethwaite and the previous Chair and outgoing SPUMS President Dr David Smart for their invaluable assistance and advice during my three year tenure.

Dr Neil Banham
Chair ANZHMG

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.

Training
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.

Examination dates for 2021
Dates for the 2021 exam will be published late 2020.

Accreditation
The ANZCA Handbook for Advanced DHM accreditation, which provides information for units seeking accreditation, is awaiting approval by Standards Australia and cannot yet be accessed online. Currently six units are accredited for DHM training and these can be found on the College website.

Transition to new qualification
Transitional arrangements for holders of the ANZCA Certificate in Diving and Hyperbaric Medicine and highly experienced practitioners of DHM seeking recognition of prior experience lapsed on 31 January 2019.

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

Carl Edmonds Memorial Scholarship

The Australasian Diving Safety Foundation is delighted to announce the release of a new Diving Medical Officers Training scholarship to honour the memory of Carl Edmonds, a Founder of SPUMS and a mentor to diving physicians throughout the world. The AUD$5,000 scholarship is to encourage doctors to attend a Royal Australian Navy Underwater Medicine Course at the School of Underwater Medicine in Sydney. One scholarship is available for each course, two of which are planned for 2020.

Application details are available at:

https://www.adsf.org.au/r/diving-medical-training-scholarships

Royal Australian Navy Medical Officers' Underwater Medicine Course 2020

Venue: HMAS Penguin, Sydney

Date: Due to Covid travel restrictions we are planning on running a modified course (19–30 October 2020) for candidates in the NSW area.

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 contraindication to diving and the diving medical assessment, together with the pathophysiology, diagnosis and management of common diving-related illnesses. The course includes scenario-based simulation focusing on the management of diving emergencies and workshops covering the key components of the diving medical.

Cost: The course cost remains at AUD$1,355 (ex GST) but may increase to AUD$2,600 (ex GST).

For information and application forms contact:
Rajeev Karekar, for Officer in Charge
rajeev.karekar@defence.gov.au

Advertising in *Diving and Hyperbaric Medicine*

Commercial advertising is welcomed within the pages of *Diving and Hyperbaric Medicine*. Companies and organisations within the diving, hyperbaric medicine and wound-care communities who might wish to advertise their equipment and services are welcome. The advertising policy of the parent societies – EUBS and SPUMS – is available for download on *Diving and Hyperbaric Medicine* website.

Further information can be obtained by contacting the Editorial Assistant of *Diving and Hyperbaric Medicine*
Email: editiorialassist@dhmjournal.com

SPUMS Diploma in Diving and Hyperbaric Medicine

Requirements for candidates (May 2014)

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

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

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

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

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

Additional information – prospective approval of projects is required

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

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

It is expected that all research will be conducted in accordance with the joint NHMRC/AVCC statement and guidelines on research practice, available at: 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, Auckland

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

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

Courses and meetings

Capita Selecta Diving Medicine

The symposia of the Capita Selecta Diving Medicine of the University of Amsterdam will resume when the COVID-19-regulations of Academic Medical Centre of the University of Amsterdam allow this.

The symposium to celebrate the 50 year anniversary of the Dutch Stichting Duik Research (SDR, Foundation of Diving Research) originally scheduled in October is postponed until 2021. Dates are to be confirmed.

Visit: http://www.duikresearch.org/

For more information: n.a.schellart@amsterdamumc.nl

Baltic International Symposium on Diving and Hyperbaric Medicine – new dates

The 2nd Baltic International Symposium on Diving and Hyperbaric Medicine (BIS_on_DHM) will now take place in Gdynia, Poland, (**new dates**) from 10–12 December 2020. There will also be two satellite Masterclasses; one on Advanced Diving Medicine and the other one on Complications in HBOT with a possibility to participate in the fire drills inside the hyperbaric chamber, to get wet under pressure!

More information at: http://www.bisdhm.events/

DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA

P O Box 347, Dingley Village
Victoria, 3172, Australia
Email: hdsaustraliapacific@hotmail.com.au
Website: www.classicdiver.org

German Society for Diving and Hyperbaric Medicine (GTÜM)

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

Scott Haldane Foundation

As an institute dedicated to education in diving medicine, the Scott Haldane Foundation (SHF) has organized more than 295 courses all over the world, over the past 26 years. SHF is targeting a more international audience with courses world wide. Due to the COVID-19 Pandemic some courses are re-scheduled. Fortunately, we were able to find new dates for all courses in 2020. Below the upcoming SHF-courses in 2020 and early 2021.

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

2020

16–17 October	In-depth course Decompression, Recompression and HBOt (Level 2d) Hoeven, NL
28–29 October	Internship different types of diving (2d) Den Helder NL
27–28 November	28th In-depth course diving and mental health, location TBD

2021

19–20 March	Medical Examiner of Divers part 1 Zeist, NL
25–27 March	Medical Examiner of Divers part 2 Amsterdam Univ Med Centre, NL
10–07 April	Medical Examiner of Divers part 2 Bonaire, Dutch Caribbean
On request	Internship HBOt (level 2d certification) NL/Belgium

The course calendar will be supplemented regularly.

For the latest information visit:
https://www.scotthaldane.org

Please also check the COVID-19 News update on the website for the latest schedule changes.

Diving and Hyperbaric Medicine: Instructions for authors (summary)

Diving and Hyperbaric Medicine (DHM) is the combined journal of the South Pacific Underwater Medicine Society (SPUMS) and the European Underwater and Baromedical Society (EUBS). It seeks to publish papers of high quality on all aspects of diving and hyperbaric medicine of interest to diving medical professionals, physicians of all specialties, scientists, members of the diving and hyperbaric industries, and divers. Manuscripts must be offered exclusively to *Diving and Hyperbaric Medicine*, unless clearly authenticated copyright exemption accompaniesthe manuscript. All manuscripts will be subject to peer review. Accepted contributions will also be subject to editing.

Address: The Editor, Diving and Hyperbaric Medicine, Department of Anaesthesiology, University of Auckland, Private Bag 92019, Auckland 1142, New Zealand
Email: editor@dhmjournal.com
Phone: (mobile) +64 (0)27 4141 212
European Editor: euroeditor@dhmjournal.com
Editorial Assistant: editorialassist@dhmjournal.com
Journal information: info@dhmjournal.com

Contributions should be submitted electronically by following the link:
http://www.manuscriptmanager.net/dhm
There is on-screen help on the platform to assist authors as they assemble their submission. In order to submit, the corresponding author needs to create an 'account' with a 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 total word count); structure of the article and abstract is at the author(s)' discretion.

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

Educational articles, Commentaries and **Consensus reports** for occasional sections may vary in format and length, but should generally be a maximum of 2,000 words and 15 references (excluded from word count); include an informative **Abstract** of no more than 200 words (excluded from word count).

Letters to the Editor: maximum 600 words, plus one figure or table and five references.

Formatting of manuscripts

All submissions must comply with the following requirements. Manuscripts not complying with these instructions will be suspended and returned to the author for correction before consideration. Guidance on structure for the different types of articles is given above.

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

- Instructions for authors (full version)
- DHM Key words
- DHM Mandatory Submission Form 2020
- Trial design analysis and presentation
- EASE participation and conflict of interest statement
- English as a second language
- Guideline to authorship in DHM 2015
- Helsinki Declaration revised 2013
- Is ethics approval needed?

DIVER EMERGENCY SERVICES PHONE NUMBERS

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1800-088200 (in Australia toll free)
+61-3-7018 3076 (International)

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+64-9-445-8454 (International)

JAPAN – DAN
+81-3-3812-4999 (Japan)

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

UNITED KINGDOM
+44-7740-251-635

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

USA – DAN
+1-919-684-9111

ADSF
AUSTRALASIAN DIVING SAFETY FOUNDATION

Scholarships for Diving Medical Training for Doctors

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

There are two categories of scholarships:

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

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