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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

Our first issue in 2021 further exemplifies the recent increase in submissions and publication activity that began early in 2020 and has not lost momentum. DHM received 50% more submissions in 2020 than 2019. Many of the 10 original articles and nine case reports in the present issue were part of that surge. This has placed considerable pressure on editorial systems calibrated over many years to cope with a substantially lower workload, and for some papers it results in publication delays. Our general rule is that an accepted paper will not miss more than one published issue after acceptance.

This issue has a healthy mix of papers oriented to both diving and hyperbaric medicine. On the hyperbaric medicine side there is a systematic review of available evidence for the use of hyperbaric oxygen treatment (HBOT) in treating necrotising soft tissue infections (NSTIs) which provides a strong basis for claiming better outcomes where HBOT is used. Unfortunately, this is not a review of randomised trials because there are none. Nevertheless, if one applies the principle that the supportive evidence quality for a therapeutic intervention should match the prevalence of the problem being treated, then the observational nature of the available evidence for HBOT in treating NSTIs is essentially what one would expect. It would be extremely difficult to conduct randomised trials of HBOT in treating this very sporadic and catastrophic condition.

There is also a fascinating paper which explores patient experiences of HBOT over courses of treatment at units in Australia and the UK. This confirms the impression formed by many experienced hyperbaric practitioners that initial patient anxiety often gives way to something close to enjoyment of the unique HBOT experience and the socialising with other patients and staff that comes with it. These insights will help hyperbaric practitioners 'smooth' the patient journey through a course of hyperbaric care.

On the diving side there is a paper that represents the most comprehensive interrogation of the recreational diver community's experience of middle ear barotrauma yet published. Although confined to divers in Finland, the findings are likely to be widely applicable and, among other things, the paper identifies important risk factors and associations with recurrent middle ear barotrauma. This pairs nicely with a cadaver study from Turkey which measured tympanic membrane burst pressures thus providing a sense of a threshold pressure/depth at which serious middle ear barotrauma may occur.

There is an uncommonly large number of case reports or case series in this issue which also cover a wide range of topics in both diving and hyperbaric medicine. There is a harrowing but instructional account of a saturation diving fatality which illustrates the complexity of dealing with accidents occurring in the unique saturation environment. There is also a timely reminder that decompression sickness can occur in inside chamber attendants after routine hyperbaric exposures.

It is with great pleasure that I note one of our editorial board members (Associate Professor David Doolette) has been awarded a US Navy commendation medal for his services as a civilian scientist (the Department of the Navy Meritorious Civilian Service Award). The citation states that the award recognises "*significant contributions to the United States Navy while serving as a Senior Research Physiologist at the Navy Experimental Diving Unit (NEDU)*". Among other things, it also states "*As a widely recognized technical authority on diving physiology, procedures and equipment, you authored or co-authored numerous scientific works, which provided the expert basis for NEDU responses on thermal physiology, oxygen toxicity, decompression, and underwater breathing apparatus*". David also makes a tremendous contribution to the review process for this journal and we are extremely lucky to have his input. On behalf of the journal, I offer David our thanks for his support and congratulations on this prestigious award (the medal is pictured above).

The journal wishes members of both societies a prosperous and safe year in 2021. It is hoped that there may be light emerging at the end of the COVID-19 tunnel and that by 2022 at the latest we may see a return of our ability to meet, exchange ideas, and enjoy scientific discourse at our societal annual meetings.

Professor Simon Mitchell
Editor, Diving and Hyperbaric Medicine Journal

Front cover photo: The editor on a February 2021 dive at Serpent Rock, Poor Knights Islands, New Zealand. Photo taken by Pete Mesley at 70 m depth showing a giant tube sponge *Callyspongia latituba* (Dendy, 1924).

Original articles

Hyperbaric oxygen treatment for mastectomy flap ischaemia: A case series of 50 breasts

Nicole E Spruijt[1], Lisette T Hoekstra[1,2], Johan Wilmink[3], Maarten M Hoogbergen[1,4]

[1] *Da Vinci Clinic, Geldrop, the Netherlands*
[2] *Department of Plastic, Reconstructive and Hand Surgery, Maastricht UMC+, the Netherlands*
[3] *Department of Plastic, Reconstructive and Hand Surgery, Maxima Medical Center Eindhoven, the Netherlands*
[4] *Department of Plastic, Reconstructive and Hand Surgery, Catharina Hospital, Eindhoven, the Netherlands*

Corresponding author: *Dr Nicole E Spruijt, Da Vinci Clinic, Nieuwendijk 49, 5664HB Geldrop, the Netherlands*
n.spruijt@davincikliniek.com

Key words
Necrosis; Nipple; Outcome; Radiotherapy; Skin; Surgery

Abstract
(Spruijt NE, Hoekstra LT, Wilmink J, Hoogbergen MH. Hyperbaric oxygen treatment for mastectomy flap ischaemia: A case series of 50 breasts. Diving and Hyperbaric Medicine. 2021 March 31;51(1):2–9. doi: 10.28920/dhm51.1.2-9. PMID: 33761535.)

Introduction: Hyperbaric oxygen treatment (HBOT) has been suggested as an effective intervention to limit necrosis of ischaemic skin flaps after mastectomy. The purpose of this study was to evaluate outcomes of HBOT in the largest series of patients to date with mastectomy flap ischaemia.

Methods: A retrospective analysis was performed of 50 breasts requiring HBOT for mastectomy flap ischaemia. The severity of the ischaemia or necrosis was evaluated by four independent observers using the skin ischaemia necrosis (SKIN) score. Multivariate logistic regression analyses were used to assess associations between risk factors and re-operation.

Results: HBOT was started a median of 3 days (range 1–23) after surgery and continued for a median of 12 sessions (range 6–22). The breast SKIN surface area scores (n = 175 observations by the independent observers) improved in 34% (of observations) and the depth scores deteriorated in 42% (both $P < 0.01$). Both the surface area and depth scores were associated with the need for re-operation: higher scores, reflecting more severe necrosis of the mastectomy flap, were associated with increased need for re-operation. Twenty-nine breasts (58%) recovered without additional operation. Pre-operative radiotherapy (OR 7.2, 95% CI 1.4–37.3) and postoperative infection (OR 15.4, 95% CI 2.6–89.7) were risk factors for re-operation in multivariate analyses.

Conclusions: In this case series, the surface area of the breast affected by ischaemia decreased during HBOT, and most breasts (58%) did not undergo an additional operation. A randomised control trial is needed to confirm or refute the possibility that HBOT improves outcome in patients with mastectomy flap ischaemia.

Introduction

When patients with breast cancer who need a mastectomy opt for a breast reconstruction, a major benefit of an immediate reconstruction is the better aesthetic outcome compared to delayed reconstruction.[1] However, mastectomy with immediate reconstruction leads to a higher rate of postoperative complications and a greater need for reoperation.[2] Mastectomy flap ischaemia leading to necrosis is reported in 4.3% (n = 178/4,158),[3] 12% (n = 112/903),[4] and 14% of patients (n = 85/606)[5] who were followed prospectively after mastectomy and immediate reconstruction.

The following risk factors have been associated with mastectomy flap ischaemia and necrosis: age, body mass index (BMI) > 30 kg·m^{-2}, larger cup size, previous or current smoking, hypertension, prior breast-reduction surgery, history of breast augmentation, previous radiation therapy, nipple-sparing mastectomy, time from incision to removal of specimen, mastectomy specimen weight (> 500 gram), one-stage breast reconstruction, use of an acellular dermal matrix, and the volume of operative tissue expander fill > 300 cm^3.[1,5–7] These factors can lead to impaired perfusion of the mastectomy flap and result in skin necrosis.

Several classifications of severity of mastectomy flap necrosis have been described by Matsen et al. (mild, moderate, severe),[5] Frey et al. (minor, major)[8] and Lemaine et al. (depth, surface area).[9] The former scores are dependent on the time to healing and the type of intervention; the latter score parallels that of burn severity classification.

Current treatments of mastectomy flap ischaemia include wait-and-see[3,9] nitroglycerin ointment,[10–14] topical silver sulfadiazine,[1] topical dimethylsulfoxide,[15] oral or intravenous

antibiotics,[1] Dextran-40 infusion,[16] and tissue expander expansion of the well-perfused tissue to create sufficient tissue for excision of full-thickness necrosis and primary closure 4–6 weeks postoperatively.[3] Five to 67% of patients with mastectomy flap ischaemia require reoperation including debridement or removal of the tissue expander or implant.[1,3,11,12,14,17,18]

Hyperbaric oxygen treatment (HBOT) has been used to treat various ischaemic skin flaps and grafts.[19–27] Although some types of flap compromise can be addressed by re-exploration,[28] when there is no correctable mechanical cause of flap ischaemia, HBOT can be used to hyperoxygenate the flap and reduce oedema.[26,28,29] HBOT may prevent the progression of ischaemia to necrosis or limit the extent of necrosis. In a case series of 65 postoperatively compromised skin flaps treated with HBOT, 36 (55%) showed "*complete healing*" and 22 (34%) "*marked improvement*".[26] Only five case reports[30–34] and a small case-control study[35] were identified reporting that HBOT can successfully prevent necrosis of ischaemic skin flaps after mastectomy.

In a recent review on the challenges and solutions for mastectomy skin flap necrosis, HBOT was mentioned as being successful in case reports. However, in that review the use of HBOT was not recommended due to lack of larger series to support its use.[13] Therefore, the purpose of this study was to evaluate outcomes of HBOT in patients with skin flap ischaemia following mastectomy. The primary outcome was the need for additional surgery following HBOT, while the secondary outcome was a decrease in tissue necrosis using the SKIN score.[9] We also sought risk factors for additional surgery despite HBOT.

Methods

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. Patients signed informed consent forms to use photographs for clinical and research purposes. A retrospective chart review was performed of the patients with mastectomy flap ischaemia who were referred to the Da Vinci Clinic (Geldrop) for HBOT between January 2013 and January 2018. During this period 44 patients with compromised mastectomy flaps (50 breasts) were referred from five hospitals in the Netherlands, including Catharina Hospital (Eindhoven), Maxima Medical Centre (Veldhoven and Eindhoven), St. Anna Hospital (Geldrop), St. Jans Hospital (Weert), and University Medical Centre (Maastricht). It is not known if all patients with mastectomy flap ischaemia were referred for HBOT. Other hospitals in the area did not refer any patients. A retrospective analysis was performed. Medical records were reviewed from both the Da Vinci Clinic and the referring hospitals to gather information concerning patient demographics, operative details, HBOT and outcomes.

The Da Vinci Clinic has a multiplace hyperbaric chamber for 12 patients (IHC Hytech, Raamsdonkveer, The Netherlands) for HBOT. Patients were treated at 253 kPa (2.5 atmospheres absolute). At this pressure, 100% oxygen was breathed via a mask during four periods for a total of 85 minutes, interspersed by three 5-minute air breaks. Including compression and decompression time, the total duration of each session was 110 minutes. Patients underwent two sessions per day for the first three days, followed by one session per day until circulation in the mastectomy flap was restored or demarcation was achieved.

Relative contra-indications for HBOT are epilepsy, history of pneumothorax or pulmonary surgery, COPD with known bullae or requiring continuous normobaric oxygen, left ventricle ejection fraction < 20%, concomitant or recent treatment with cisplatinum, doxorubicin or bleomycin, previous middle ear reconstruction, or pregnancy.[36] None of the patients referred with mastectomy flap ischaemia had any relative contra-indications to HBOT.

To assess the decrease of tissue necrosis during HBOT, pre- and post-HBOT photographs were scored by the four authors independently using a previously validated system;[9] the SKIN (Skin Ischaemia Necrosis) score. Photographs were taken before the first session of HBOT and after completion of the course of HBOT, and scored in a random order. The SKIN score includes surface area and depth of ischaemia or necrosis of the mastectomy skin flap and nipple-areolar complex. The affected surface area was scored: 1 = 0%; 2 = 1–10%; 3 = 11–30%; and 4 = > 30%. The estimated affected depth was scored: A = none; B = colour change; C = partial thickness skin flap necrosis; and D = full thickness skin flap necrosis.

When the SKIN score improves, secondary surgery may be avoided or minimised.[9] The outcome of the mastectomy flap ischaemia treated with HBOT was collected from the medical records at the referring hospitals, and scored using the grades of Matsen et al., (mild, moderate, severe)[5] and Frey et al. (minor, major).[8]

STATISTICAL ANALYSIS

Analyses were performed per breast, not per patient. Statistical analyses were performed using SPSS version 22.0 software (SPSS inc., Illinois, USA) and with SAS version 9.2 (SAS Institute, North Carolina, USA). Differences were considered significant at a value of $P < 0.05$. Missing and inconsistent data were excluded. Descriptive statistics were reported as number and percentage of breasts, median with range, and counts of the SKIN scores. A binomial test was used for comparisons between pre-HBOT and post-HBOT SKIN scores. For assessment of inter-observer agreement for SKIN scores, Fleiss' kappa was calculated. Cross tabulations were used to assess the association between post-HBOT measurements, SKIN scores and reoperation. No statistical tests were applied to investigate the significance here.

Table 1
Demographics and comorbidity (n = 44 patients). *Six of the 19 patients who underwent bilateral mastectomies had bilateral mastectomy flap ischaemia. ASA – American Society of Anesthetists classification. BMI – body mass index

Parameter	Median (range)
Age (years)	52 (23–72)
BMI (kg·m^{-2})	23 (18–31)
	***n* (%)**
ASA > 2	5 (10)
Diabetes mellitus	2 (5)
Hypertension	3 (7)
Coagulation disorder	5 (11)
Current smoker	12 (27)
Use of immunosuppressant	2 (5)
Preoperative chemotherapy	12 (27)
Preoperative radiotherapy	11 (25)
Previous breast augmentation	4 (9)
Previous breast reduction	3 (7)
Bilateral breast surgery	19 (43)*

Table 2
Operative characteristics (n = 50 breasts)

Operative characteristics	*n* (%)
Nipple-sparing	22 (44)
Indication for surgery	
- Ductal carcinoma in situ	11 (22)
- Breast cancer	29 (58)
- Prophylactic	4 (8)
- BRCA mutation and breast cancer	2 (4)
Operation	
- Tissue expander	
Primary	16 (32)
Hammond	13 (26)
Latissimus dorsi flap	1 (2)
- Implant	
Primary	6 (12)
Hammond	4 (8)
Latissimus dorsi flap	3 (6)
Acellular dermal matrix	3 (6)
- Deep inferior epigastric artery perforator-flap	1 (2)
- Reversed abdominoplasty	1 (2)
- No reconstruction	2 (4)

Table 3
Outcome after mastectomy flap ischaemia and HBOT. * Matsen et al.[5], mild: no intervention needed, healing complete at eight weeks, moderate: office debridement, healing complete at eight weeks, severe: operating room debridement, implant loss, or healing not complete at eight weeks. **Frey et al.[8], minor: requiring only local wound care, major: requiring debridement either in the office or in the operating room

Postoperative outcome	*n* (%)
Infection	12 (24)
Seroma	9 (18)
Hematoma	4 (8)
Reoperation	21 (42)
- Removal of tissue expander or implant	15 (30)
- Partial debridement of skin flap	4 (8)
- Full-thickness skin graft	1 (2)
- Latissimus dorsi-flap	1 (2)
Matsen et al.*	
- Mild	26 (52)
- Moderate	3 (6)
- Severe	21 (42)
Frey et al.**	
- Minor	26 (52)
- Major	24 (48)

The Spearman test was used for evaluation of correlations between risk factors and the degree of necrosis as defined by Matsen et al.[5] Step-wise multivariate logistic regression analyses were used to assess associations between risk factors and re-operation.

Results

Between January 2013 and January 2018, 44 patients with 50 breasts with skin flap ischaemia after mastectomy were referred for HBOT. Patient demographics and comorbidity are presented in Table 1. Operative characteristics are presented in Table 2. Most underwent mastectomy for breast cancer (29 breasts; 58%), and most had an immediate breast reconstruction with a tissue expander (30 breasts; 60%) or breast implant (16 breasts; 32%). Two breasts were not immediately reconstructed after mastectomy because of ischaemic skin flaps peri-operatively. In addition to mastectomy flap ischaemia, some breasts had other postoperative complications, including infection, seroma, and haematoma (Table 3).

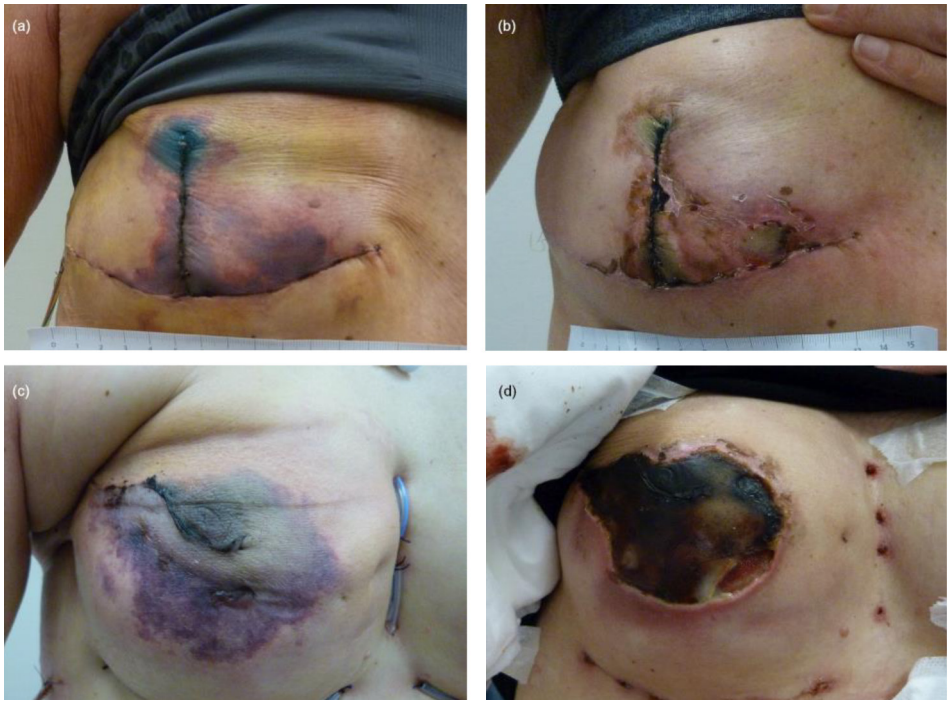
HBOT was started at a median of 3 (range 1–23) days following mastectomy. Patients underwent a median of 12 sessions of HBOT (range 6–22). The most common side effect of HBOT was problems equalising the ears: 10/44 patients (23%) used nasal decongestant spray and 4/44 patients (9%) needed myringotomy tubes. No central nervous system oxygen toxicity or visual changes were reported due to HBOT. No patients prematurely terminated the treatment.

OUTCOMES

Most breasts recovered without reoperation (n = 29/50 (58%), Table 3 and Figure 1 A–B). Reoperation was required for 21/50 breasts (42%), including removal of the

Figure 1

Breasts before and after the course of HBOT; (a) Breast on presentation for HBOT two days after Hammond mastectomy and immediate reconstruction with a tissue expander, and (b) after a course of 20 treatments with HBOT. Beneath the superficial necrosis, the breast reconstruction remained intact. No further surgery was necessary. (c) Breast on presentation for HBOT three days after nipple-sparing mastectomy and immediate reconstruction with an implant and (d) after course of 22 treatments with HBOT. The full thickness necrosis of the nipple areola complex and surrounding skin flap resulted in exposure of the implant, which needed to be removed.



tissue expander or implant in 13/46 (28%), Figure 1 C–D), debridement of the skin flap in 4/50 (8%) or secondary reconstruction with a full-thickness skin graft (one patient) or latissimus dorsi flap (one patient).

SKIN scores (surface area and depth) were given by four independent observers, with separate scores for the affected breast skin ($n = 50$) and the nipple areola complex ($n = 22$) at the start and end of the course of HBOT.

Pre- and post-HBOT breast SKIN scores were complete for 175 observations (Table 4). The changes between the pre- and post-HBOT surface area and affected depth scores showed a mix of improvement, no change, or deterioration. Overall, the surface area scores improved more often than they deteriorated (34% vs. 5%, $P < 0.01$), and the depth scores deteriorated more often than they improved (42% vs. 17%, $P < 0.01$). The inter-observer Kappa was low (0.213 and 0.282 respectively).

Pre- and post-HBOT nipple-areolar complex SKIN scores were complete for 64 observations (Table 5). The changes between the pre- and post-HBOT surface area and affected depth scores also showed a mix of improvement, no change or deterioration. Overall, the change in surface area scores was not statistically significant (27% improvement vs. 17%

Table 4

Difference between pre- and post-HBOT breast SKIN score ($n = 175$ observations)

	Surface area	Depth
Deteriorated	8 (5%)	74 (42%)
Unchanged	107 (61%)	72 (41%)
Improved	60 (34%)	29 (16–17%)
P-value	< 0.01	< 0.01
Interobserver Kappa (95% CI)	0.213 (0.061–0.366)	0.282 (0.157–0.407)

Table 5

Difference between pre- and post-HBOT nipple areola complex SKIN score ($n = 64$ observations)

	Surface area	Depth
Deteriorated	11 (17%)	32 (50%)
Unchanged	36 (56%)	28 (44%)
Improved	17 (27%)	4 (6%)
P-value	0.13	< 0.01
Inter-observer Kappa (95% CI)	0.138 (-0.037–0.313)	0.073 (-1.156–0.302)

Figure 2
Association between the post-HBOT breast SKIN scores and re-operation (n = 183 observations). Affected surface area was scored: 1 – 0%; 2 – 1–10%; 3 – 11–30%; and 4 – > 30%. The estimated affected depth was scored: A – none; B – colour change; C – partial thickness skin flap necrosis; and D – full thickness skin flap necrosis

deterioration, $P = 0.13$), and the depth scores deteriorated more often than they improved (50% vs. 6%, $P < 0.01$). The interobserver Kappa was low (0.138 and 0.073 respectively).

Post-HBOT breast SKIN scores were available for 46 breasts (183 observations). Both the surface area and depth scores were associated with the need for re-operation: higher scores, reflecting more severe necrosis of the mastectomy flap, were associated with a more likely need for re-operation (Figure 2). The combined surface area and depth scores were categorized into three groups with differing prognosis (Table 6): good (5% re-operation), moderate (27% re-operation), and poor (67% re-operation). The interobserver Kappa was moderate (0.438, range 0.341–0.535).

MULTIVARIATE ANALYSES

Associations between risk factors and necrosis were analysed by Spearman correlations, and between risk factors and re-operation by multivariate analyses (Table 7). The correlations were evaluated between risk factors and the

Table 6
Association between the post-HBOT breast SKIN scores and re-operation (n = 183 observations)

Depth	Surface area	n	Re-operation	Prognosis
A	1	61	5%	Good
B	2			
B	3	33	27%	Moderate
C	2			
B	4	89	67%	Poor
C	3, 4			
D	2, 3, 4			

degree of necrosis as graded by Matsen et al.[5] Previous breast reduction (Spearman's rho 0.3; $P = 0.04$), pre-operative radiotherapy (Spearman's rho 0.3; $P = 0.03$), and infection (Spearman's rho 0.4; $P = 0.001$) were significantly related to the degree of necrosis. Pre-operative radiotherapy (OR 7.2, 95% CI 1.4–37.3) and infection (OR 15.4, 95% CI 2.6–89.7) were risk factors for re-operation in multivariate analyses.

Discussion

The aim of this study was to investigate the outcome of mastectomy flap ischaemia after HBOT in a series of 50 breasts. Our primary outcome was the need for additional surgery following HBOT, while our secondary outcome was a decrease in tissue necrosis using the SKIN score. We also sought risk factors for additional surgery despite HBOT.

HBOT improves oxygenation of poorly perfused tissue and reduces oedema, and may thereby prevent the progression of ischaemia to necrosis or limit the extent of necrosis of vascularly compromised skin grafts or flaps.[29,36] Postoperative skin flap ischaemia can progress to full thickness necrosis, resulting in wound dehiscence. In the case of mastectomy flap ischaemia and an immediate reconstruction with a tissue expander or implant, the exposed device must be removed, delaying further surgery and compromising the aesthetic outcome. Timely HBOT may sustain the ischaemic tissue until perfusion is restored, thereby preventing progression to necrosis or limiting the necrosis to partial thickness of the flap which can heal by secondary intention without additional surgery.[30–33]

In this study the SKIN score depth did deteriorate as the tissue demarcated, but the affected surface area decreased significantly with HBOT (see Figure 1 and Table 4). The SKIN score was developed to translate into groups with clinically meaningful differences: when the affected surface area decreases the likelihood of re-operation decreases[9] (Figure 2 and Table 6). The inter-rater reliability for the change in SKIN score from before to after HBOT was low (Kappa 0.073–0.282); this was mostly due to differences in the pre-HBOT scores when the tissue colour is not clear. Once the tissue has demarcated post-HBOT, the interrater

Table 7

Risk factors for necrosis and re-operation. *Matsen et al.,[5] mild: no intervention needed, healing complete at eight weeks, moderate: office debridement, healing complete at eight weeks, severe: operating room debridement, implant loss, or healing not complete at 8 weeks. Results in bold are statistically significant.

Risk factor	Degree of necrosis*		Re-operation	
	Spearman's rho	*P*-value	Odds ratio	95% CI
Age	0.241	0.092		
BMI	0.268	0.060		
Cup size	-0.056	0.697		
Previous or current smoking	-0.004	0.976		
Hypertension (*n* = 4)	0.043	0.766		
Diabetes (*n* = 3)	-0.046	0.751		
Prior breast augmentation (*n* = 5)	-0.175	0.225		
Previous breast reduction (*n* = 3)	0.286	0.044		
Previous radiation therapy (*n* = 11)	**0.302**	**0.033**	**7.2**	**1.4–37.3**
Previous chemotherapy (*n* = 14)	0.153	0.288		
Infection (*n* = 12)	**0.443**	**0.001**	**15.4**	**2.6–89.7**
Nipple-sparing mastectomy (*n* = 22)	0.011	0.939		
Hammond (*n* = 17)	-0.013	0.927		
Weight of mastectomy specimen	0.173	0.229		
Use of an acellular dermal matrix (*n* =3)	0.029	0.843		
Number of days of delay to HBOT	-0.166	0.250		
Total number of sessions of HBOT	0.130	0.367		

agreement was better. We used the post-HBOT scores to calculate the prognosis for reoperation (Kappa 0.438, range 0.341–0.535).

Only 21/50 (42%) of breasts with mastectomy flap ischaemia that were treated with HBOT underwent additional surgery (Table 3). In the literature, the need for further surgery in cases with mastectomy flap ischaemia ranges from 5–67% following treatments other than HBOT. The reoperation rate in this study with HBOT falls in the middle of that range. In a small study where mastectomy flap ischaemia was treated with a wait-and-see approach, 6/11 cases (55%) required debridement and coverage.[37] Another small study where all patients with mastectomy flap necrosis were treated with oral antibiotics, 10/15 patients (67%) required readmission with intravenous antibiotics, surgical debridement, and removal of their tissue expander.[1] In a series of nipple-sparing mastectomies, only 1/20 of nipples (5%) with necrosis required reoperation.[18] In another larger study where mastectomy flap necrosis was treated with a wait-and-see approach, 18/69 breasts (26%) required skin excision, debridement, or implant removal.[9] In a large series of nipple-sparing mastectomies, reoperation was required in 69/141 (49%) of ischaemic nipples.[38] In the largest study of 178 patients with mastectomy flap necrosis who were treated with expansion of the tissue expander, 120 (67%) healed spontaneously and 58 (33%) required surgical excision of the eschar or removal of the tissue expander.[3] It is not possible to compare the outcomes of these studies to our study since each uses a different definition of mastectomy flap ischaemia, leading to selection bias.

Until now only one large study had investigated the efficacy of HBOT in limiting necrosis of ischaemic skin flaps. The study reviewed the outcome of 65 compromised flaps in a heterogeneous population, including soft tissue injuries and osteomyelitis.[26] The treatment outcome was judged on the appearance of the flap. Following HBOT 55% had 'no flap necrosis', 34% had 'minimal flap necrosis', and 11% had 'flap necrosis requiring a further covering procedure or extensive healing by secondary intention'. The authors concluded that 89% of compromised flaps were 'salvaged' by HBOT. Patients whose outcome were unsuccessful were older (60 vs. 48 years), had a longer delay to initiation of HBOT (20 vs. 5 days), a greater number of HBOT treatments (42 vs. 28 sessions), and a greater number of risk factors associated with poor wound healing (soft tissue infections, radiation therapy, peripheral vascular disease, and diabetes mellitus). Delay to HBOT and total number of HBOT treatments were not significant risk factors for reoperation in our study (Table 7).

In previous studies, various risk factors have been identified for necrosis; the common mechanism in all is impaired perfusion. In this study risk factors that were associated with necrosis and reoperation were previous breast reduction, preoperative radiotherapy, and infection (Table 7). These risk factors were also found to be significant in other studies. Prior surgical scars can compromise skin perfusion leading to a higher prevalence of necrosis in these patients.[39] Severe skin necrosis was 14 times more likely in previously irradiated patients.[37] Patients with mastectomy skin necrosis have a 15 times higher odds of developing an infection

requiring intervention and an almost 16 times the odds of requiring their tissue expander to be prematurely removed.[1] Other risk factors that were also shown to be associated with necrosis and reoperation in other studies were not correlated in this study, perhaps due to small patient numbers in those subgroups.

Interestingly, in this study previous or current smoking was not correlated with the degree of necrosis nor the need for additional surgery. Other studies have shown that smoking was significantly associated with necrosis[3,7] and excision,[3] and reduced the effect of HBOT on compromised flaps.[25] In fact, in some HBOT centres, smoking is considered so detrimental to the effect of HBOT that the treatment was discontinued for patients who refused to refrain from smoking.[26]

The main limitation of this study is that there was no control group with mastectomy flap ischaemia who did not undergo HBOT. It is unknown what proportion would have resolved with a wait-and-see approach,[3,5,9] and what proportion would be re-operated. Another limitation is that the indication for reoperation was not clear: haematoma, seroma, infection, and flap necrosis could all be independent indications for reoperation.

Conclusions

Limiting necrosis is important to reduce morbidity and the costs of repetitive reoperation.[1,26] In this case series of patients with mastectomy flap ischaemia, the surface area of the breast affected by ischaemia decreased during HBOT, and most breasts (29/50, 58%) did not undergo an additional operation. A randomised controlled trial is needed to confirm or refute the possibility that HBOT improves outcome in patients with mastectomy flap ischaemia.

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An insight to tympanic membrane perforation pressure through morphometry: A cadaver study

Derya Ümit Talas[1], Orhan Beger[2], Ülkü Çömelekoğlu[3], Salim Çakır[4], Pourya Taghipour[4], Yusuf Vayisoğlu[1]

[1] *Mersin University Faculty of Medicine, Department of Otorhinolaryngology, Mersin, Turkey*
[2] *Mersin University Faculty of Medicine, Department of Anatomy, Mersin, Turkey*
[3] *Mersin University Faculty of Medicine, Department of Biophysics, Mersin, Turkey*
[4] *Mersin University Faculty of Medicine, Mersin, Turkey*

Corresponding author: *Orhan Beger, Mersin University Faculty of Medicine, Department of Anatomy, Ciftlikkoy Campus, 33343, Mersin, Turkey*
obeger@gmail.com

Key words
Bursting pressure, Cadaver, Ear barotrauma; Eardrum; Diving; Transducer

Abstract
(Talas DÜ, Beger O, Çömelekoğlu Ü, Çakır S, Taghipour P, Vayisoğlu Y. An insight to tympanic membrane perforation pressure through morphometry: A cadaver study. Diving and Hyperbaric Medicine. 2021 March 31;51(1):10–17. doi: 10.28920/dhm51.1.10-17. PMID: 33761536.)
Introduction: A cadaveric experimental investigation aimed to show the rupture pressure of the tympanic membrane (TM) for otologists to evaluate its tensile strength.
Methods: Twenty adult ears in 10 fresh frozen whole cadaveric heads (four males, six females) mean age 72.8 (SD 13.8) years (range 40–86) were studied. The tensile strength of the TM was evaluated with bursting pressure of the membrane. The dimensions of the membranes and perforations were measured with digital imaging software.
Results: The mean bursting pressure of the TM was 97.71 (SD 36.20) kPa. The mean area, vertical and horizontal diameters of the TM were 57.46 (16.23) mm^2, 9.54 (1.27) mm, 7.99 (1.08) mm respectively. The mean area, length and width of the perforations were 0.55 (0.25) mm^2, 1.37 (0.50) mm, and 0.52 (0.22) mm, respectively. Comparisons of TM dimension, bursting pressure, and perforation size by laterality and gender showed no significant differences. The bursting pressure did not correlate (positively or negatively) with the TM or perforation sizes.
Conclusions: The TM can rupture during activities such as freediving or scuba diving, potentially leading to serious problems including brain injuries. Studying such events via cadaveric studies and data from case studies is of fundamental importance. The minimum experimental bursting pressures might better be taken into consideration rather than average values as the danger threshold for prevention of TM damage (and complications thereof) by barotrauma.

Introduction

The tympanic membrane (TM), a thin, oval-shaped, semi-transparent drum, transmits sound waves from the external auditory canal (EAC) to the ear ossicles, and then to the cochlea.[1] The shape, elasticity and size of the TM are influential on its function.[2] Hearing loss due to perforation or rupture can occur when the TM is exposed to air or water pressures that exceed its mechanical capacity.[3–8] Perforations depend on environmental conditions in addition to the intrinsic properties of the membrane.[6] For instance, air pressure at 20,000 m altitude (5.47 kPa) is approximately 5.4% of sea level (101.32 kPa).[6] Moreover, in seawater, ambient pressure increases by 101.3 kPa (i.e., 760 mmHg or 1 atmosphere) every 10 m depth.[6] A TM rupture may occur due to pressure changes during diving, scuba diving, freediving, slap in face, martial arts, air travel, and blast injury.[9–13] These conditions causing overpressure (e.g., scuba diving or freediving) can lead to symptoms such as otalgia, vertigo, and hearing loss if appropriate equipment (maybe special equipment for individuals with scarred TM) is not used.[7,9,11] Knowledge of the perforation threshold of the TM could contribute to the adaptation of devices used during sports activities (e.g., diving or martial arts), and to the development of protective military equipment (e.g., against combat explosions).[7,8,10,11,13] There is currently insufficient data regarding the bursting pressure of the TM in humans.[7,8,14]

TM rupture may be repaired with paper patches, cartilage, deep temporalis fascia, dura, or fat during myringoplasty or tympanoplasty.[4,5,9,15,16] The perforation dimension and in the diameters of the TM are important for ear surgeons in terms of preoperative choice of graft sizes.[15] The classic textbook reports that TM diameters lie in a narrow range (9–10 mm height, and 8–9 mm width);[1] however, some ear specialists have observed that the TM sizes vary widely among individuals (5 mm for the horizontal diameter).[5] New studies focusing on the dimensions of the TM can help ear surgeons estimate its size. In previous studies,[6–8] the

location of perforations has been evaluated after exposure to bursting pressure; however, it seems that variation of perforation dimensions in relation to the pressure exposure has been ignored. Furthermore, the relationship between TM diameters and bursting pressure could be useful for understanding the effect of dimension on its mechanical capacity. This study aimed to measure perforation pressure of the TM to provide a better understanding of its tensile strength, and to measure perforation size in terms of preoperative graft design.

Methods

The study was approved by the ethical board of the Mersin University Faculty of Medicine.

PREPARATION OF THE EARS

Twenty ears in 10 fresh frozen cadaver heads (4 males, 6 females) mean age 72.8 (SD 13.8) years (range 40–86 years) were included in the study. The heads were positioned in accordance with otologic surgery, and then the senior otologist (DÜT) cleared the EAC under a surgical microscope (Carl Zeiss f170, Carl Zeiss Meditec AG, Germany). Photographs of the TMs were taken before and after exposure to perforation pressure.

MEASUREMENT OF TM PERFORATION PRESSURE

Two plastic tubes were placed inside a rubber ear plug. The proximal end of one of the tubes was connected to an air-filled syringe (20 cc), while the other proximal end was connected to a pressure transducer. The ear plug and the distal ends of the tubes were tightly bonded with glue to prevent air leaks. After the plug was placed in the ear, the air in the syringe was delivered to the EAC by the same researcher (OB). Pressure data were collected by an electrophysiological recording acquisition system (BIOPAC MP 100, Systems Inc., Santa Barbara, CA, USA) and then transferred to a computer via a 16 bit analog to digital converter for off line analysis (Figure 1). The sampling rate was 200·sec^{-1}. BIOPAC Acknowledge Analysis Software (ACK 100 W) was used to evaluate the pressure data. The highest pressure before a sudden brief downward deflection in the graphs was recorded as the perforation pressure of the TM (Figure 2 arrow).

MEASUREMENTS OF THE TM AND PERFORATION SIZES

Using a 0°, 4 mm diameter, 18 cm length endoscope (Karl Storz Gmbh & Co., Tuttlingen, Germany), photographs of the TM were taken using a SPIES H3-Z three-chip full HD camera connected to a monitor (Karl Storz Gmbh., Tuttlingen, Germany) with a millimeter scale. To determine the TM size including its surface area, height and width, the photos were transferred to digital image analysis software (Rasband WS, ImageJ, US National Institutes of Health,

Figure 1
Experimental set-up showing syringe and transducer configuration, monitoring, and preparation of the cadaver ear

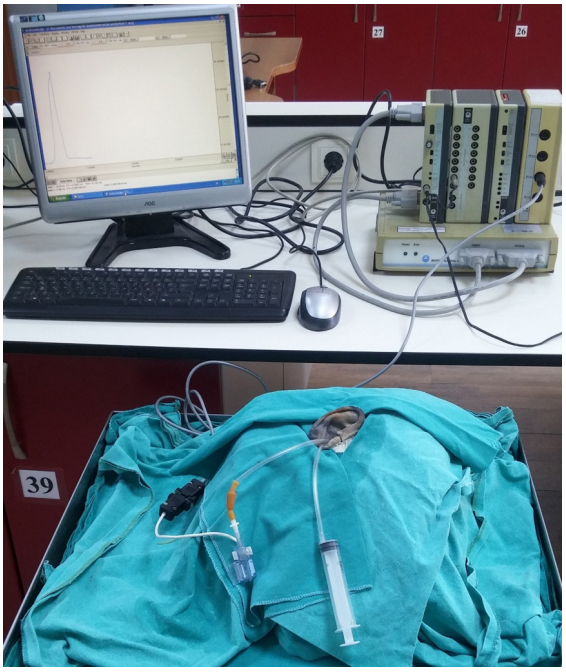


Figure 2
Representative pressure chart during a perforation pressure experiment showing the characteristic notch at the point of perforation (arrow)

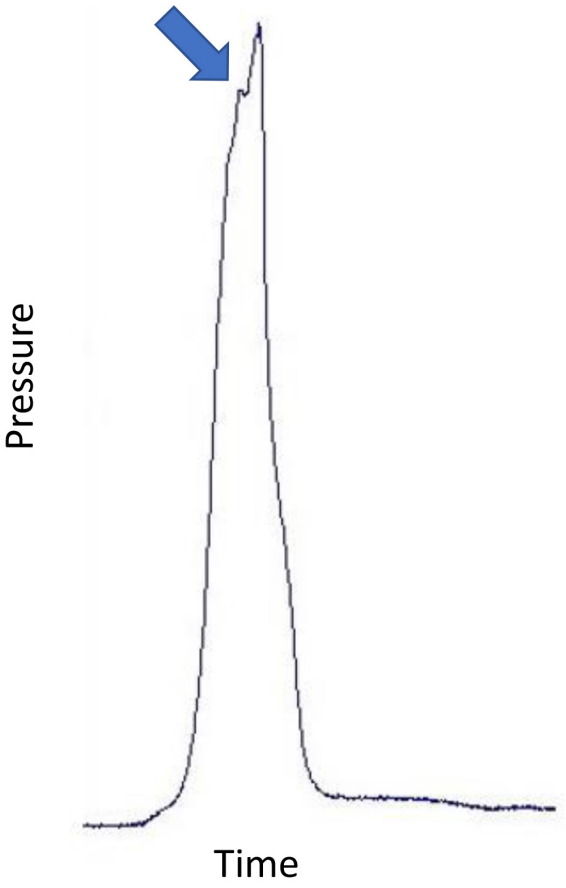
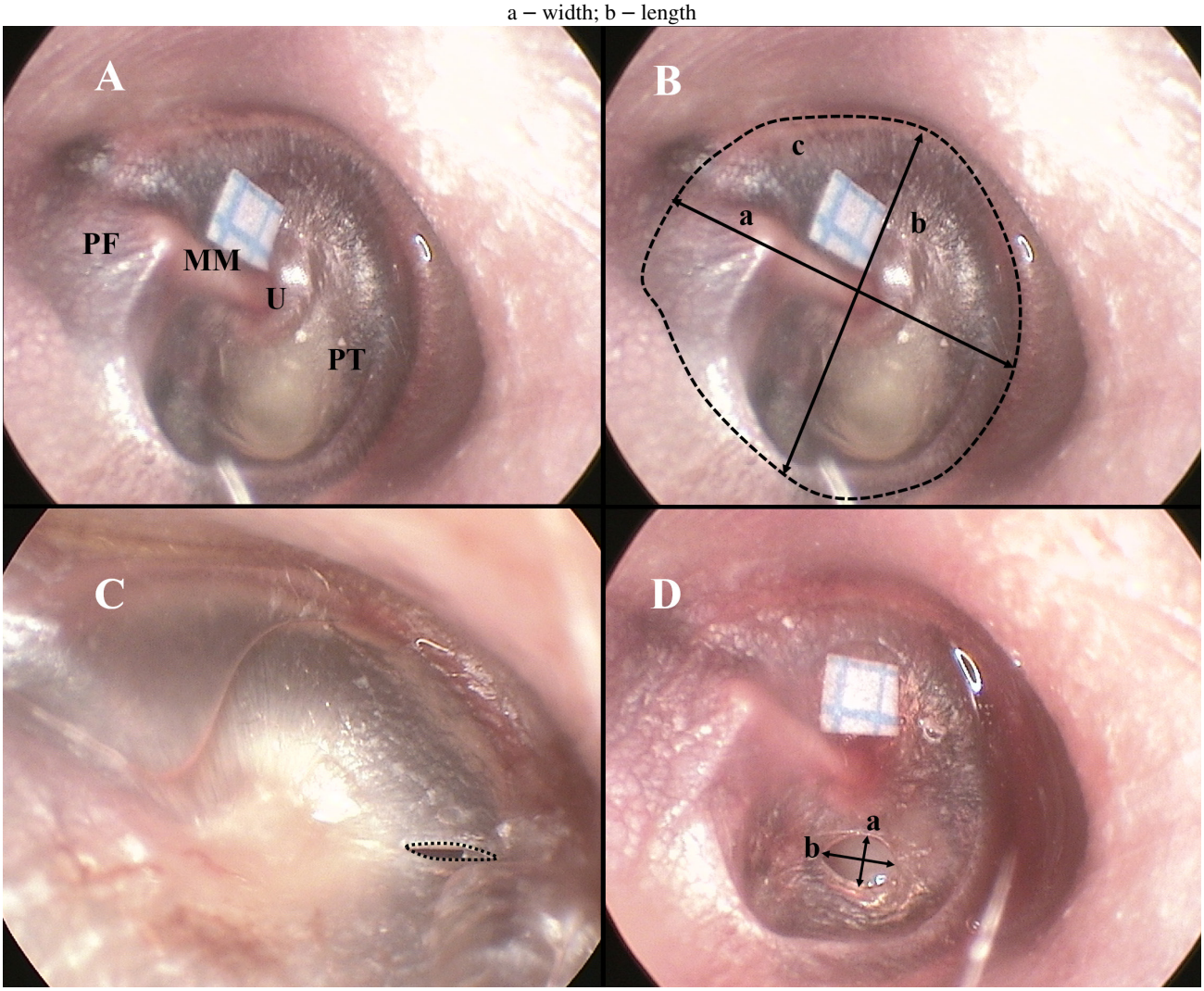


Figure 3

A: Typical tympanic membrane. MM – manubrium mallei; U – umbo; PT – pars tensa; PF – pars flaccida. B: Tympanic membrane measurements. a – vertical diameter; b – horizontal diameter; c – the surface area. C: Perforation area. D: Perforation dimensions. a – width; b – length



Bethesda, Maryland, USA). After the pressure exposure, the TM was re-photographed to determine the location of the perforation, and to measure the perforation length, width and area. Measured parameters related to the TM and perforation (Figure 3) were: the surface area of the TM (TMA); the vertical diameter of the TM (the line passing through the manubrium mallei) (TMVD); the horizontal diameter of the TM (the line passing through the umbo perpendicular to the handle of malleus) (TMHD); the length of the perforation (at the longest level) (PL); the width of the perforation (at the widest level) (PW); the surface area of the perforation (PA).

STATISTICAL ANALYSIS

Normality checks of the dataset including dimensional and pressure measurements were performed with the Shapiro-Wilk test. Student's *t*-tests were used to compare TMVD – TMHD (paired sample *t*-test), male – female (independent sample *t*-test), and right – left sides (paired sample *t*-test). Correlations between the parameters including dimension and pressure measurements of the TM were evaluated with the Pearson correlation coefficient test. A P-value < 0.05 was considered statistically significant.

Results

Data were normally distributed and therefore data were presented as mean and standard deviation (SD).

The mean perforation pressure for all ears was 97.71 (SD 36.20) kPa, range 35.79–151.78. In terms of sexes or sides, the TM size (length, width and area), perforation pressure, and perforation size (length, width and area) did not show statistically significant differences (Table 1).

The TMVD - TMHD (P < 0.001, r = 0.710), the TMVD - TMA (P < 0.001, r = 0.870), and the TMHD - TMA (P = 0.001, r = 0.788) showed strong positive correlations

Table 1

Perforation pressure and TM dimensional data. Data are mean (SD) [range]. PA – perforation area; PL – perforation length; PP – perforation pressure; PW – perforation width; TMA – tympanic membrane area; TMHD – tympanic membrane horizontal diameter; TMVD – tympanic membrane vertical diameter

Parameter	All ears	Right	Left	*P*	Female	Male	*P*
PP (kPa)	97.71 (36.20) [35.79–151.78]	96.86 (33.72)	98.57 (40.35)	0.92	100.22 (38.30)	93.95 (35.00)	0.72
TMVD (mm)	9.54 (1.27) [7.23–11.61]	9.99 (1.15)	9.09 (1.28)	0.12	9.64 (1.37)	9.38 (1.18)	0.67
TMHD (mm)	7.99 (1.08) [5.85–9.74]	8.26 (1.20)	7.73 (0.94)	0.28	7.89 (1.12)	8.15 (1.08)	0.62
TMA (mm^2)	57.46 (16.23) [33.45–93.43]	62.03 (16.16)	52.89 (15.88)	0.22	58.16 (17.72)	56.41 (14.84)	0.82
PL (mm)	1.37 (0.50) [0.51–2.51]	1.42 (0.42)	1.32 (0.59)	0.67	1.35 (0.56)	1.40 (0.43)	0.82
PW (mm)	0.52 (0.22) [0.22–1.04]	0.61 (0.25)	0.42 (0.12)	0.06	0.55 (0.23)	0.46 (0.20)	0.40
PA (mm^2)	0.55 (0.25) [0.19–1.20]	0.59 (0.30)	0.50 (0.19)	0.45	0.52 (0.28)	0.59 (0.18)	0.59

Table 2

Correlation coefficients and *P*-values between parameters related to the TM. Bold and italic are statistically significant correlations. PA – perforation area; PL – perforation length; PP – perforation pressure; PW – perforation width; TMA – tympanic membrane area; TMHD – tympanic membrane horizontal diameter; TMVD – tympanic membrane vertical diameter

Parameter	TMVD	TMHD	TMA	PL	PW	PA
PP	-0.054 0.820	-0.194 0.412	-0.118 0.620	0.022 0.926	-0.249 0.290	-0.096 0.688
TMVD		***0.710*** ***< 0.001***	***0.870*** ***< 0.001***	-0.439 0.053	-0.093 0.698	***-0.493*** ***0.027***
TMHD			***0.788*** ***< 0.001***	-0.133 0.576	0.010 0.967	-0.288 0.218
TMA				-0.228 0.334	-0.168 0.479	***-0.544*** ***0.013***
PL					0.122 0.608	***0.452*** ***0.045***
PW						0.527 0.017

(Table 2). The PL - PA ($P = 0.045$, r = 0.452) and the PW - PA ($P = 0.017$, r = 0.527) showed weak positive correlations (Table 2). The TMVD - PA (P = 0.027, r = 0.493) and the TMA - PA ($P = 0.013$, r = 0.544) showed weak negative correlations (Table 2).

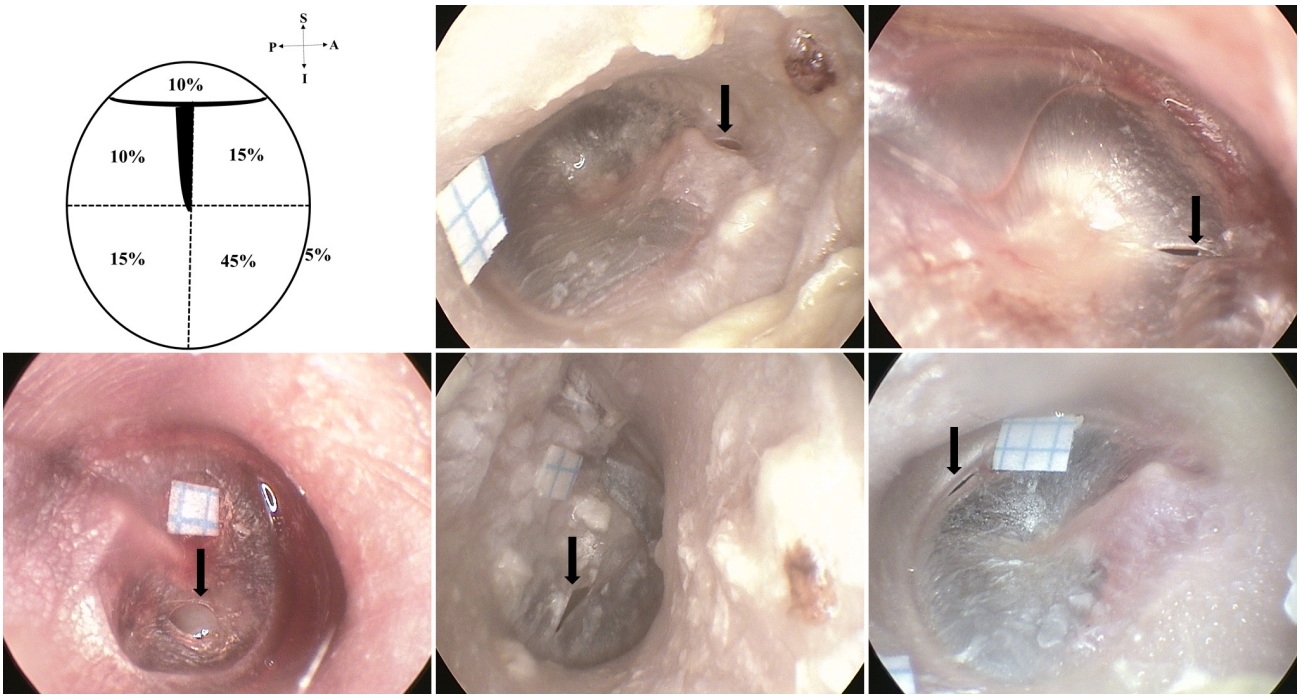
Two of 20 ruptures occurred in the pars flaccida, nine in the anterior-inferior quadrant, three in the anterior-superior quadrant, three in the posterior-inferior quadrant, and two in the posterior-superior quadrant. There was just one marginal perforation (Figure 4).

Discussion

Barotrauma caused by scuba diving, freediving, a slap to the face, martial arts, air travel, and blast injury can occur in the middle or inner ear.[3,5,9–13] When the pressure rises above the hazardous level for the middle ear, the Eustachian tube fails to balance the pressure.[6,7] Rupture of the TM is one of the important indicators that the dangerous level has been exceeded.[6–8,14,17,18] The average overpressure value for perforation of the TM in one study was 171.99 kPa (1,290 mmHg).[8] Others observed that this pressure level could be reached at 17.6 metres' sea water (msw).[6] In

Figure 4

Schematic TM and percentages of perforations (upper left panel). Two perforations (10%) in the pars flaccida (upper middle photo), nine (45%) in the anterior-inferior quadrant (upper right photo), three (15%) in the posterior-inferior quadrants (lower left photo), two (10%) in the posterior-superior quadrant (lower middle photo), one (5%) marginal perforation (lower right photo)



addition, it was suggested that epidural tears at pressures above this level (pressures between 171.99–246.65 kPa, i.e., 1290–1850 mmHg; depths between 17.6–25.2 msw) might be seen.[6] In a scuba diver who descended 9.14 msw (92.03 kPa) and developed otalgia, vertigo and hearing loss, computed tomography showed haemorrhage in the temporal lobe due to barotrauma causing the rupture of the tegmen tympani (gas in the middle cranial fossa).[19] In this regard, knowledge of the perforation pressure threshold of the TM may be useful for otologists to evaluate possible pathological lesions located at the brain or temporal bone. The vast majority of information on perforation pressure in the literature was obtained from animal models.[6,17,18,20,21] However, those studies focused on different animals (e.g., rabbit, dogs, cattle, foxes, cats, or guinea pigs) and indicated that the perforation pressure of the TM in humans was greater than that in other species.[6,17,18,20,21] Considering that the data focusing on humans were limited and contradictory,[7,8,14] the present study aimed to further investigate perforation pressure of the TM in humans to better define the tensile strength of the membrane.

Pre-existing data related to perforation pressure of the TM in human cadaveric models are given in Table 3. The mean perforation pressure (97.71 (SD 36.20) kPa, range 35.79–151.78 kPa) in the present study was lower than those (means 117.68–172.37 kPa, range 40.53–303.97 kPa) reported in previous articles[7,8,14] that used similar methodology. However, the present study mean perforation pressure was higher than those studies which tested the effect of blast overpressure (i.e., sound wave) (21–62.1 kPa).[22,23] The present perforation pressure was also higher than reported to be provoked by ear irrigation with water (32 kPa, range: 26.66–40 kPa).[24] Proposed reasons for differences in human TM perforation pressures include: demographics (e.g., region, age); methodology (e.g., transducer or bicycle pump); anatomical variations (e.g., the size and shape of the TM, external ear canal, or pinna); and present or past pathologic lesions (e.g., scarred TM or eustachian tube dysfunction).[7,21] Similar to the present work, one study reported that sex and side differences did not affect the bursting pressure of the TM.[14] Some authors have observed that the perforation pressure was higher in children and decreased with age.[7,14] The perforation pressure in a German study[14] (160.09 kPa) was higher than that in a Danish study[7] (117.68 kPa) raising the possibility of regional differences. The perforation pressure in the present study (syringe and transducer) was lower than studies using a bicycle pump[7,14] and sphygmomanometer;[8] thus the pressure generator and measurement device might account for differences between studies. Some authors[7,14] found that the bursting pressure of a scarred TM (29.42–78.45 kPa) was significantly lower than that of a normal TM (40.53–303.97 kPa). One group suggested that the scarred TM could rupture when descending to 3 msw (30.20 kPa).[7] Therefore, many factors potentially influence the perforation pressure of the TM. Given that the TM can rupture during many activities such as spearfishing, swimming, diving, freediving, or scuba diving, we believe that the minimum values of bursting pressures reported

Table 3

Studies reporting TM perforation pressures expressed both in kPa and seawater depth equivalent (standard deviation) [range]. CB – closed bulla; msw – metres' sea water; OB – open bulla; PP – perforation pressure; PPMT – perforation pressure measurement technique; Sphygmo – sphygmomanometer

Study	Year	Region	*n*	Species	TM	PPMT	PP (kPa)	PP (msw)
14	1906	Germany	111	Humans	Normal	bicycle pump	160.09 [40.53–303.97]	15.90 [4.02–30.19]
			12	Humans	Atrophic	bicycle pump	51.68	5.13
			12	Humans	Scarred	bicycle pump	30.40	3.02
7	1993	Denmark	144	Humans	Normal	bicycle pump	117.68 [49.03–205.94]	11.67 [4.87–20.45]
			23	Humans	Scarred	bicycle pump	58.84 [29.42–78.45]	5.84 [2.92–7.79]
18	2003	Australia	9	Pigs	Normal	Pressure gauge	121.59 (30.40)	12.07 (3.02)
17	2000	Norway	26	Cattle	Normal	Pressure gauge	39.52 [17.23–82.68]	3.92 [1.71–8.21]
			5	Foxes	Normal	Pressure gauge	59.78 [52.18–72.35]	5.94 [5.18–7.18]
20	1942	USA	–	Cats	Normal	Manometer	11.15	1.11
6	1971	USA	9	Guinea pigs	TM+OB	Transducer	26.66 (4.80) [18.53–32.53]	2.65 (0.48) [1.84–3.23]
			9	Guinea pigs	TM+CB	Transducer	33.46 (5.47) [25.86-41.60]	3.32 (0.54) [2.57–4.13]
8	1958	USA	15	Humans	Normal	Sphygmo	172.37 [96.53-227.53]	17.12 [9.59–22.60]
22	2019	USA	16	Humans	Normal	Blast chamber	32 [21–61]	3.19 [2.08–6.06]
23	2018	USA	41	Humans	Normal	Test chamber	[52.40–62.10]	[5.20–6.17]
24	1995	Denmark	20	Humans	Normal	Transducer	32 [26.66–40.00]	3.18 [2.65–3.97]
Present study	2020	Turkey	20	Humans	Normal	Transducer	97.71 (36.20)	9.70 (3.59)

Table 4

Studies reporting TM dimensions. All studies used dissected cadaveric ears. TMA – the area of the TM; TMHD – the horizontal diameter of the TM; TMVD – the vertical diameter of the TM; y – years

Study	Year	Region	*n*	Age	TMVD (mm)	TMHD (mm)	TMA (mm^2)
2	1991	Belgium	–	Adult	–	–	59.74–65.35
28	1960	Japan	25	Adult	7.50 (0.50)	7.90 (0.80)	55.40 (4.50)
29	1970	USA	20	Adult	9.00–10.20	8.50–9.00	–
30	1991	Italy	280	Adult	9.40 (1.50)	8.60 (0.90)	–
31	1987	Israel	28	Adult	8–9	9–10	–
32	1993	Australia	3	0–0.5 y	9.3 (0.3)	8.7 (0.6)	–
			4	2–4 y	9.1 (0.6)	9.0 (0.7)	–
			5	4–6 y	8.9 (0.4)	9.4 (0.2)	–
			3	6–8 y	9.5 (0.5)	9.0 (0.9)	–
			3	8–10 y	8.8 (0.3)	9.0 (0.9)	–
			2	10–14 y	8.8 (0.4)	9.5	–
			7	14–18 y	9.4 (0.3)	9.3 (0.6)	–
			3	> 18 y	9.0	9.3 (0.4)	–
Present study	2020	Turkey	20	Adult	9.54 (1.27)	7.99 (1.08)	57.46 (16.23)

in the experimental studies (35.79–96.53 kPa) should be taken instead of the average values (97.71–172.37 kPa) as the danger threshold for the membrane (taking into account studies using methodology similar to our study). In this way, possible middle/inner ear damage due to barotrauma with or without coexistent pathologies such as brain injury might be prevented.

Perforation of the pars flaccida was found in 2/20 of cases depending on the bursting pressure, while the pars tensa perforated in the other 18 cases (Figure 4). The present finding of perforation predominantly in the anterior quadrants (60%) was compatible with previous data (~63% in two studies).[7,8] These findings indicate that the posterior quadrants have more elastic fibers than the anterior quadrants.[7] In clinical studies, perforations are mostly found in the central or anterior-central part of the TM.[25–27] For example, one study[26] reported that the TM perforation rate was 2.8% in the attic region (i.e., pars flaccida), 38.2% in the central region, 7.4% in the marginal region, 32.3% in the anterior-central region, and 19.3% in the posterior-central region.[26] These clinical findings are therefore broadly compatible with experimental findings.

Studies reporting TM dimensions are summarised in Table 4,[2,28–32] The data are broadly confluent with the present study findings; 9.54 (SD 1.27) mm for the TMVD, 7.99 (1.08) mm for the TMHD, and 57.46 (16.23) mm^2 for the TMA. In Gray's Anatomy,[1] narrow ranges for TMVD (9–10 mm) and TMHD (8–9 mm) were cited while others have quoted different measurements (TMVD 10 mm and TMHD as 5 mm).[5] It has also been claimed that the TMHD (9–10 mm) was greater than the TMVD (8–9 mm).[31] However, the present study found that the TMHD was statistically smaller than the TMVD as reported by others.[29,30,33] It has been suggested that measurement variations between studies may be attributable to the methodology (e.g., in situ vs. ex situ measurement).[28,30] The present study showed that the measurement range was quite wide (7.23–11.61 mm for the TMVD, and 5.85–9.74 mm for the TMHD). This information may be beneficial for otologists during preoperative graft design. It is notable that Treacher Collins syndrome, congenital aural atresia, and congenital cholesteatoma may be associated with anomalies of the TM;[34–38] therefore, knowledge of TM size in normal ears may be useful for interpreting anatomical variations of the EAC and middle ear in patients with congenital anomalies.

Conclusion

The TM can rupture during many activities such as spearfishing, freediving, and scuba diving. This may be complicated by more serious problems including brain injuries. The establishment of accurate estimates of perforation pressure through experimental studies, cadaveric studies and clinical cases is of fundamental importance. Minimum values of the experimental studies (35.79–96.53 kPa) might better represent the danger threshold for the bursting pressure of the TM than average values (97.71–172.37 kPa) in the prevention of TM damage.

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A survey on the health status of Dutch scuba diving instructors

Prashant Komdeur[1], Thijs T Wingelaar[2,3], Rob A van Hulst[3]

[1] *Sports Medical Center Papendal, Hengstdal 3, 6574 NA Ubbergen, the Netherlands*
[2] *Diving Medical Center, Royal Netherlands Navy, Den Helder, the Netherlands*
[3] *Department of Anaesthesiology, University Medical Center, Amsterdam, the Netherlands*

Corresponding author: *Dr Prashant Komdeur, Sports Medical Center Papendal, Hengstdal 3, 6574 NA Ubbergen, the Netherlands*
p.komdeur@smcp.nl

Key words
Age; Drugs; Fitness to dive; Health surveys; Medications; Medical conditions and problems; Risk factors

Abstract
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Introduction: As the diving population is ageing, so are the diving instructors. Health issues and the use of prescribed medications are more common when ageing. The death of two diving instructors during one weekend in 2017 in the Netherlands, most likely due to cardiovascular disease, motivated investigation of the prevalence of relevant comorbidities in Dutch diving instructors.
Methods: All Dutch Underwater Federation diving instructors were invited to complete an online questionnaire. Questions addressed diving experience and current and past medical history including the use of medications.
Results: A response rate of 27% yielded 497 questionnaires (87% male, average age 57.3 years [SD 8.5]). Older instructors were over-represented among responders (82% of males and 75% of females > 50 years versus 66% of males and 51% of females among the invited cohort). Forty-six percent of respondents reported no current medical condition. Hypertension was the most commonly reported condition followed by hay fever and problems equalising ears and sinuses. Thirty-two percent reported no past medical condition. Problems of equalising ears and sinuses was the most common past medical condition, followed by hypertension, joint problems or surgery, and hay fever. Fifty-nine percent used non-prescription medication; predominantly analgesics and nose or ear drops. Forty-nine percent used prescription medicine, mostly cardiovascular and respiratory drugs. Body mass index (BMI) was > 25 $kg \cdot m^{-2}$ in 66% of males and 38% of females. All instructors with any type of cardiovascular disease were overweight.
Conclusions: Nineteen percent of responding diving instructors suffered from cardiovascular disease with above-normal BMI and almost 60% used prescribed or non-prescribed medication. Some dived while suffering from medical issues or taking medications, which could lead to medical problems during emergency situations with their students.

Introduction

In 2017, during one weekend, three experienced Dutch scuba divers (two diving instructors), all of them over 60, died during their dive. Two of them were taking medication for cardiovascular disease, so the cause of death was considered to be cardiac. These events motivated us to investigate the prevalence of relevant comorbidity in Dutch diving instructors.

The scuba diving community is ageing.[1,2] In general medicine, the ageing population shows an increase in co-existing medical conditions, whether recognised or not.[3] Studies show that experienced recreational scuba divers continue to dive despite medical contraindications.[2,4] Older scuba divers are overrepresented in fatality reports, and co-existing medical conditions are one of the factors.[5] The DAN Annual Diving Report has shown that most fatalities occur between the ages of 50–59 in US divers and it is hypothesised that cardiovascular disease is a major factor.[6,7] There are currently recommendations for the medical (cardiovascular) fitness of recreational divers; however, there are no specific recommendations for diving instructors.[8] In the Netherlands, diving instructors are currently not obliged to comply with annual or recurring medical evaluations by a physician.

The aim of this descriptive study was to obtain information about the current and past medical history of Dutch scuba diving instructors in order to define future guidelines for the physical assessment of scuba diving instructors.

Methods

CONTEXT

The Dutch Underwater Federation (DUF) has 14,000 members and is the largest national diving organisation in the Netherlands providing education in diving training and issuing certifications. Levels of diving instructor are: one star, two-star, instructor trainer and instructor teacher.

In November 2018, all of the 1,819 DUF diving instructors received a regular newsletter in which they were invited to participate voluntarily in an online survey about diving experience and actual and past medical history related to scuba diving. Although this survey was exempt from the Medical Research Involving Human Subjects Act (WMO), we adhered to the guidelines as defined in the Declaration of Helsinki of the World Medical Association and the Association of Universities in the Netherlands (VSNU).[9,10]

SURVEY

The survey, which was designed in collaboration with the medical information technology faculty of the Amsterdam University Medical Center, consisted of a three-part questionnaire. The full questionnaire is shown in the Appendix* available on-line. The first part of the survey covered questions about demographics. The second part included questions on current and past medical conditions. A current condition was a condition present at the time of the survey or in the past year. A past condition was a condition that has been present at least a year before the time of the survey and may have disappeared or has led to a current condition. These conditions were divided into the following categories: cardiovascular, respiratory, ear, nose and throat (ENT) or eye diseases, neurological, psychiatry and 'other', which included musculoskeletal and endocrine problems. In the final part, the diving instructors were asked if they use prescribed and/or non-prescribed pharmaceutical agents. All the categories were subdivided to provide some more detailed information about the type of medication used.

DATA HANDLING

Responses were downloaded into Microsoft Excel (version 16.37, 2020, Microsoft Corporation, Redmond WA) for collation. All reported conditions were included in the analysis. A descriptive analysis based on means and standard deviations (SD) was conducted using Microsoft Excel.

Results

After a five-month period in which the instructors were able to complete the online questionnaire, 497 out of the 1,819 instructors (27%) completed the survey; 432 (87%) men and 65 (13%) women. This gender balance was in accordance with the group of diving instructors as a whole. Of the respondents, the average male age was 57.3 (SD 8.5) years and the average female age was 55.5 (SD 10.0) years In the respondent group 82% of the males and 75% of the females were older than 50 years of age, while in the instructor group as a whole the corresponding proportions were 66% and 51%, respectively. The average Body Mass Index (BMI) for males was 26.7 $kg{\cdot}m^{-2}$ and for females 25.3 $kg{\cdot}m^{-2}$. Table 1 summarises these results and provides details per 10-year age groups. Of the total male respondents 50.9% were overweight (BMI 25–30 $kg.m^{-2}$) and 15.3% obese (BMI > 30 $kg.m^{-2}$). About 29.2% and 14.9% of the female population were overweight and obese respectively.

When asked about their diving experience, 65% of the men and 51% of the women had more than 20 years of scuba diving. About 59% made between 1–50 dives in the past year and 30% made between 50–100 dives. A previous diving-related injury was reported by 27% where ENT problems (16%) and decompression illness (4%) were the most common, with others due to toxic or dangerous sea animals (3%) and hypothermia (2%). Tobacco use in this population was quite low: 95% of the men and 91% of the woman did not currently smoke. Of the respondents, 41% of the men and 34% of the women consumed 1–5 alcohol units per week.

Table 2 shows the current and past medical issues (all percentages are shown in relation to all 497 respondents): 231 (46%) of the respondents did not mention any current medical condition. The most prevalent current medical condition was hypertension (75, 15%), followed by hay fever (69, 14%), and equalising problems of the ears and

Table 1
Age and body mass index (BMI) ($kg{\cdot}m^{-2}$) of the scuba diving instructors

Age Years	Male *n* (%)	BMI male Mean (SD)	Female *n* (%)	BMI female Mean (SD)
21–30	3 (1)	25.4 (2.4)	3 (5)	22.6 (1.4)
31–40	13 (3)	22.8 (2.0)	2 (3)	24.2 (3.0)
41–50	59 (14)	27.0 (3.7)	11 (17)	26.4 (2.7)
51–60	211 (49)	26.9 (3.2)	30 (46)	26.1 (5.2)
61–70	122 (28)	26.8 (3.1)	16 (25)	23.8 (3.4)
71–80	24 (6)	26.1 (2.3)	3 (5)	25.4 (1.7)
Total	432 (87)	26.7 (4.6)	65 (13)	25.3 (4.3)

Footnote: * The Appendix is available on DHM Journal's website: https://www.dhmjournal.com/index.php/journals?id=82

Table 2
Current and past medical conditions among scuba diving instructors. ENT – ear nose throat, MSK – musculoskeletal

Condition	Current *n* (%)	Past *n* (%)
Cardiovascular	**92 (18.5)**	**92 (18.5)**
Hypertension	75 (15.1)	73 (14.7)
Heart rhythm disorder	13 (2.6)	12 (2.4)
Heart valve disease	4 (0.8)	6 (1.2)
Coronary artery disease	1 (0.2)	2 (0.4)
Myocardial infarction	1 (0.2)	2 (0.4)
Angina pectoris	1 (0.2)	2 (0.4)
Persistent (patent) foramen ovale	–	1 (0.2)
Respiratory	**81 (16.3)**	**84 (16.9)**
Hay fever	69 (13.9)	62 (12.5)
Asthma	9 (1.8)	10 (2.0)
Pulmonary infection	4 (0.8)	12 (2.4)
Chronic obstructive pulmonary disease	5 (1.0)	6 (1.2)
Sarcoidosis	–	1 (0.2)
Other	**80 (16.1)**	**127 (25.5)**
Joint problems or joint surgery	31 (6.2)	69 (13.9)
Miscellaneous	31 (6.2)	44 (8.9)
Diabetes I / II	17 (3.4)	15 (3.0)
Rheumatoid arthritis	6 (1.2)	5 (1.0)
Crohn's disease or ulcerative colitis	2 (0.4)	3 (0.6)
Stomach ulcer	–	3 (0.6)
ENT / Eye	**75 (15.1)**	**115 (23.1)**
Equalising problems ears or sinus	64 (12.9)	83 (16.7)
Perforated eardrum	6 (1.2)	20 (4.0)
Eye surgery	1 (0.2)	11 (2.2)
Chronic sinusitis	2 (0.4)	5 (1.0)
Retinal detachment	1 (0.2)	2 (0.4)
Inner ear surgery	–	2 (0.4)
Chronic Otitis media	1 (0.2)	1 (0.2)
Neurological	**33 (6.6)**	**60 (12)**
Migraine	16 (3.2)	24 (4.8)
Spinal hernia	4 (0.8)	17 3.4)
Recurrent headache	12 (2.4)	13 (2.6)
Brain tumor	2 (0.4)	2 (0.4)
Transient ischaemic attack	–	4 (0.8)
Meniere's disease	–	3 (0.6)
Epilepsy	1 (0.2)	1 (0.2)
Brain- or spinal cord injury	–	1 (0.2)
Psychiatric	**13 (2.6)**	**24 (4.8)**
Depression	7 (1.4)	17 (1.3)
Attention deficit hyperactivity disorder. Attention deficit disorder	3 (0.6)	3 (0.6)
Claustrophobia	2 (0.4)	2 (0.4)
Anxiety disorder	1 (0.2)	2 (0.4)
Drug or alcohol addiction	–	1 (0.2)
No medical condition	**231 (46.5)**	**160 (32.2)**

Table 3
OTC (Over-the counter) medications used by 497 instructors. More than one agent may be used

OTC medication	*n* (%)
Analgesics	185 (37.2)
Nose/eardrops	105 (21.1)
Antihistamines	38 (7.6)
Motion sickness	38 (7.6)
Antacids	37 (7.4)
Antidiarrhoeal drugs	23 (4.6)
Other	23 (4.6)
Antiemetics	3 (0.6)
None	203 (40.8)

sinuses (64, 13%). Cardiac problems (angina pectoris, coronary artery disease, myocardial infarction, heart valve disease and arrhythmias) accounted for 4% of the current medical conditions. Pulmonary problems (asthma, COPD and pulmonary infection) accounted for about 16% of the current medical conditions.

Concerning the past medical history, 160 of the respondents (32%) did not report any past medical condition. Equalising problems of the ears and sinuses (83, 16.7%) was the most common past medical condition, followed by hypertension (73, 14.7%), joint problems or surgery (69, 13.9%), and hay fever (62, 12.5%).

Regarding medication, 59% used over-the-counter (OTC) medication (Table 3). Most respondents mentioned analgesics, followed by decongestants (nose sprays and eardrops) and motion sickness medications and antacids. With respect to prescribed medication (Table 4), 28% consisted of medication prescribed for cardiovascular diseases (such as cholesterol-lowering medications and diuretics), which accounted for the largest group, followed by prescribed medication such as analgesics and antibiotics (24%). The third group contained medications prescribed for respiratory problems (10%), like corticosteroids inhalations, long-acting beta-2-agonist and nasal anti-inflammatory sprays.

One or more current medical conditions were reported by 266 (53.5%) of the instructors and 337 (67.8%) had one or more past medical conditions. OTC medications were used by 294 (59.1%) and 242 (48.7%) used one or more prescribed medication. Table 5 shows the distribution of the multiple medical conditions and multiple medications.

Of the 432 male instructors, 75 (17.4%) and 357 (82.6%) < 50 and ≥ 50 years respectively. Clinical conditions related to cardiovascular disease (angina pectoris, coronary artery disease and previous myocardial infarction) were present in 4 of the 75 male instructors < 50 years of age, and in 82 of the 357 (23%) ≥ 50 years. All male instructors with cardiovascular disease had a BMI above 25 $kg \cdot m^{-2}$. Female

Table 4
Prescribed medications used by 497 instructors. More than one agent may be used. H2 – histamine receptor 2; NSAIDS – non-steroidal anti-inflammatory drugs; PPI – proton pump inhibitor

Prescribed medication	*n* (%)
Cardiovascular	**140 (28.1)**
Lipid lowering agents	40 (8.0)
Miscellaneous	33 (6.6)
Diuretics	24 (4.8)
Angiotensin converting enzyme inhibitor	20 (4.0)
Betablockers	17 (3.4)
Calcium channel blockers	6 (1.2)
Miscellaneous	**120 (24.1)**
Analgesics	47 (9.4)
Antibiotics	33 (6.6)
Rheumatoid medications	14 (2.8)
Antidiabetic drugs	12 (2.4)
Antimalarial drugs	8 (1.6)
Miscellaneous	5 (1.0)
Antiepileptic drugs	1 (0.2)
Respiratory	**52 (10.4)**
Nasal sprays with steroids	19 (3.8)
Inhaled steroids	17 (3.4)
Short-acting beta-2 agonists	12 (2.4)
Inhaled combined steroid and long acting beta-2 agonists	4 (0.8)
Gastrointestinal	**43 (8.6)**
PPI / H2 receptor antagonists	36 (7.2)
NSAIDS	7 (1.2)
Haematology	**26 (5.2)**
Aspirin	13 (2.61)
New generation anticoagulant	10 (2.0)
Vitamin K antagonist	3 (0.6)
Hormonal	**25 (5.0)**
Oral contraceptives	10 (2.0)
Thyroid medications	6 (1.2)
Insulin	3 (0.6)
Psychiatric	**6 (1.2)**
Antidepressants	6 (1.2)
None	**255 (51)**

instructors < 50 years did not suffer from cardiovascular disease. Of the female instructors ≥ 50 years, 5 out of 65 (8%) suffered from a cardiovascular problem, hypertension in particular.

Discussion

To our knowledge, this is the first study to describe the health status of Dutch diving instructors. About one in five of the males over 50 years old was overweight and suffered from

Table 5
Distribution of instructors with multiple medical conditions and multiple medications

n	Medical conditions		Medications	
	Current *n* (%)	Past *n* (%)	OTC *n* (%)	Prescribed *n* (%)
0	231 (46.5)	160 (32.2)	203 (40.8)	255 (51.3)
1	188 (37.8)	209 (42.1)	175 (35.2)	163 (32.7)
2	56 (11.3)	95 (19.1)	45 (9.1)	61 (12.3)
> 2	22 (4.3)	33 (6.6)	74 (14.9)	18 (3.6)

cardiovascular disease. Additionally, they suffered from several other comorbidities and 59% used OTC medications and 49% prescribed medication, while 106 of the 497 (21%) used one or more cardiovascular drugs. We could not find any comparative data describing the average age of diving instructors in other organisations in the Netherlands. In wider diver populations, the average age varies from 35 years old (PADI Members and Divers)[11] up to circa 50 years old (DAN Asia Pacific members or DAN USA members).[2,11,12]

Medical issues are more common with increasing age, especially cardiovascular problems. Hypertension was seen frequently in this diving instructor population as in previous studies on divers.[13,14] Pre-existing hypertension is one of the risk factors for developing immersion pulmonary oedema and it is therefore paramount to detect and treat it accordingly.[15,16] Diving physicians should continue to monitor whether diving with hypertension and the use of various antihypertensive drugs is advisable. Guidelines should be drawn up to define whether someone is fit to dive while taking particular medications.[17] The present results show that diving instructors with cardiovascular problems were also overweight, with more than 60% having a BMI above 25 $kg \cdot m^{-2}$, which is in line with other literature.[2,11–13] Analysis of the non-cardiovascular disease population also showed that they have a mean BMI 26.9 (SD 4) suggesting that this apparently healthy population could be at risk for cardiovascular disease.[18]

The most frequently reported medications in this survey were analgesics (both OTC and prescribed), nose/eardrops (OTC) and prescribed cardiovascular medication, which is consistent with a previous study.[19] Among cardiovascular medications, antihypertensive drugs are also the most common in Australian cohorts.[13] In the present study, cardiovascular medications were more prevalent than shown in other studies on divers, which could be explained by the older population compared to the aforementioned studies.[19] Reported present and past respiratory tract problems, such as asthma and COPD, were mentioned less frequently compared to some scuba diver cohorts.[13,20] This could be attributed to a healthy worker effect, although selection bias cannot be excluded because the present cohort smoked significantly less than the average Dutch population. On the other hand, in concordance with recent literature, the present study also found a low prevalence of mental health disorders. While this could also be attributed to a healthy worker effect, it might reflect a reluctance to report psychological issues.[4]

Some limitations of this study need to be addressed. Firstly, although a similar response rate (~30%) is common in online surveys, there may be a selection bias.[21] The male-female ratio of the respondents (87% men and 13% women) seemed to represent the whole diving instructor population (87% men) accurately. However, the survey respondents are older than the total group of dive instructors who were invited which could lead to overrepresentation of certain medical conditions. Of the respondents, 18% of the males and 25% of the females were younger than 50 years old, while in the instructor community 34% of the males and 49% of the females respectively were younger than 50 years old. The self-reported percentage of smokers is in line with other studies on general diving populations, with a rate between 5% and 11%.[2,12,19] Only 5% of the male instructors and 9% of the female instructors in the present study were smokers. This compares favorably to the national average of around 25%.[22] The 'previous smoking status' was not investigated in the present survey, so it cannot be determined if the current non-smokers never smoked or quit smoking. Secondly, there might be a recollection bias. Diving instructors mention some medical conditions as a past medical issue, such as hypertension, even though they still use antihypertensive medication. From the perspective of a physician this would be regarded as an ongoing medical condition, while the patient regards it as a past condition, with the hypertension now being normalised due to medication. The questionnaire did not ask for hyperlipidaemia or hypercholesterolaemia which could have provided more detail on cardiovascular risk.

From the diving instructor population that responded to the questionnaire, 20% had an increased risk of cardiovascular events perhaps reflecting the older age compared to the average diving population. Considering the increased responsibilities of an instructor, especially when teaching inexperienced divers, it could be argued that instructor should have a higher level of cardiovascular and physical fitness than the average diver. To date, no such special requirements are imposed on diving instructors. It has been suggested for recreational divers that high cholesterol, hypertension, high BMI and smoking status should be addressed during routine assessments of diving fitness by physicians to reduce the risk of mortality while diving[23] and this should also apply to diving instructors. Even though there is a continuing debate about the pros and cons of performing a periodic health examination of healthy divers, the present results show that assessing the cardiovascular and pulmonary status of diving instructors might be necessary in certain groups at risk. As a suggested minimum, diving instructors should consult a dive medical examiner regularly and discuss their fitness to dive. For example, cardiovascular risk can be assessed by means of the Systematic Coronary

Risk Evaluation (SCORE) and this could determine if further assessment is necessary.[24] The SCORE has high and low cardiovascular risk charts based on sex, age, total cholesterol, systolic blood pressure and smoking status. Additional testing, with a resting electrocardiogram and assessment of the physiologic reserve with a bicycle ergometer, can be considered to evaluate cardiovascular status.[25] The South Pacific Underwater Medicine Society diving medical provides an algorithm for the recommended cardiovascular screening of divers in which the SCORE can be used.[26] Another algorithm based on the Global Lung Initiative might be used for pulmonary assessment.[27] With effective usage of screening tools, not every diving instructor needs to be fully examined and everyone can get an appropriate health assessment. The authors also feel that educating diving instructors with respect to the use of prescribed and non-prescribed medications during diving is necessary to create greater awareness of safe diving with pharmaceutical agents.

Conclusions

This is the first questionnaire survey of Dutch diving instructors about their medical history and use of medications. Almost 20% of instructors ≥ 50 years old had cardiovascular disease (mainly hypertension) and obesity, which can lead to medical problems during emergency scenarios with their students. Although the value of regular diving-medical assessments is debated, certain populations, such as the aforementioned group, could benefit from a more frequent assessment. The use of cardiovascular and pulmonary screening tools by the medical diving examiner can help target the population which is at risk and can lead to appropriate additional assessment. Further research is necessary to evaluate these screening tools in the diving instructor population.

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Snorkelling and breath-hold diving fatalities in New Zealand, 2007 to 2016

John Lippmann[1,2], Chris Lawrence[3,4], Michael Davis[5]

[1] *Australasian Diving Safety Foundation, Ashburton, Victoria, Australia*
[2] *Department of Public Health and Preventive Medicine, Monash University, Victoria, Australia*
[3] *Department of Pathology, Christchurch Hospital, Christchurch, New Zealand*
[4] *Statewide Forensic Medical Services, Royal Hobart Hospital, Tasmania, Australia*
[5] *Honorary Senior Lecturer, Department of Anaesthesiology, School of Medicine and Health Sciences, The University of Auckland, Auckland, New Zealand*

Corresponding author: *Dr John Lippmann, Australasian Diving Safety Foundation, PO Box 478, Canterbury, VIC 3126, Australia*
johnl@adsf.org.au

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Apnoeic hypoxia; Cardiovascular; Diving deaths; Diving incidents; Drowning; Obesity; Pulmonary oedema

Abstract
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Introduction: New Zealand's (NZ) long coastline offers a diverse underwater environment with abundant opportunities for harvesting seafood and for recreation. Fatalities from snorkelling/breath-hold diving have been reported from the 1960s through to 2006. Those from 2007 to 2016 are reported here.
Methods: The National Coronial Information System, the Australasian Diving Safety Foundation diving fatality database, and the Water Safety NZ "*Drownbase*" were searched and additional coronial data provided by the NZ Ministry of Justice. An anonymised database was created and analysed for multiple factors. A chain of events analysis was performed for each case.
Results: There were 38 snorkelling or breath-hold-related deaths in NZ, 33 men and five women. Twenty-nine were breath-hold divers involved in gathering seafood, and six 'surface snorkellers', predominantly sightseeing. Two-thirds were diving alone and/or were not being observed by anyone out of the water. Twenty-eight victims were classified as overweight or obese and 19/38 were Māori. Pre-existing health factors that may have or definitely contributed to the fatality were present in 30 cases. The most common of these were cardiac (18/38). Two divers had insulin-dependent diabetes mellitus, one each epilepsy and asthma whilst cannabis and/or alcohol were possible factors in seven deaths. Five (possibly six) deaths resulted from apnoeic hypoxia.
Conclusions: Overall, death from snorkelling/breath-hold diving was an uncommon event (38 in 10 years). Poor judgement was a common feature. Middle-aged Māori men with pre-existing disease feature strongly. This suggests an on-going need for appropriate water safety education within and beyond the Māori community.

Introduction

With its 15,000 km of coastline bordered by the Tasman Sea and Pacific Ocean, New Zealand (NZ) offers an accessible and diverse underwater environment. With two main islands (North and South) and the smaller Stewart Island in the far south, NZ covers a latitudinal range of more than 12 degrees and offers a range of conditions, from sub-tropical with wide sandy beaches in the far north, to temperate waters and steep fiords in the south. The abundant marine life provides the opportunity for harvesting a variety of seafood (kai moana in Māori), which is a fundamental customary activity for Māori, and spearfishing is popular throughout NZ. Whales and dolphins are also common in some areas and provide the opportunity for close snorkelling encounters. Therefore, it is unsurprising that snorkelling and breath-hold diving are popular activities among locals and tourists, with 11% of 1,094 respondents to a 2018 online Water Safety New Zealand (WSNZ) survey of the NZ public reporting that they had snorkelled or dived during the previous year.[1]

As with any physical activity, especially in a potentially hostile environment, there is an associated risk of morbidity and mortality. Earlier reports have reviewed snorkelling-related deaths in NZ from 1961 through to 1973,[2,3] 1981–1986,[4–6] 1980–2000[7] and 2000–2006.[8] This study examines snorkelling and breath-hold diving-related deaths in NZ waters from 2007 to 2016, with the aim of identifying underlying factors and risks in order to assess and inform appropriate preventative measures.

Methods

This was a case series of snorkelling and breath-hold diving fatalities that occurred in NZ waters from 2007 to 2016, inclusive. For inclusion in this series, a victim must have

been reported to have been wearing at least a mask and/or was breath-holding to collect food.

ETHICS APPROVAL

Ethics approvals for the collection and reporting of these data were received from the Victorian Department of Justice Human Research Ethics Committee to access the National Coronial Information System (CF/18/12735),[9] the Chief Coroner, New Zealand Department of Justice, to access additional coronial records and WSNZ to access "*Drownbase*". The benefits of reviewing multiple data sources have been described previously.[10]

SEARCH

All fatalities reported to NZ coroners since July 2007 have been added to the Australian-based National Coronial Information System (NCIS).[9] A comprehensive key word search was made of the NCIS for snorkelling-related deaths from July 2007 to December 2016. Key words included snorkel*, spear fish*, underwater fish* and breath-hold div*. Data obtained from the NCIS was matched with that listed on the Australasian Diving Safety Foundation (ADSF) diving fatality database and "*Drownbase*" to minimise the risk of over- or under-reporting. Coronial data not included on the NCIS (i.e., prior to July 2007 and for 'open' cases) were provided by the NZ Ministry of Justice.

REVIEW PROCEDURE

The principal investigator (JL) reviewed all datasets to resolve any discrepancies between the various sources, then prepared initial incident summaries for each case, and created a protected Microsoft Excel® spreadsheet. These summaries were then independently reviewed by each of the co-investigators (CL and MD), any differences in interpretation debated, and consensus reached; CL focusing, in particular, on the reported autopsy findings. Based on these reviews, the Excel® database was finalised. A chain of events analysis (CEA) was performed for each case using a pre-prepared snorkelling CEA template, similar to one validated for scuba fatalities.[11]

Each CEA is based on the evidence in the coronial and autopsy reports. However, in some cases the authors disagreed with the interpretation of the findings, so the disabling agents and disabling injuries reported in the CEAs are based on our consensus interpretations, but the cause of death given is that of the pathologist conducting the autopsy or by the coroner where no autopsy was performed.

OUTCOME MEASURES

A range of outcome measures were extracted. Where available, these included demographics, health factors, training and experience, dive location and conditions, buddy circumstances and oversight, dive purpose and depth, equipment used, resuscitation factors; then a possible CEA of the incident was created. Descriptive analyses based on means and standard deviations (SD) or medians and ranges, and Mann-Whitney U and χ^2 tests for comparisons of age or BMI, as appropriate, were conducted using SPSS Version 25 (IBM Armonk, NY; 2017). The level of significance assumed was $P = 0.05$.

Results

From 01 January 2007 to 31 December 2016 there were 38 identified snorkelling or breath-hold-related deaths in NZ territorial waters, 33 men and five women (Table 1). Ethnicity is documented in NZ and 19 of the 38 victims were Māori, 16 of European decent and three of Asian descent. Thirty-two victims were NZ residents and six were tourists.

Mean (SD) age was 47 (13) years, and there were no differences in age between the sexes ($P = 0.34$) or between Māori and non-Māori ($P = 0.67$). Body mass index (BMI) was available for 35 victims (mean [SD] 29.2 [5.8] $kg{\cdot}m^{-2}$) and was similar between the sexes (29.3 [6.0] for 31 men, 29.1 [4.2] for four women). Fourteen of the victims were classified as overweight (11 men and three women; BMI 25–29.9 $kg{\cdot}m^{-2}$) and 14 were obese (13 men and one woman; BMI ≥ 30 $kg{\cdot}m^{-2}$) (Table 1). The mean BMI for Māori victims was higher than that of non-Māori ($P = 0.002$).

NCIS also included two deaths abroad of NZ citizens (one in Fiji, a woman run over by a boat, and one in the Cook Islands, a man who died from an acute cardiac event) which were investigated by the authorities in NZ. These and other deaths of NZ residents and citizens abroad, not investigated by NZ authorities, were not included.

LOCATION

Deaths occurred the length and breadth of NZ, from Stewart Island in the south to Raoul Island in the Kermadics; however, the majority (30) occurred in the warmer waters of the northern half of North Island. The fatal incident appears

Table 1
Body mass index (BMI) classification of 35 snorkelling fatality victims according to their ethnicity. Classification: normal (18.5–24.9); overweight (25–29.9); obese (≥ 30). There were no data for three divers. * $P = 0.02$ for the difference between Māori and non-Māori

BMI ($kg.m^{-2}$)	Māori ($n = 16$)	European ($n = 16$)	Asian ($n = 3$)
Normal (n)	2	2	3
Overweight (n)	3	11	0
Obese (n)	11	3	0
Mean BMI (SD)*	32.6 (5.9)	27.4 (3.5)	21.2 (0.7)

to have occurred on the surface in 25 cases, four underwater, whilst there were no data for nine.

ACTIVITY AND SETTING

All but three of the 38 fatalities occurred during private activities. Twenty-four were harvesting seafood (recreationally), another four were spearfishing, and six were sightseeing; two of these on commercial dolphin-watching tours and another on a commercial sightseeing tour. In a double fatality, one victim died while trying to rescue his friend who was unconscious on the seabed; another died attempting the body recovery of a scuba diver in shallow water. The activity was unknown in two cases. Based on the reported activities at the time, it seems reasonable to consider 29 as breath-hold divers as they were involved in hunting or harvesting seafood, whilst seven were designated as 'surface snorkellers', generally sightseeing. There was insufficient information on two individuals. Five freedivers succumbed to apnoeic hypoxia, whilst a sixth possibly did so.

HEALTH ISSUES

Pre-existing health factors that may have or definitely contributed to the fatality were present in 30 of the 38 cases. The most common of these pre-existing conditions were cardiac (18/38), particularly moderate to severe ischaemic heart disease (9) and left ventricular hypertrophy (13). Fourteen victims were classified as obese (BMI ≥ 30 $kg \cdot m^{-2}$), of whom nine also had known cardiac disease. Of the 16 victims who identified as Māori for whom the BMI could be calculated, 11 were obese. Two divers had insulin-dependent diabetes mellitus, whilst epilepsy (poorly controlled) or asthma contributed to two deaths. Medications being taken were reported in 14 victims, whilst the remainder were either reported to not be on any medication or there was no information. In only three deaths might prescription medicines have been relevant to the incident; multiple drug interactions likely contributing to cardiac arrhythmia in one, non-therapeutic levels of anti-epileptics in another and the third had an elevated clozapine level. Alcohol and/or cannabis was a likely factor in seven divers, including two who suffered apnoeic hypoxia.

TRAINING AND EXPERIENCE

Whether victims had received any training was not reported for 28 of the 38 victims. However, two victims were known to be untrained, one was snorkel-trained, six had scuba training and one had been a commercial paua (abalone) diver. The level of experience was not indicated in 12 cases. Nineteen victims were reported to have been 'experienced', six to have had some experience and one no experience, although no objective measures of experience were provided. In the majority of cases, the diver's swimming ability was not documented.

BUDDY AND OBSERVER CIRCUMSTANCES

Twenty-five of the 38 victims were alone at the time of the incident. Thirteen had set out solo, and another 12 had separated from their buddy or group before the incident. Four became separated during the incident, and nine had not separated. Twenty-three victims were not being observed or supervised by anyone out of the water. Up to 13 were being watched from the boat or shore, and there was no information on supervision in two cases.

ENVIRONMENT

Sea conditions (currents and/or surface swell/waves) may have contributed to 15 incidents. The depth of water where the victims were snorkelling was not reported in 15 of the 38 cases. However, in the 23 incidents where it was recorded, the median depth was 3 metres' seawater (msw) (range 1–22.5 msw). Twenty-six of the incidents were reported to have occurred at the surface, five underwater (at unknown depths) and where the incident occurred was unknown in seven cases.

EQUIPMENT

The reports varied in detail about exactly what equipment was worn by the victims. However, at least 30 were recorded as having worn masks, at least 25 had snorkels, at least 25 wore fins, and at least 29 wore wetsuits. Overall, at least 21 of the snorkellers were recorded to have been wearing mask, snorkel, fins and wetsuit. One victim had none of these but was included as he was breath-hold diving for seafood.

Thirty-one of the 38 victims were known to have been wearing weight belts when they set out and 21 of these were still wearing their belt when found. The amount of weights worn was only recorded in seven cases, ranging from 2.5 kg to approximately 18 kg (the latter reported by police to be approximately 9 kg over-weighted). One victim used an inflated tyre tube for support whilst 32 of the victims did not use a specific buoyancy aid, such as an adjustable buoyancy life jacket. There was no information in the other five cases.

RESCUE AND RESUSCITATION

Rescue attempts were made in 22 of the 38 cases, the remainder being body recoveries, whilst one diver was never found. Of the 29 cases where such information was available, some form of in-water resuscitation (presumably rescue breathing) was reported to have been attempted on only one victim. In the 36 cases with available information, basic life support (BLS) was performed on 22 victims on boat or shore. In the remaining incidents, BLS was not performed because of delays in recovering the victim's body or the body was not available. There were only five reports indicating that a defibrillator was used during resuscitation attempts. Four were in a hospital or paramedic setting, and

Table 2

Chain of events analysis for 38 snorkellers and breath-hold divers in New Zealand waters; CAD – coronary artery disease; COPD – chronic obstructive pulmonary disease; cerv. spond – cervical spondylosis; IDDM – insulin-dependent diabetes mellitus; IHD – ischaemic heart disease; IPO – immersion pulmonary oedema, NR – not recorded

Case	Sex	Age	BMI	Predisposing factor	Trigger	Disabling agent	Disabling condition	Cause of death
1	M	38	24.9	Activity; Planning (solo)	Extended apnoea	Apnoeic hypoxia	Asphyxia	Drowning
2	M	57	33.8	Health (obesity); Planning (solo)	Unknown	Unknown	Unknown	Drowning
3	F	47	NR	Health (cardiac); Equipment (snorkel)?	Environment (immersion) Aspiration?	Medical (cardiac)	Cardiac (Ischaemic and rheumatic valvular heart disease)	Cardiac
4	M	51	19.4	Health (cardiac)	Exertion	Medical (cardiac)? Buoyancy?	Cardiac (arrhythmia?)	Drowning
5	M	49	28.0	Health (asthma, cardiac)	Exertion Environment (immersion)	Medical (asthma, cardiac?)	Cardiac	Asthma
6	M	70	34.0	Health (cardiac, obesity)	Unknown	Medical (cardiac?)	Unknown	Drowning
7	M	26	21.6	Health (alcohol)	Unknown	Unknown	Asphyxia	Drowning
8	M	59	39.9	Health (cardiac, obesity, COPD)	Environment (immersion)?	Medical (arrhythmia?)	Cardiac (arrhythmia?)	Cardiac
9	M	70	27.1	Health (IDDM, neuropathic disease)	Unknown	Unknown	Asphyxia? Cardiac (arrhythmia?)	Drowning
10	M	55	30.4	Health (cardiac, obesity); Planning (solo) Equipment (weight belt, no fins)	Diver error OR Environment (conditions)	Buoyancy (overweighted)	Asphyxia	Drowning
11	F	27	34.9	Health (obesity, drug combination)	Unknown	Medical (arrhythmia?)	Cardiac (arrhythmia?)	Cardiac
12	F	56	25.1	Health (cardiac); Organisational (supervision); Equipment (no oxygen)	Environment (immersion) Aspiration?	Medical	IPO	IPO
13	M	58	31.8	Health (obesity, sarcoidosis)	Environment (immersion) Aspiration?	Medical? Aspiration?	Cardiac (arrhythmia?) Asphyxia?	Drowning
14	M	51	30.0	Health (cardiac, obesity)	Environment (conditions, immersion?)	Medical (IHD)	Cardiac (arrhythmia?)	Cardiac?
15	M	42	33.9	Health (obesity); Equipment (no fins) Planning (conditions)	Environment (conditions)	Environment (adverse conditions)	Asphyxia	Drowning
16	M	58	27.7	Health (cardiac)	Unknown	Unknown	Cardiac (arrhythmia?)	Drowning
17	M	24	25.2	Activity	Extended apnoea	Apnoeic hypoxia	Asphyxia	Drowning
18	F	58	29.4	Health (cardiac)	Environment (conditions, immersion?)	Environment (immersion) Medical (cardiac)?	IPO? Cardiac?	Drowning?

Table 2 continued.

19	M	30	NR	Planning (conditions) Equipment (overweighted)	Environment (conditions) Other (Cramp)	Buoyancy (overweighted)	Asphyxia	Drowning
20	M	25	21.7	Inexperience, poor skills	Unknown	Unknown	Asphyxia	Drowning
21	M	52	26.5	Activity	Extended apnoea	Apnoeic hypoxia	Asphyxia	Drowning
22	M	53	27.1	Planning (solo) Health (migraine)?	Unknown	Unknown	Asphyxia	Drowning
23	M	33	20.4	Planning (solo) Equipment (no fins or knife)	Environment (entrapment)	Buoyancy	Asphyxia	Drowning
24	M	53	29.3	Health (cardiac); Other (drug toxicity); Planning (no buddy system)	Unknown; Clozapine toxicity?	Unknown Medical (cardiac)?	Asphyxia? Cardiac (arrhythmia?)	Drowning
25	M	43	36.0	Health (obesity, cardiac)	Exertion	Medical (cardiac)?	Cardiac (arrhythmia?)	Drowning
26	F	64	27.1	Health (cervical spondylosis)	Other (light-headedness)?	Medical (cerv. spond.)?	Medical (stroke)	Cerebral ischaemia
27	M	41	36.7	Health (epilepsy, obesity, cardiac) Equipment (no mask, snorkel, fins)	Environment (immersion)? Breath-holding?	Medical (seizure; cardiac?)	Asphyxia	Drowning
28	M	33	39.0	Health (cardiac, obesity)	Environment (immersion)	Medical (cardiac)?	Cardiac (arrhythmia?) Asphyxia?	Cardiac
29	M	56	20.9	Health (alcohol) Planning (alcohol)	Environment (conditions)?	Environment (conditions)? Buoyancy?	Asphyxia	Drowning
30	M	25	NR	Planning (solo)	Unknown Extended apnoea?	Unknown Apnoeic hypoxia?	Asphyxia	Drowning
31	M	42	25.8	Planning (solo) Health (cannabis, smoker)?	Aspiration?	Unknown	Asphyxia	Drowning
32	M	53	42.4	Health (cardiac, obesity); Planning (conditions); Equipment (no fins)	Environment (current)	Environment (conditions)? Head trauma?	Asphyxia? Cardiac?	Drowning
33	M	37	27.8	Health (cannabis)?	Unknown	Unknown	Asphyxia	Drowning
34	M	31	23	Planning (night + alcohol) Equipment (overweighted)	Diver error (?intoxicated);Extended apnoea; Buoyancy (overweighted)	Apnoeic hypoxia	Asphyxia	Drowning
35	M	55	27.1	Health (alcohol); Planning (night) Equipment (overweighted)	Diver error (alcohol); extended apnoea; Buoyancy (overweighted)	Apnoeic hypoxia	Asphyxia	Drowning
36	M	62	35.8	Health (diabetes, hypertension, cardiac, obesity)	Environment (immersion) Exertion?	Medical (cardiac)	Cardiac	Cardiac
37	M	46	29.9	Health (issue from previous dive, hypertension, cardiac); Planning (solo)	Unknown	Unknown Medical?	Cardiac	Drowning
38	M	44	30.0	Health (undiagnosed CAD, cannabis); Planning (solo, conditions); Equipment	Environment (conditions); Exertion; Stress (likely)	Medical (CAD) Environment	Cardiac? Asphyxia?	Drowning

the other involved the use of a lifesaving club's defibrillator by an off-duty paramedic.

CHAIN OF EVENTS ANALYIS

Sixty-three possible or likely predisposing factors to the 38 incidents were identified, these included health-related (30), poor planning (14) and equipment-related (10). No triggers could be identified in 10 incidents and 44 possible or likely triggers were identified in the remaining 28 cases. The commonest of these were environmental (19), which included the cardiovascular effects of immersion *per se*, poor conditions and entrapment. Extended apnoea was implicated as the likely trigger in five incidents and a possible trigger in a sixth. Thirty-six possible or likely disabling agents were identified in 31 incidents, the main being medical (17), apnoeic hypoxia (6), and buoyancy problems (5). No disabling conditions (DC) could be identified with confidence in two cases. In another six cases the DC was unclear but likely to be either asphyxia or cardiac. In one case, the DC was likely to be either cardiac or immersion pulmonary oedema (IPO). The DCs identified in the remaining 30 cases were asphyxia (17), cardiac (10), stroke (1) and IPO (1). A possible chain of events analysis for each case is presented in Table 2.

AUTOPSY FINDINGS AND CAUSE OF DEATH

In two cases either no autopsy was done or the body was decomposed. Pulmonary oedema was a common finding (30/36). There were 15 medical conditions which likely acted as disabling agents mostly cardiac, with left ventricular hypertrophy in more than a third (13/36) of cases, moderate to severe ischaemic heart disease in nine and two cases of known valvular heart disease. Significant cardiac disease was found at autopsy in 20/36 (Table 3).

The cause of death was recorded as drowning in 29 cases and cardiac in five. There was one case of IPO which showed left ventricular hypertrophy and contraction band necrosis, a histological feature indicative of stressed myocardium from various causes and which has been reported in IPO.[12] In one case there was cervical spondylitis that was postulated to have caused brain stem ischaemia. Cause of death was undetermined in two cases.

Table 3

Autopsy findings of 38 snorkelling deaths (multiple abnormalities found with many, e.g., coronary atherosclerosis and left ventricular hypertrophy); * heart weight over normal range for BMI; † severe coronary artery disease > 75% vessel occlusion; ‡ moderate coronary artery disease between 50–75% occlusion; CoD – cause of death; -ve – negative

Conditions identified at autopsy	Divers (n = 38)
Pulmonary oedema fluid	30
Left ventricular hypertrophy	13
Cardiomegaly*	10
Severe coronary artery disease†	8
Moderate coronary artery disease‡	6
Significant drug effect	7
Elevated alcohol	4
Cardiac fibrosis	7
Cardiomyopathy	4
Genetic tests for long QT (all -ve)	4
Significant valvular heart disease	2
Asthma/chronic lung disease	2
Sarcoidosis of heart/lungs	1
Too decomposed to ascertain CoD	1
No autopsy	1

Discussion

Historically, NZ has had a high prevalence of unintentional (accidental) drownings, with the tenth highest death rate out of 32 Organisation for Economic Co-operation and Development (OECD) countries.[13] Over 2007–2016, the reported average annual preventable drowning death rate was 89 per 100,000 population, with snorkelling and breath-hold diving deaths from drowning (n = 29) representing only approximately 3% of these, similar to previous reviews.[7,8] From 1980 to 2005, there were a total of 74 snorkelling and breath-hold fatalities (average 3, range 1–6 per year);[7,8] so the rate covered in this report (4, range 1–9 per year) is similar despite an approximately 60% increase in the NZ population over that time.

As in the previous NZ reports,[7,8] most victims were male, although there appeared to be an increase in women dying (5/38 compared to 1/74). The average age has slowly increased from 34 years pre-2000 to 47 years in the present study. A similar increase in the age of snorkelling and breath-hold diving fatalities is seen in Australian data from the same period.[14] In many respects, it is difficult to compare the NZ studies since the quality of the data was limited in earlier reports. With respect to Australian fatalities, the patterns of diving and the people involved are different; predominantly food gathering in NZ compared to more-elderly tourists, inexperienced in snorkelling, visiting the Great Barrier Reef on commercial charter boats in Australia.[14–16] The increase in ages likely accounts for the higher incidence of cardiac deaths in the Australian cohorts.

One feature common to NZ and Australia is the relatively low numbers of breath-hold divers dying as a result of apnoeic hypoxia; 22 of 175 (12.5%) in Australia[14] and five (possibly six) of 37 in NZ. However, although relatively few, deaths from apnoeic hypoxia are often easily preventable by avoiding pre-dive hyperventilation and the pushing of breath-hold limits, as well as having close oversight by a

buddy who can perform a rapid rescue (the 'one up, one down' principle).

A large proportion of victims in this and other studies had pre-existing medical problems, especially cardiac disease and obesity, either known or identified at autopsy, which were likely contributory factors to their death.[7,15,16] This reflects that immersion *per se* and the physical challenges of being in the ocean, with waves and currents to contend with, are demanding and should never be taken lightly. When this is combined with commonly reported poor assessments of sea conditions and/or poor diving practices, particularly solo diving or separation from diving 'buddies', a potentially dangerous situation is created. A particular example of poor diving practices was that in none of the cases in which hypoxic apnoea was the likely cause of death was the diver practicing the 'one up, one down' safety routine. Although, compared to previous NZ studies,[3,7,8] the quality of information in the NCIS reports has improved considerably, there remain deficiencies such as in the recording of the equipment used, including the amount of weight carried. It appears a number of victims may not have been wearing swim fins, a concern identified in a previous NZ study.[7]

Māori, predominantly middle-aged men, consistently feature strongly in these studies (30/74 between 1980 and 2005[7,8] and 19/38 in the present study; overall 44%). Fifteen per cent of the NZ population were registered as Māori in 2010 so they are disproportionately represented in these data and in all other water safety statistics. However, adult Māori are more likely than other parts of the NZ community to participate in gathering seafood, which has a strong cultural tradition. Nevertheless, the frequent lack of proper equipment, especially swim fins, poor diving practices and poor judgement relating to individuals' health and physical capabilities and the sea conditions reflect a clear need for improved education within Māori communities. The same concerns were raised in two previous NZ studies.[7,8] Water Safety New Zealand and the NZ Police National Dive Squad have a number of on-going national programmes to promote water safety, including some specifically aimed at the Māori community. The data from this and a forthcoming report on NZ scuba-related deaths over the same period (2007 to 2016) will contribute towards these programmes. A recent large grant from the Accident Compensation Corporation will help to facilitate these educational endeavours.

In aquatic incidents it is important to get the victim onto a solid platform as soon as possible for assessment and commencement of resuscitation. BLS was not attempted, mainly due to considerable retrieval delays in up to 40% of these incidents, higher than in a comparable Australian series (29%).[17] This is likely largely a consequence of the higher prevalence of solo snorkelling in the NZ cohort (32% versus 26%) and the smaller proportion involved in a supervised activity (8% versus 37%). There was sparse information about resuscitation, including complications, use of supplemental oxygen and on-site defibrillation. Given the generally non-organised nature of snorkelling and breath-holding diving in NZ, it is likely such adjuncts were rarely available. However, such information is worth recording for later research into improving outcomes from future incidents.

CAUSE OF DEATH

Pulmonary oedema was recorded at autopsy in the majority of cases but is not particularly discriminating as it is present in drowning, drowning following a cardiac event, cardiac events and IPO. Its absence can suggest a cardiac event. In determining the cause of death at autopsy where there is evidence of significant heart disease and evidence of drowning it can be very difficult to tell whether:

- a cardiac arrhythmia preceded drowning due to a loss of consciousness (i.e., secondary drowning);
- the heart disease accelerated death due to drowning; or
- the heart disease played no direct role in the drowning.

Left ventricular hypertrophy is an independent predictor of sudden cardiac death, probably owing to cardiac arrhythmia, and its role in drowning is likely similar to that of severe ischaemic heart disease.[18] Cardiac pathology is commonly seen in snorkellers who die and seems to be important in causation of such fatalities.[14,19]

Careful description of the heart and lungs in autopsy reports is helpful.[20,21] Useful information includes:

- height and weight of the individual and heart weight relative to BMI or body weight;
- thickness of the right and left ventricles and maximum diameter of the left ventricle;
- degree of occlusion of the coronary arteries by atherosclerosis (macroscopic and microscopic);
- description of the heart valves;
- whether the lungs were over-expanded and presence of pulmonary oedema in the upper airway;
- histology, including cardiac fibrosis and contraction band necrosis; and
- results of toxicology.

Imaging of the body may be useful for examination of the sinuses for water but, unlike in scuba divers, is not necessary for detection of intravascular gas.

The diagnosis of drowning at autopsy is a diagnosis of exclusion in the appropriate circumstances. There was a significant number of Māori in this group. It is likely in the future that there may be more objections to autopsy of Māori victims on cultural grounds, which may obscure the role of heart disease, hypertension and obesity in these deaths.

LIMITATIONS

Even using multiple sources, it is possible that some fatalities were not recorded due to limitations in recording and NCIS searches. One additional case from 2016, which

was not identified in the databases or search, was later found incidentally, whilst several cases that were initially documented by authorities as snorkelling or breath-hold divers proved not to be so and were excluded. In previous studies,[2,7,8] a few cases in which the cause of death was not recorded as 'drowning', and not documented in WSNZ's Drownbase, may have been missed, but the current search was wider than those.

Information from immersion incidents is notoriously patchy and incomplete; especially when unwitnessed. However, in the majority of cases in this series, the coronial and autopsy reports were quite detailed and provided good insight into what happened. Health records were often deficient, so there is a strong subjective element to determining what personal factors contributing to a death were important. The CEA attempts to identify the predominant features of each case, but there always remains an element of uncertainty. Nevertheless, some clear lessons can be learned, such as the high frequencies of pre-existing deleterious medical conditions, the contribution of environmental conditions and/or poor diving practices and the disproportionate number of Māori.

Conclusions

Overall, death from snorkelling/breath-hold diving in NZ between 2007 and 2016 was an uncommon event (38 in 10 years), largely associated with seafood gathering, whilst only five (possibly six) deaths resulted from apnoeic hypoxia. Poor judgement was a common feature. Obese, middle-aged Māori men with pre-existing disease, particularly cardiac, feature strongly. This suggests, as it did two decades ago, an ongoing need for continued water safety education and appropriate medical surveillance of prospective aquatic participants both within and beyond the Māori community. There is also an ongoing need to remind all breath-hold divers of the potential for apnoeic hypoxia with extended breath-holding, with or without hyperventilation, and the benefit of having a vigilant buddy on-hand in the event of unconsciousness.

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Adjunctive hyperbaric oxygen treatment for necrotising soft-tissue infections: A systematic review and meta-analysis

Morten Hedetoft[1,2], Michael H Bennett[2], Ole Hyldegaard[1]

[1] *Department of Anaesthesia, Centre of Head and Orthopaedics, Rigshospitalet, University of Copenhagen, Denmark*
[2] *Department of Anaesthesia and Hyperbaric Medicine, Prince of Wales Hospital, Sydney, Australia*

Corresponding author: *Dr Morten Hedetoft, Department of Anaesthesia, Rigshospitalet, University of Copenhagen, Blegdamsvej 8, 2100 Copenhagen, Denmark*
morten.friis.fiskbaek.hedetoft@regionh.dk

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Abstract
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Introduction: Surgical intervention, broad-spectrum antibiotics and intensive care support are the standard of care in the treatment of necrotising soft-tissue infections (NSTI). Hyperbaric oxygen treatment (HBOT) may be a useful adjunctive treatment and has been used for almost 60 years, but its efficacy remains unknown and has not been systematically appraised. The aim was to systematically review and synthesise the highest level of clinical evidence available to support or refute the use of HBOT in the treatment of NSTI.
Methods: The review was prospectively registered (PROSPERO; CRD42020148706). MEDLINE, EMBASE, CENTRAL and CINAHL were searched for eligible studies that reported outcomes in both HBOT treated and non-HBOT treated individuals with NSTI. In-hospital mortality was the primary outcome. Odds ratio (ORs) were pooled using random-effects models.
Results: The search identified 486 papers of which 31 were included in the qualitative synthesis and 21 in the meta-analyses. Meta-analysis on 48,744 patients with NSTI (1,237 (2.5%) HBOT versus 47,507 (97.5%) non-HBOT) showed in-hospital mortality was 4,770 of 48,744 patients overall (9.8%) and the pooled OR was 0.44 (95% CI 0.33–0.58) in favour of HBOT. For major amputation the pooled OR was 0.60 (95% CI 0.28–1.28) in favour of HBOT. The dose of oxygen in these studies was incompletely reported.
Conclusions: Meta-analysis of the non-random comparative data indicates patients with NSTI treated with HBOT have reduced odds of dying during the sentinel event and may be less likely to require a major amputation. The most effective dose of oxygen remains unclear.

Introduction

Necrotising soft-tissue infections (NSTI) are a heterogeneous group of infections characterised by a rapidly progressive clinical course with necrosis of any layer of the soft-tissues.[1] NSTI encompasses a series of diseases including necrotising fasciitis, Fournier's gangrene and gas gangrene in which the conditions may differ due to different microbiological aetiology or anatomical site of infection; however, the clinical approaches to diagnosis and overall treatment remains identical. The annual incidence of NSTI varies considerably but is often reported at approximately four per 100,000 in developed countries.[2,3] Mortality rates highlight the severity of disease with a 90-day mortality of 18% reported in a multicentre study including more than 400 patients.[4]

The initial event in the onset of NSTI is the introduction of bacteria into the soft tissues through trauma (accidental or surgical) or spontaneously without a defined portal of entry (cryptogenic infection).[5] Rapid bacterial proliferation and endotoxin release cause a cascade of pathophysiological reactions including platelet-leukocyte aggregation, endothelial damage, capillary leakage and progressive occlusion of blood vessels that results in tissue hypoxia, oedema and necrosis.[5–8]

NSTI can be rapidly fatal. Early and radical surgery, broad-spectrum antibiotics and intensive care support remain the cornerstone of treatment.[9] Hyperbaric oxygen treatment (HBOT) might improve outcome when employed as an adjunct to conventional treatment and has been used in NSTI for almost 60 years.[10] Despite this, the use of HBOT remains controversial. It is not standard of care in many centres and a registry study in the USA suggested only 0.88% of cases received HBOT.[11]

HBOT involves the inhalation of 100% oxygen at pressures above 101.3 kPa (one atmosphere absolute [atm abs]). The precise protocol for NSTI varies among centres but

usually consists of one to two sessions of 60–120 minutes at 202.6–303.9 kPa (2–3 atm abs) within the first 24 hours. Thereafter, one to two daily sessions for several days or until further necrosis is no longer evident is a common protocol. The markedly increased serum partial pressure of oxygen during treatment results in a wide variety of biochemical effects which theoretically could improve the outcome of patients with NSTI.

The clinical evidence for the effectiveness of HBOT in these infections is sparse and of generally low quality. A Cochrane review highlighted the absence of randomised controlled trials (RCTs) in this area.[12] While a systematic review on the effectiveness of HBOT for NSTI has recently been published,[13] the combination of both newly published material and missing historical studies in that review have prompted this new and comprehensive systematic review with meta-analysis.

Within the field there is an understanding that a large RCT is required to properly define any place for the use of HBOT in these infections. The present aim was to synthesise the highest current level of clinical evidence in order to provide the best basis upon which to plan a subsequent multicentre RCT.

Methods

Eligibility criteria were agreed based on the formulation of a focused clinical question (Table 1). We included all trials reporting adult patients treated for NSTI and where the trial compared the effect of a regimen including HBOT with any treatment not including HBOT. HBOT was defined as 100% oxygen administered in a compression chamber between pressures of 152.0 and 405.2 kPa (1.5–4.0 atm abs) over treatment times from 30 to 120 minutes at least daily.

Table 1
PICO (population, intervention, comparison, outcome) criteria of included studies. HBOT – hyperbaric oxygen treatment; NSTI – necrotising soft tissue infection

Population	Adults with NSTI based on surgery
Intervention	HBOT
Comparison	HBOT versus Non-HBOT (sham or no treatment)
Outcome	***Primary*:** Mortality (In-hospital and 30-day) ***Secondary*:** Mortality (6 month and 1-year) Major amputation rate (above ankle/wrist or above) Number of surgical debridements Hospital length of stay Ventilator days Cost of therapy Functional outcomes (e.g., Quality of Life score) Adverse effect of all therapies

The primary outcomes were mortality during the sentinel admission and at 30 days from admission. The secondary outcomes were mortality at six months and one year, major amputation rate (above mid-foot), the number of surgical debridements, intensive care and hospital length of stay, mechanical ventilation days, the cost of therapy, quality of life scores and any adverse events of treatment (Table 1).

A comprehensive search of MEDLINE, EMBASE, CENTRAL and CINAHL was conducted, from inception to 20 April 2020. Citations in the included studies were searched for further comparative trials as were all previous relevant reviews available[12,13] and the US National Library of Medicine trials registry.[14] Authors of potentially eligible studies were contacted to provide any required data that would allow inclusion. The search strings used appear in Appendix 1*. Relevant journals and conference proceedings published since 1980 were hand searched (see Appendix 2*). No language restrictions were applied.

One author (MH) screened all identified citations by title and abstract. Potentially relevant studies were examined in full-text and independently reviewed by two authors (MH and MB) for compliance with eligibility criteria. Disagreements on eligibility were resolved by consensus. All studies where the full text was appraised were either accepted into the review or a reason given for rejection (Figure 1). Findings were reported in accordance with the "*Meta-analysis of Observational Studies in Epidemiology, (MOOSE)*"–guidelines (Appendix 3*).[15] This study was registered at the International Prospective Register of Systematic Reviews (PROSPERO), registration number CRD42020148706.

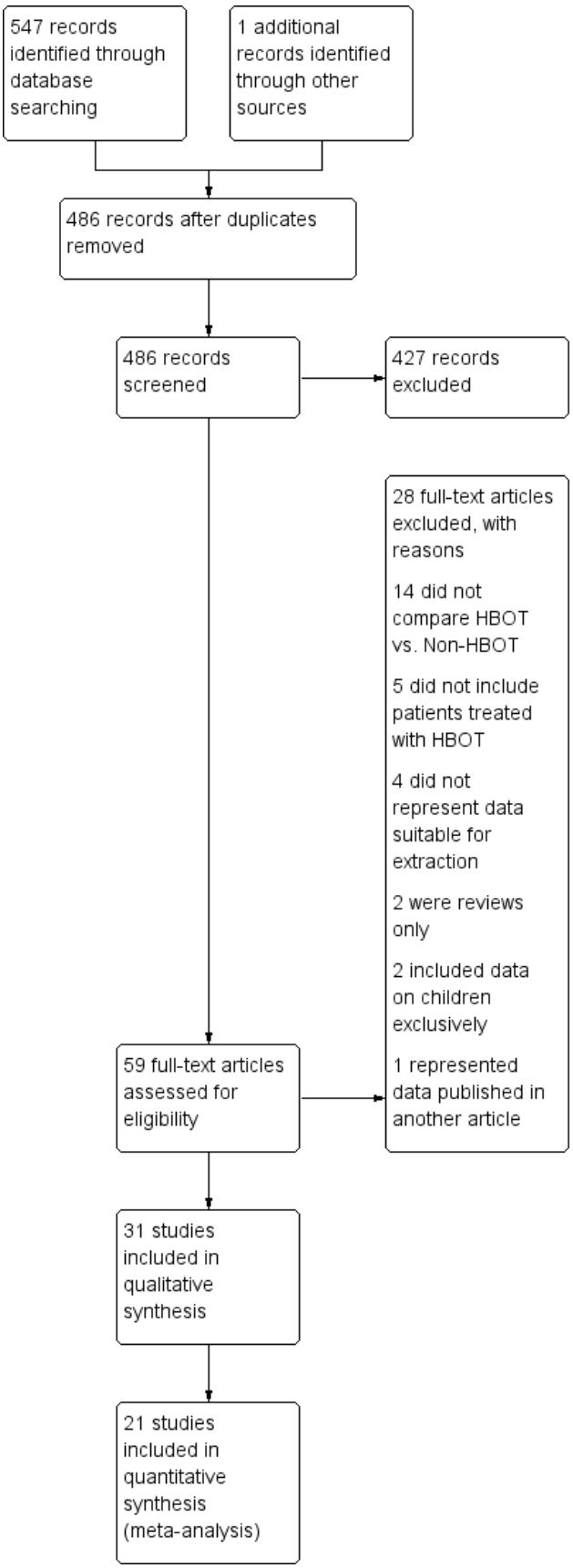
DATA EXTRACTION AND ANALYSIS

Two authors (MH and MB) independently extracted information into a pre-piloted data extraction form. Both the Newcastle-Ottawa Scale (NOS)[16] and the Cochrane-recommended ROBINS-I assessment[17] for non-random comparative trials were used (see Appendix 4*).

Review Manager 5.3 was used for pooled measures of treatment effect. Odds ratios (ORs) and 95% confidence intervals (95% CIs) were used for dichotomous outcomes. If there were no events in one arm an automatically fixed value of 0.5 of an event was applied to allow that study to contribute to analysis. If there were no events in either arm

Footnote: * Appendices 1–10 are available on DHM Journal's website: https://www.dhmjournal.com/index.php/journals?id=81

Figure 1
PRISMA flowchart for the review. HBOT – hyperbaric oxygen treatment



the study did not contribute to the analysis. For continuous data we used the mean difference (MD) between treatment and control groups in each trial and aggregated MDs using inverse variance weights to estimate an overall MD and 95% CI. A random-effect model was applied as clinical heterogeneity between studies was likely.

We considered clinical heterogeneity between studies and refrained from quantitative analysis where the heterogeneity was high. Statistical heterogeneity was assessed using the I^2 statistic and the appropriateness of pooling and meta-analysis was considered. Subgroup analysis based on the nature of the control group (historical versus contemporary), anatomical location (trunk versus peripheral), principal infecting organism and illness severity was also considered.

Sensitivity analyses for study quality were performed based on the inclusion and exclusion of those trials deemed to be at serious risk of bias. If inclusion of the latter did not substantially alter the result we chose to pool the two sub-groups. Studies at critical risk of bias were excluded from meta-analysis.

Results

The systematic search identified 486 studies. Of these, a total of 31 studies met the inclusion criteria (Figure 1).[11,18–47] All studies were retrospective observational studies and published between 1985 and 2020. Two (6%) of the included studies were written in languages other than English (German and Danish). Participant characteristics from all included studies are presented in Appendix 5*. Most included studies provided HBOT at 202.6–283.6 kPa (2.0–2.8 atm abs) for at least 90 minutes at different frequencies (Appendix 6*). Three (14%) of the included studies used historical non-HBOT controls, whereas 18 (86%) used contemporary non-HBOT controls. Study quality assessed by NOS and ROBINS-I are presented in Appendix 6*.

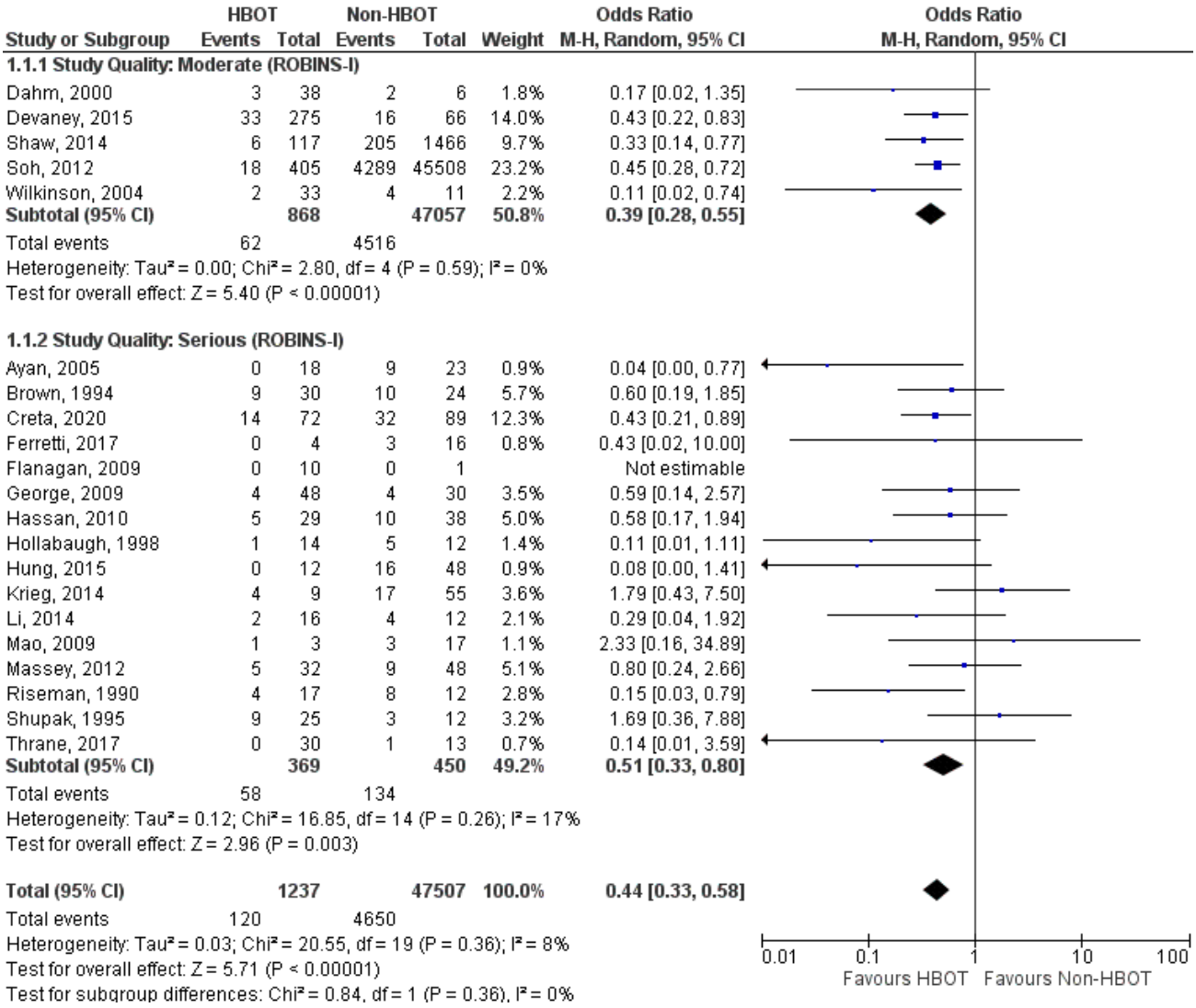
PRIMARY OUTCOMES

Mortality (31 reports)

No studies reported 30-day mortality. Reported mortality was interpreted as in-hospital mortality. Mortality was plotted chronologically and did not show any visual trend over time (see Appendix 7*).

Ten of the 31 studies were judged to be at critical risk of bias (ROBINS-I), and in line with Cochrane Collaboration recommendations were not included in the quantitative estimates.[48] Seven of these reported results in favour of HBOT.[18,19,28,32,40,42,43] The pooled estimates included 21 studies with a total of 48,744 participants and mean age from 43 to 67 years. Overall, the odds of dying after receiving

Figure 2
Forrest plot of the pooled effect of HBOT on in-hospital mortality. A random-effects model was used for meta-analysis



HBOT were lower, OR 0.44 (95% CI 0.33–0.58, I^2 = 8%, Figure 2). Sensitivity analysis for study quality did not substantially alter this estimate. One study[11] dominated the patient numbers, so a sensitivity analysis removing that study was performed. The results were not significantly affected: pooled OR 0.44 (95% CI 0.31–0.62).

Eighteen studies used contemporary controls and three historical controls. Subgroup analysis showed the pooled estimate was OR 0.24 (95% CI 0.03–1.87) for historical controls vs. OR 0.45 (95% CI 0.35–0.59) for contemporary controls (See Appendix 8*).

The possibility of publication bias was evaluated using visual assessment of the funnel plot (Figure 3). There is some suggestion of bias in favor of HBOT, with a paucity of smaller studies in the bottom right of the graph (smaller studies less favourable to HBOT are missing).

SECONDARY OUTCOMES

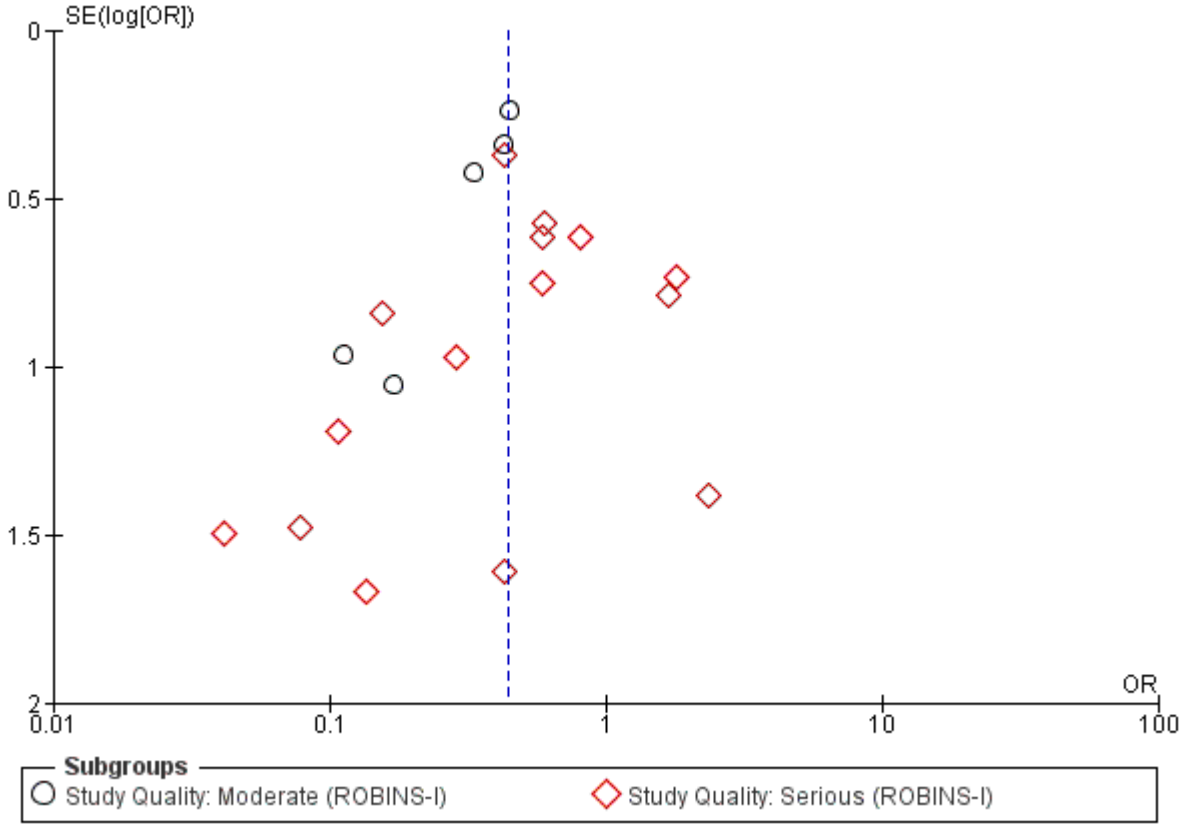
Major amputation (5 reports)

For the pooled estimate a total of five studies reported a total of 45,632 participants; three studies were of moderate quality.[11,23,37] Overall, the odds of requiring a major amputation with HBOT were 0.60 (95% CI 0.28–1.28, I^2 = 54%, P = 0.07. Figure 4).

Number of surgical debridements (11 reports)

Only one study[23] judged at low or moderate risk of bias could be included in this outcome and five studies judged at serious risk of bias were also included (see below). As the estimate of Devaney et al.[23] was very different to the other five, a combined estimate of effect is not provided (See Appendix 9*). Devaney et al.[23] enrolled 341 patients,

Figure 3
Funnel plot on primary outcome; in-hospital mortality



275 of whom received HBOT (81%). Analysis suggested there were more debridements in the HBOT group (mean 4.8 versus 3.0 per patient, difference 1.8 (95% CI 1.15–2.45), $P < 0.001$).

Five studies[21,24,26,29,35] were pooled and any effect of HBOT in these studies was unclear (MD 0.63 more debridements per patient with HBOT [95% CI -0.49–1.75], $I^2 = 90\%$). The high chance of important heterogeneity suggests this estimate should be treated with great caution.

Five further studies reported on this outcome but could not be included in the quantitative analysis. One[19] was judged at a critical risk of bias and reported a mean of 13.3 debridements in the HBOT groups compared to 4.8 in the non-HBOT group. Another[37] reported non-parametric data (median of 5 (IQR 1–16) debridements in the HBOT group and 1 (IQR 1–4) in the non-HBOT group. The third[39] included only one patient in one arm. The fourth[25] only reported the number of debridements in a sub-group of participants, and the last[34] did not provide standard deviations.

Hospital length of stay (6 reports)

Three studies[24,29,44] reported this outcome in 68 participants, MD -1.98 days (95% CI -9.93–5.97, $I^2 = 47\%$) (see Appendix 10*).

Additionally, three studies[11,23,38] reported non-parametric data with length of stay as medians with interquartile ranges. One study[23] demonstrated a median of 21.8 days (IQR 9–36.7) in the HBOT group and 24 days (IQR 10–39) in the non-HBOT group. The second[11] reported a median of 14.3 days (IQR 13–16) in the HBOT group and 10.7 days (IQR 10–11) in the non-HBOT group. The third[38] reported a median of 16 days (IQR 11–23) in the HBOT group and 14 days (IQR: 8–23) in the non-HBOT group.

Ventilator days (3 reports)

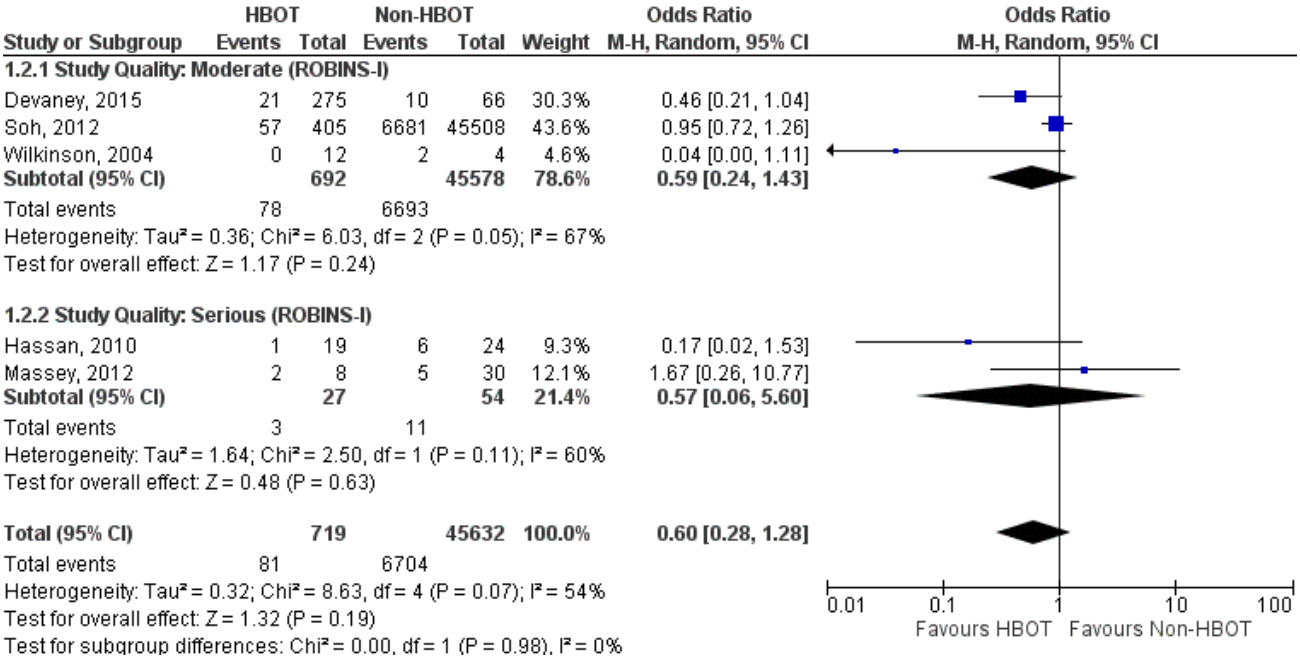
One study[21] reported ventilator days with a mean of 7.3 (SD 7.1) and 3.5 (SD 6.2) days in the HBOT and non-HBOT groups respectively. Another two studies[23,33] reported non-parametric data with medians of 4.9 in the HBOT groups and 2.6 and 2 in the non-HBOT groups, respectively.

Cost of therapy (3 reports)

Three studies[11,33,38] provided data on cost of therapy, but not in a uniform way to allow pooling the results. One[38] reported the cost of therapy was US$35,808 (IQR 23k–65k) in the HBOT group compared to US$27,504 (IQR 14k–51k) in the non-HBOT group. Another[11] reported US$107,000 in the HBOT group and US$86,000 in the non-HBOT group but

Figure 4

Forrest plot of the pooled effect of HBOT on risk of major amputation. A random-effects model was used for meta-analysis



the study didn't provide standard deviations of the reported means. The third[33] reported a median cost of US$63,199 (range 31,858–256,741) with HBOT and US$51,185 (range 8,691–427,283) without HBOT.

Discussion

To our knowledge, this is the most comprehensive analysis to date of the effect of HBOT for patients with NSTI. With data from 21 non-randomised studies including 48,744 patients, this meta-analysis indicates patients with NSTI treated with HBOT have reduced odds of dying during the sentinel hospital admission. This suggests a number needed to treat of approximately 19 patients with HBOT in order to prevent one death (calculated from OR).[49] Patients treated with HBOT may also be at a lower risk of major amputation (OR 0.6). Data on ventilator days and cost of therapy were not appropriate for meta-analysis. Both length of stay and the days on a ventilator may be affected by many factors, such as differences in the severity of illness and the use of intensive treatment regimens, but both may also simply reflect longer survival. The cost of therapy was rarely reported, and all studies that did so were from the United States. Caution is needed in extrapolating these costs to other systems where the cost of treatment is likely to be lower.

While there is some indication of a publication bias in favour of HBOT on inspection of the funnel plot, this is by no means established and the analysis suggests those reports at a lower risk of bias show a greater benefit with HBOT than those judged at higher risk of bias. A sensitivity analysis excluding the largest study[11] did not substantially affect the pooled estimate, indicating the overall result was not biased by the inclusion of this study and increasing confidence in the overall pooled estimate of effect.

Many older studies with poor methodology were identified, however a chronological assessment of the OR over time did not suggest an historical bias and the overall estimate of benefit with HBOT has been stable over time.

Most of the identified studies used a HBOT protocol of 90 minutes at 202.6–283.6 kPa (2.0–2.8 atm abs). However, treatment frequency varied greatly from once daily[20] to more aggressive treatment regimens with three sessions within 24 hours and thereafter twice daily.[24,34,37] Nine studies failed to provide any information on the treatment table used or frequency of treatment (Appendix 6*).

Treatment of NSTI requires a multidisciplinary approach including surgery, broad-spectrum antibiotics and intensive care treatment. Detailed information on the standard of care (e.g., type and dosage of antibiotics, number of surgical debridements and treatment interventions performed in the intensive care unit) is key when evaluating potential adjuncts to NSTI treatment. However, these were in general incompletely reported in the included studies. NSTI encompasses a variety of diseases (e.g., necrotising fasciitis, Fournier's gangrene and clostridial myonecrosis). While these diseases all produce widespread necrosis and require similar treatment, they differ substantially in aetiology, microbiology and anatomical site, and are a likely cause of clinical heterogeneity between included studies. Incomplete reporting meant we were unable to perform

any of our planned sub-group analyses to investigate the influence of clinical heterogeneity, including the dose of oxygen, anatomical location, principal infecting organisms and illness severity.

We have found limited data on which to base our estimates for all planned outcomes. While mortality was universally reported, the times from onset to death were not. Our own experience leads us to assume mortality here was for the sentinel event admission. Only the minority of the included studies reported comparable outcomes for our secondary endpoints. Future studies need to address endpoints with clear and reproducible definitions.

Pooled analysis of data from non-randomised studies remains controversial. Indeed, it has been suggested that pooling estimates in this area would be susceptible to high uncertainty and misinterpretation.[13] Critics have suggested that when meta-analyses include low-quality studies, fundamental errors will be transferred into the meta-analyses – the 'garbage in, garbage out' metaphor.[50] While we agree a meta-analysis can be misleading when confounders are not adequately addressed in the trial design and analysis, there is also a counter argument. Avoiding formal data synthesis and simply listing all the trials and their individual characteristics for the reader to interpret is unsatisfactory. It leaves the reader free to continue in their own biased interpretation and avoids a clear statement of the most likely consequences of adopting a particular treatment. Linked with sound interpretations of the implications for both practice and research, we believe meta-analysis can be justified. If the purpose of a systematic review is to inform the reader of the best evidence and also to inform future triallists of the most appropriate treatment and outcomes to include in any future study, then the calculation of an overall estimate of effect may do more good than harm.

A potential advantage of including non-randomised trials into systematic reviews is that they are more likely to include the full spectrum of patients and therefore be more generalisable to the population at large.[51] The inclusion criteria of the present systematic review are broad in order to reflect the variety of different aetiologies, pathogenic agents and anatomical locations. All are united by requiring the same multidisciplinary approach.

A good discussion of the potential mechanisms of action of HBOT highlights the importance of the controlled release of active oxygen and nitrogen species through the use of HBOT.[52] Several mechanisms have been proposed by which HBOT may achieve clinically important benefits in this group of infections. HBOT exposure at 222.9 kPa (2.2 atm abs) results in the achievement of gross arterial hyperoxia and a PaO_2 above 100 kPa is achievable with reasonable cardiorespiratory function.[53] Gross arterial hyperoxia results in vasoconstriction, increased oxygen diffusion distances, a reduction in leucocyte adherence, bacteriostasis and osmotic reduction in tissue oedema, all of which may be clinically important.

Hyperoxic vasoconstriction will maintain oxygen delivery while limiting or improving tissue oedema, extending the diffusion distance of oxygen and restoring local tissue oxygenation.[54,55] Elevated capillary oxygen tension will inhibit the adherence of neutrophils to damaged endothelium via a specific nitric oxide mediated pathway that inhibits β_2-integrin function. This prevents microvascular plugging and further tissue hypoxia without otherwise compromising neutrophil function.[56,57] The local release of reactive oxygen species in hypoxic tissues also has direct bacteriostatic effects, particularly against anaerobic bacteria, and enhances the antimicrobial effects of some antibiotics.[58–60] In addition, while biofilm formation in NSTI[61] protects bacteria utilising anaerobic metabolism from antibiotics in an hypoxic environment, HBOT may restore the susceptibility to antibiotics by inducing aerobic metabolism. This has been demonstrated in *Pseudomonas aeruginosa* and *Staphylococcus aureus* biofilm models.[60,62] Finally, HBOT may interrupt the pathology of NSTI by acting as an intravascular osmotic agent.[63]

HBOT carries a limited number of risks complicating the therapeutic process for patients with NSTI. Middle ear barotrauma occurs in about 2% of awake patients[64] but is avoided in unconscious patients by the use of trans-tympanic ventilation tubes.[65] Rarely, pulmonary barotrauma may occur during decompression in patients with airway obstruction;[64] however, to our knowledge pulmonary barotrauma has not occurred in a ventilated patient, where the airway is likely to remain open. Oxygen has toxic effects with both pulmonary and neurologic manifestations. Pulmonary toxicity requires prolonged exposure to hyperbaric doses and is not a practical problem,[64] while the incidence of oxygen seizures is approximately 0.01% of treatments with no evidence of long-term sequelae.[64,66]

Patients with NSTI are often critically unwell and unstable. Inter-hospital transportation may be inadvisable in some cases, preventing the application of this therapy if HBOT is unavailable at the treating hospital. In-hospital HBOT chambers with ICU-capabilities are essential for the safe delivery of HBOT,[64,67,68] particularly as HBOT may reduce mortality in the most critically ill patients.[38]

There are several limitations to our review. Mortality, co-morbidities, illness severity and co-interventions were all incompletely reported leading to some doubt these patients are directly comparable between studies. Additional important variables include geographical location and the year of reporting. Our results should be applied with caution to any single subset of NSTI.

The absence of randomised trials of HBOT for NSTI has been highlighted. We urge researchers to consider remedying

this. We emphasise such a study needs careful preparation including power calculations based on the data in this review, reliable randomisation with blinding of patient and investigators, uniform approaches to hyperbaric oxygen doses, antibiotic administration, intensive management and surgical approach, rigorous data collection and well-defined outcomes. Such a study cannot be achieved by any single clinical unit and will involve close co-operation across many centres.

Conclusions

Meta-analysis of the non-random comparative data indicates patients with NSTI treated with HBOT have reduced odds of dying during the sentinel event and may be less likely to require a major amputation. Other benefits are uncertain. The most effective dose of oxygen remains unclear in terms of treatment profile, the optimal interval between treatments and the total number of treatments required for the best outcome. A high quality RCT is justified.

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Middle ear barotrauma in diving

Oskari H Lindfors[1], Anne K Räisänen-Sokolowski[2,3], Jari Suvilehto[4], Saku T Sinkkonen[1]

[1] *Department of Otorhinolaryngology – Head and Neck Surgery, Head and Neck Center, Helsinki University Hospital and University of Helsinki, Helsinki, Finland*
[2] *Department of Pathology, Helsinki University Hospital and University of Helsinki, Helsinki, Finland*
[3] *Centre for Military Medicine, Finnish Defence Forces, Helsinki, Finland*
[4] *Mehiläinen Oy, Helsinki, Finland*

Corresponding author: *Oskari H Lindfors, Department of Otorhinolaryngology – Head and Neck Surgery, Head and Neck Center, Helsinki University Hospital, P.O. Box 263, FI-00029 HUH, Helsinki, Finland*
oskari.lindfors@helsinki.fi

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Abstract
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Introduction: Middle ear barotrauma (MEBt) is the most common medical complication in diving, posing a serious risk to dive safety. Given this prevalence and the continuing growth of the diving industry, a comprehensive overview of the condition is warranted.
Methods: This was a survey study. An anonymous, electronic questionnaire was distributed to 7,060 recipients: professional divers of the Finnish Border Guard, the Finnish Rescue Services, and the Finnish Heritage agency; and recreational divers registered as members of the Finnish Divers' Association reachable by e-mail (roughly two-thirds of all members and recreational divers in Finland). Primary outcomes were self-reported prevalence, clinical characteristics, and health effects of MEBt while diving. Secondary outcomes were adjusted odds ratios (OR) for frequency of MEBt with respect to possible risk factors.
Results: A total of 1,881 respondents participated in the study (response rate 27%). In total, 81% of the respondents had experienced MEBt while diving. Of those affected, 38% had used medications and 1% had undergone otorhinolaryngology-related surgical procedures due to MEBt. Factors most associated with MEBt were poor subjective success in Valsalva ('occasionally' versus 'always' successful: OR 11.56; 95% CI 7.24–18.47) and Toynbee ('occasionally' versus 'always' successful: OR 3.51; 95% CI 1.95–6.30) manoeuvres.
Conclusions: MEBt is common in both recreational and professional divers, having affected 81% of the respondents. The main possible risk factors include poor success in pressure equalisation manoeuvres.

Introduction

Middle ear barotrauma (MEBt) while diving is considered to result from inadequate Eustachian tube (ET) function during rapid ambient pressure changes,[1,2] this being generally considered the mildest form of ET dysfunction (ETD).[3] The symptoms typically include pressure sensations or pain in the ears, hearing loss or muffled hearing and sometimes tinnitus.[4–7] Rarely, the condition may manifest as alternobaric vertigo,[8,9] perilymphatic fistula[10] and even less frequently, in the case of a dehiscent cranial nerve VII, as facial baroparesis.[8,11–16] A further, extremely rare complication is a temporal bone fracture[17] (most likely resulting from a dehiscent *tegmen tympani*), leading to subsequent intracranial sequelae.[17] These symptoms are widely considered to be the most prevalent disorder in all diving and subaquatic medicine,[4–7] sometimes seriously compromising dive safety.

Reports on the prevalence vary. While an incidence of 10.1% has been documented in hyperbaric pressure chamber testing of Taiwanese Navy recruits,[18] numbers as high as 23.2% have been reported in military divers in more representative, open-water conditions.[19] Moreover, even higher prevalences (45.0%) have been reported[20] among recreational divers with numbers increasing further (71–72%)[21,22] when considering otoscopic/tympanometric diagnoses instead of symptom reporting alone.

Given the large scale of the diving industry, the high prevalence of MEBt while diving and its potentially hazardous consequences and complications, a thorough examination of the condition is justified. With this in mind, the primary objective of the study was to determine the frequency, clinical characteristics, and the short-term health effects of MEBt while diving. The secondary goal was to examine possible risk factors. The tertiary goal was to examine whether repetitive exposure to barometric stress might lead to an increase in MEBt over the years.

Methods

The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of the Hospital District of Helsinki and Uusimaa (§6164/HUS/2508/2018). The need for informed consent was waived as the study was conducted anonymously.

STUDY DESIGN

Previous literature describing questionnaires on MEBt while diving was reviewed. None of the published questionnaires were directly applicable to the objectives of the study, so a new questionnaire was developed by the research group, utilising previous literature.

The questionnaire consisted of 22–55 questions (depending on answers) designed to examine the respondents' diving and medical histories and frequency of MEBt while diving. Furthermore, the respondents were asked about possible pressure-chamber testing, clinical characteristics and their need for medications and otorhinolaryngology-related (ORL-related) surgical procedures due to MEBt. The English translation of the questionnaire is presented in Appendix 1*.

The questionnaire was electronically sent via e-mail to 7,060 recipients: professional divers of the Finnish Border Guard, the Finnish Rescue Services, and the Finnish Heritage agency; and recreational divers registered as members of the Finnish Divers' Association reachable by email (roughly two-thirds of all members and all recreational divers in Finland). Data acquisition was carried out between November 2018 and September 2019, consisting of the primary email and repeated reminder emails at approximately 1–2 month intervals. Full details of data acquisition are presented in Appendix 2*.

STATISTICAL ANALYSIS

All statistical analyses were performed using SPSS Statistics for Windows, version 25.0, 2017 (IBM Corp, Armonk, NY, USA). A two-tailed *P*-value of < 0.05 was interpreted to indicate statistical significance.

Descriptive statistics are presented as numbers and percentages for categorical variables and as medians and interquartile ranges (IQR) for continuous variables. Categorical data were analysed using Fisher's exact test (two-tailed) or where appropriate, the Chi-Square test. Continuous variables were analysed using the Mann-Whitney U test or the Kruskal-Wallis test as appropriate. The Bonferroni correction was applied to account for multiple comparisons.

Multivariable binary logistic regression analyses were performed to identify factors associated with MEBt while diving. Variables included in the models were sex, number of diving years, age, body mass index (BMI), pollen allergies, smoking, number of upper respiratory tract infections (URTI) per year and subjective success in Valsalva and Toynbee manoeuvres. The results are presented as adjusted odds ratios (OR) with 95% confidence intervals (CI). The frequency of MEBt was dichotomised at two different cut-off points: between "*never*" and at least "*sporadically*" suffering from MEBt during one's life; and between suffering from MEBt only "*sporadically*" and at least "*occasionally*". These two separate cut-off points were chosen to gain a better overall understanding of factors associated with the condition.

Results

OVERVIEW OF THE STUDY SAMPLE AND MIDDLE EAR BAROTRAUMA WHILE DIVING

The survey achieved a final response rate of 26.6% (1,881/7,060). Details of the study sample and the frequency of MEBt while diving are presented in Tables 1 and 2, respectively.

In total, males made up the majority of the study sample, comprising 79.8% of the respondents. A quarter (23.2%) of the respondents were professional divers, the other three quarters (76.8%) recreational ones. Most respondents reported being scuba divers (91.9%), some reporting technical diving (18.3%) and some free diving (15.6%) as their respective diving types. Median (IQR) age was 43 (35–52) years, 44 (36–53) in males and 41 (33–50) in females. Further characteristics of the study sample are presented in Table 1.

Subjective success in Valsalva and Toynbee manoeuvres was also aksed for (see Table 2). A small minority (6.3%) reported succeeding in the Valsalva manoeuvre "*occasionally*" or "*never*", while 51.9% reported "*almost always*" (but not during an URTI) succeeding in the manoeuvre. The final 41.7% reported success "*always*", even during an URTI.

Subjective success in Toynbee manoeuvre was generally poorer among the respondents, compared to success in Valsalva. A total of 47.2% of the respondents reported succeeding "*occasionally*" or "*never*", another 35.8% "*almost always*", the final 17.0% reporting "*always*" succeeding in the manoeuvre.

MEBt while diving had affected 80.7% of the sample. More than half (61.2%) of the respondents reported symptoms "*sporadically*", another 15.5% "*occasionally*" and the final 4.0% "*almost always*" or "*always*" while diving. The proportion of respondents having never experienced symptoms was 20.5% in males and 14.7% in females, while the proportion of those having experienced symptoms at least "*occasionally*" was 17.4% in males and 27.8% in females.

Footnote: * Appendices 1–3 are available on DHM Journal's website: https://www.dhmjournal.com/index.php/journals?id=83

Table 1

Overview of the study sample. Categorical data presented as *n* (%) and continuous data presented as median (IQR). * Data missing in two cases. BET – balloon eustachian tuboplasty; BMI – body mass index; FESS – functional endoscopic sinus surgery; ORL – otorhinolaryngology; RFA – radiofrequency ablation; URTI – upper respiratory tract infection

Variable	All (*n* = 1,881)	Female (*n* = 380)	Male (*n* = 1,501)
Age (years)	43 (35–52)	41 (33–50)	44 (36–53)
Height (cm)	178 (172–183)	167 (163–171)	180 (175–184)
Weight (kg)	83 (74–91)	68 (62–75)	85 (78–94)
BMI ($kg \cdot m^{-2}$)	26 (24–28)	24 (22–27)	26 (25–28)
Diving years	10 (4–17)	6 (3–11)	10 (4–20)
Number of dives*	200 (80–550)	150 (50–400)	200 (100–600)
Diving type			
Professional	436 (23.2%)	30 (7.9%)	406 (27.0%)
Recreational	1,445 (76.8%)	350 (92.1%)	1,095 (73.0%)
Diving mode			
Free diving	293 (15.6%)	53 (13.9%)	240 (16.0%)
Scuba diving	1,728 (91.9%)	359 (94.5%)	1,369 (91.2%)
Technical diving	344 (18.3%)	46 (12.1%)	298 (19.9%)
Smoking			
Never	1,542 (82.0%)	318 (83.7%)	1,224 (81.5%)
Occasionally	242 (12.9%)	37 (9.7%)	205 (13.7%)
Regularly	97 (5.2%)	25 (6.6%)	72 (4.8%)
Allergies			
Any allergy	629 (33.4%)	157 (41.3%)	472 (31.4%)
Pollen	451 (24.0%)	96 (25.3%)	355 (23.7%)
Animal	226 (12.0%)	64 (16.8%)	162 (10.8%)
Food	151 (8.0%)	54 (14.2%)	97 (6.5%)
Other	99 (5.3%)	42 (11.1%)	57 (3.8%)
Surgical procedures (ORL-related)			
Any procedure	696 (37.0%)	127 (33.4%)	569 (37.9%)
Adenoidectomy	492 (26.2%)	93 (24.5%)	399 (26.6%)
Myringotomy	198 (10.5%)	43 (11.3%)	155 (10.3%)
Tympanostomy	93 (4.9%)	19 (5.0%)	74 (4.9%)
BET	14 (0.7%)	4 (1.1%)	10 (0.7%)
Myringoplasty	22 (1.2%)	2 (0.5%)	20 (1.3%)
FESS	84 (4.5%)	12 (3.2%)	72 (4.8%)
Septoplasty	60 (3.2%)	2 (0.5%)	58 (3.9%)
RFA (inf. turbinates)	16 (0.9%)	4 (1.1%)	12 (0.8%)
Cleft palate	2 (0.1%)	0 (0.0%)	2 (0.1%)
URTI per year			
0	313 (16.6%)	52 (13.7%)	261 (17.4%)
1	958 (50.9%)	193 (50.8%)	765 (51.0%)
2	424 (22.5%)	96 (25.3%)	328 (21.9%)
≥ 3	186 (9.9%)	39 (10.3%)	147 (9.8%)

FACTORS ASSOCIATED WITH THE FREQUENCY OF MIDDLE EAR BAROTRAUMA

Factors associated with the frequency of MEBt while diving are presented as odds ratios (OR) and 95% confidence intervals (CI) in Table 3. Poor subjective success in Valsalva and Toynbee manoeuvres were both strongly associated with the frequency of MEBt, while female sex and a high number of URTIs per year were somewhat associated with the condition. Factors such as allergies, smoking, or the

Table 2
Middle ear barotrauma in 1,881divers. Data presented as *n* (%). URTI – upper respiratory tract infection

Variable	All (*n* = 1,881)	Female (*n* = 380)	Male (*n* = 1,501)
Subjective success in Valsalva			
Never/Occasionally	119 (6.3%)	39 (10.3%)	80 (5.3%)
Almost always (not when URTI)	977 (51.9%)	222 (58.4%)	755 (50.3%)
Always	785 (41.7%)	119 (31.3%)	666 (44.4%)
Subjective success in Toynbee			
Never/Occasionally	887 (47.2%)	199 (52.4%)	688 (45.8%)
Almost always (not when URTI)	674 (35.8%)	130 (34.2%)	544 (36.2%)
Always	320 (17.0%)	51 (13.4%)	269 (17.9%)
Pressure equalisation test before dive			
No	648 (34.4%)	166 (43.7%)	482 (32.1%)
Yes	1,233 (65.6%)	214 (56.3%)	1,019 (67.9%)
Middle ear barotrauma in diving			
Never	363 (19.3%)	56 (14.7%)	307 (20.5%)
Sporadically	1,151 (61.2%)	218 (57.4%)	933 (62.2%)
Occasionally	292 (15.5%)	75 (19.7%)	217 (14.5%)
Almost always	64 (3.4%)	26 (6.8%)	38 (2.5%)
Always	11 (0.6%)	5 (1.3%)	6 (0.4%)

number of diving years had no association with frequency of MEBt while diving.

Poor success in Valsalva was the variable most strongly associated with the frequency of MEBt while diving. Respondents who reported succeeding in the Valsalva manoeuvre only "*occasionally*" or "*never*" had an adjusted OR of 4.80 (95% CI 2.44–9.44) for experiencing MEBt at least "*sporadically*" and an OR of 11.56 (95% CI 7.24–18.47) for experiencing such symptoms at least "*occasionally*", compared to respondents who reported "*always*" succeeding in the manoeuvre.

Poor success in Toynbee was also associated with MEBt. Respondents who reported succeeding in the manoeuvre "*occasionally*" or "*never*" had respective ORs of 1.83 (95% CI 1.32–2.53) and 3.51 (95% CI 1.95–6.30) for experiencing MEBt at least "*sporadically*" and "*occasionally*", compared with those who reported "*always*" succeeding in the manoeuvre. Overall, the ORs for experiencing MEBt more often increased as subjective success in Valsalva and Toynbee manoeuvres decreased.

CHARACTERISTICS OF ME BAROTRAUMAS

Characteristics of MEBt and its circumstances are presented in Table 4. The table reports questionnaire results from respondents affected by MEBt (n = 1,518) and is divided into three categories based on the respondents' subjective success in Valsalva (as it was shown to be highly associated with the condition in Table 3).

Half (54.6%) of the respondents had experienced MEBt 1–9 times, the other half either 10–19 times (19.1%) or 20 or more times (26.3%) throughout the years. The number of MEBt events experienced while diving increased as subjective success in Valsalva decreased ($P < 0.001$).

Symptoms predominantly appeared when descending, 94.6% of the respondents reporting symptoms mainly on descent and a minority of 14.3% mainly on ascent. Furthermore, the symptoms most often manifested in relatively shallow waters (where the relative pressure differentials are the largest). In total, 38.4% of respondents reported symptoms at a depth of 0–4 metres' seawater (msw), a further 43.1% at a depth of 5–9 msw and the last 18.4% at a depth ≥ 10 msw.

Symptoms of MEBt varied. Symptoms such as pain (80.0%) and pressure sensations (52.2%) of the ears were by far the most prevalent, others such as hearing loss (5.9%) and ringing in the ears (4.7%) appearing less frequently. Among other symptoms, tympanic membrane perforations had been experienced by 3.0% of the respondents, whereas vertigo and nausea had affected proportions of 10.7% and 1.7%, respectively. Of note, vertigo (34.1% versus 6.5%, $P < 0.001$) and nausea (6.0% versus 1.0%, $P < 0.001$) were more often reported by those who reported symptoms mainly on ascent.

Symptom laterality was also examined. Symptoms were reported in both ears in 43.4% of cases and in one ear in 22.8% of cases. The remaining 33.8% of the respondents were unable to specify the number of affected ears, the proportion of which decreased as subjective success in Valsalva decreased ($P < 0.001$).

Table 3
Multivariate logistic regression analyses of factors associated with middle ear barotrauma while diving. An adjusted OR over 1 indicates an increase in the odds of experiencing MEBt in diving. BMI – body mass index; CI – confidence interval; OR – odds ratio; URTI – upper respiratory tract infection

Variable	Frequency of middle ear barotrauma in diving	
	Never (n = 363) versus Sporadically, Occasionally Almost always, Always (n = 1,518)	Never, Sporadically (n = 1,514) versus Occasionally, Almost always, Always (n = 367)
	OR (95% CI)	**OR (95% CI)**
Age	1.00 (0.98–1.01)	1.01 (1.00–1.02)
Diving years	1.03 (1.01–1.04)	0.99 (0.98–1-01)
BMI	1.00 (0.97–1.04)	0.98 (0.95–1.02)
Sex		
Male	1.00	1.00
Female	1.36 (0.97–1.91)	1.43 (1.07–1.92)
Allergies (pollen)		
No	1.00	1.00
Yes	1.11 (0.82–1.50)	1.30 (0.99–1.71)
Smoking		
Never	1.00	1.00
Occasionally	1.27 (0.86–1.86)	0.93 (0.64–1.36)
Regularly	0.88 (0.51–1.49)	0.91 (0.51–1.60)
URTI per year		
0	1.00	1.00
1	1.08 (0.78–1.48)	0.97 (0.66–1.44)
2	1.34 (0.90–2.00)	1.37 (0.89–2.10)
≥ 3	1.37 (0.81–2.32)	2.11 (1.30–3.42)
Subjective success in Valsalva		
Always	1.00	1.00
Almost always (not when URTI)	4.22 (3.14–5.66)	3.13 (2.25–4.36)
Occasionally/Never	4.80 (2.44–9.44)	11.56 (7.24–18.47)
Subjective success in Toynbee		
Always	1.00	1.00
Almost always (not when URTI)	1.53 (1.08–2.16)	1.98 (1.08–3.63)
Occasionally/Never	1.83 (1.32–2.53)	3.51 (1.95–6.30)

Symptom duration was highly variable. The symptoms typically dissipated in ≤ 2 min in 67.3% of cases, between 2–120 min in 22.7% of cases and between 2 h–2 d in 6.7% of cases. In the remaining 3.2% of cases, the symptoms typically lasted for > 2 d and generally, the duration of symptoms increased as subjective success in Valsalva decreased ($P < 0.001$). Symptoms typically lasting for > 2 h were more often reported by those who reported symptoms mainly on ascent.

Changing vulnerability to MEBt concerned a minority of respondents. The majority, 56.1%, reported no change in either direction, whereas 37.9% reported experiencing less MEBt than previously throughout their diving careers. Conversely, the remaining 5.9% reported currently experiencing more symptoms than previously, and the proportion of such respondents increased as subjective success in Valsalva decreased ($P < 0.001$).

HEALTH EFFECTS OF MIDDLE EAR BAROTRAUMA

Health effects of MEBt are presented in Table 5. The table consists of questionnaire results from affected respondents (n = 1,518) and is divided into three categories based on the subjective success in Valsalva. Medication use in response to symptoms was reported by 37.5% of affected divers. A total of 27.5% reported having used prescribed medications and 22.3% the use of nonprescribed ones. The use of all medications increased as subjective success in Valsalva decreased ($P < 0.001$).

Table 4

Characteristics of middle ear barotrauma while diving and the effect of subjective success in Valsalva manoeuvre. Data are presented as *n* (%) and analysed using Fisher's exact (two-tailed) or Chi-Square tests*. The Bonferroni correction was applied to multiple comparisons. Each subscript letter (a, b, and c) denotes a subset of categories whose column proportions do not differ significantly from each other at the 0.05 level. [xx] data missing in 582 cases. TM – tympanic membrane; URTI – upper respiratory tract infection

Variable	All (*n* = 1,518)	Subjective success in Valsalva manoeuvre			*P*-value
		Always (*n* = 519)	Almost always (not during URTI) (*n* = 890)	Occasionally or never (*n* = 109)	
Symptoms					
1–9 times	829 (54.6%)	357 (68.8%)$_a$	439 (49.3%)$_b$	33 (30.3%)$_c$	< 0.001*
10–19 times	290 (19.1%)	84 (16.2%)	187 (21.0%)	19 (17.4%)	
≥ 20 times	399 (26.3%)	78 (15.0%)$_a$	264 (29.7%)$_b$	57 (52.3%)$_c$	
% of symptomatic times related to URTI[xx]					
100%	355 (37.9%)	123 (46.6%)$_a$	220 (35.4%)$_b$	12 (23.5%)$_b$	0.005
51–99%	137 (14.6%)	31 (11.7%)	96 (15.5%)	10 (19.6%)	
≤ 50%	444 (47.4%)	110 (41.7%)	305 (49.1%)	29 (56.9%)	
Symptoms during dive					
Mainly ascending	217 (14.3%)	67 (12.9%)	139 (15.6%)	11 (10.1%)	0.175
Mainly descending	1,436 (94.6%)	487 (93.8%)	843 (94.7%)	106 (97.2%)	0.396
Symptoms manifested at:					
0–4 msw	583 (38.4%)	190 (36.6%)	355 (39.9%)	38 (34.9%)	0.550*
5–9 msw	655 (43.1%)	227 (43.7%)	375 (42.1%)	53 (48.6%)	
≥ 10 msw	280 (18.4%)	102 (19.7%)	160 (18.0%)	18 (16.5%)	
Symptoms manifested as:					
Ear pressure	793 (52.2%)	254 (48.9%)	483 (54.3%)	56 (51.4%)	0.153
Ear pain	1,215 (80.0%)	384 (74.0%)$_a$	733 (82.4%)$_b$	98 (89.9%)$_b$	< 0.001
Ear ringing	71 (4.7%)	21 (4.0%)	43 (4.8%)	7 (6.4%)	0.483
Hearing loss	90 (5.9%)	27 (5.2%)	56 (6.3%)	7 (6.4%)	0.688
TM perforation	46 (3.0%)	12 (2.3%)	32 (3.6%)	2 (1.8%)	0.361
Vertigo	162 (10.7%)	44 (8.5%)	110 (12.4%)	8 (7.3%)	0.042
Nausea	26 (1.7%)	6 (1.2%)	16 (1.8%)	4 (3.7%)	0.140
Other	31 (2.0%)	12 (2.3%)	15 (1.7%)	4 (3.7%)	0.280
Symptoms manifested in:					
One ear	346 (22.8%)	101 (19.5%)	213 (23.9%)	32 (29.4%)	< 0.001*
Both ears	659 (43.4%)	174 (33.5%)$_a$	428 (48.1%)$_b$	57 (52.3%)$_b$	
Not sure	513 (33.8%)	244 (47.0%)$_a$	249 (28.0%)$_b$	20 (18.3%)$_b$	
Symptoms lasted for:					
≤ 2 min	1,022 (67.3%)	384 (74.0%)$_a$	577 (64.8%)$_b$	61 (56.0%)$_b$	0.001*
≤ 2 hours	345 (22.7%)	93 (17.9%)$_a$	219 (24.6%)$_b$	33 (30.3%)$_b$	
≤ 2 days	102 (6.7%)	27 (5.2%)	67 (7.5%)	8 (7.3%)	
> 2 days	49 (3.2%)	15 (2.9%)	27 (3.0%)	7 (6.4%)	
Symptoms before dive					
Yes	446 (29.4%)	126 (24.3%)$_a$	292 (32.8%)$_b$	28 (25.7%)$_{a,b}$	0.002
No	1,072 (70.6%)	393 (75.7%)$_a$	598 (67.2%)$_b$	81 (74.3%)$_{a,b}$	
Changing vulnerability over the years					
Less	576 (37.9%)	233 (44.9%)$_a$	316 (35.5%)$_b$	27 (24.8%)$_b$	< 0.001
Same	852 (56.1%)	272 (52.4%)$_a$	509 (57.2%)$_{a,b}$	71 (65.1%)$_b$	
More	90 (5.9%)	14 (2.7%)$_a$	65 (7.3%)$_b$	11 (10.1%)$_b$	

Table 5

Health effects of middle ear barotraumas while diving and the effect of subjective success in Valsalva manoeuvre. Medication refers to use of medication as a result of MEBt symptoms. Data are presented as *n* (%) and analysed using Fisher's exact test (two-tailed) (Bonferroni correction applied). Each subscript letter (a and b) denotes a subset of categories whose column proportions do not differ significantly from each other at the 0.05 level. BET – balloon eustachian tuboplasty; URTI – upper respiratory tract infection

Variable	All (*n* = 1,518)	Subjective success in Valsalva manoeuvre			*P*-value
		Always (*n* = 519)	Almost always (not during URTI) (*n* = 890)	Occasionally or never (*n* = 109)	
All medication					
All	570 (37.5%)	138 (26.6%)$_a$	377 (42.4%)$_b$	55 (50.5%)$_b$	< 0.001
All, last 12 months	294 (19.4%)	57 (11.0%)$_a$	214 (24.0%)$_b$	23 (21.1%)$_b$	< 0.001
All, earlier	329 (21.7%)	92 (17.7%)$_a$	201 (22.6%)$_a$	36 (33.0%)$_b$	0.001
Prescribed					
All	417 (27.5%)	94 (18.1%)$_a$	279 (31.3%)$_b$	44 (40.4%)$_b$	< 0.001
Last 12 months	213 (14.0%)	40 (7.7%)$_a$	152 (17.1%)$_b$	21 (19.3%)$_b$	< 0.001
Earlier	231 (15.2%)	61 (11.8%)$_a$	145 (16.3%)$_{a,b}$	25 (22.9%)$_b$	0.005
Non-prescribed					
All	338 (22.3%)	76 (14.6%)$_a$	229 (25.7%)$_b$	33 (30.3%)$_b$	< 0.001
Last 12 months	168 (11.1%)	30 (5.8%)$_a$	127 (14.3%)$_b$	11 (10.1%)$_{a,b}$	< 0.001
Earlier	184 (12.1%)	48 (9.2%)$_a$	113 (12.7%)$_a$	23 (21.1%)$_b$	0.003
Surgical procedures due to symptoms					
All	19 (1.3%)	5 (1.0%)	11 (1.2%)	3 (2.8%)	0.285
Myringotomy	6 (0.4%)	1 (0.2%)	4 (0.4%)	1 (0.9%)	0.387
Tympanostomy	2 (0.1%)	0 (0.0%)	2 (0.2%)	0 (0.0%)	0.599
BET	14 (0.9%)	4 (0.8%)	8 (0.9%)	2 (1.8%)	0.503

Surgical procedures due to the symptoms were reported by 1.3% of affected divers. In total, six respondents (0.4%) had undergone a myringotomy, two (0.1%) a tympanostomy tube insertion and 14 (0.9%) a balloon Eustachian tuboplasty. The proportion of respondents having undergone procedures seemed to increase as subjective success in Valsalva decreased.

Discussion

COMPARISON WITH PREVIOUS RESEARCH

In this study, subjective success in Valsalva and Toynbee manoeuvres were the factors most strongly associated with MEBt while diving. Even though tympanometric measurements before and after the Valsalva manoeuvre (and measurements before and after swallowing, i.e., effectively the Toynbee manoeuvre) have been investigated as predictors of (otoscopic) MEBt,[23,24] no studies exist examining *subjective* success in Valsalva or Toynbee in relation to MEBt symptoms specifically. Additionally, in this study, female sex and a high number of URTIs per year were associated with MEBt (ORs of 1.43 and 2.11 respectively); both have been previously noted as possible risk factors.[9,25] While no connection was detected to smoking or pollen allergies, a connection between allergic rhinitis and alternobaric vertigo has been previously reported in patients with ETD (hypothesised to occur via the Toynbee phenomenon).[26]

MEBt while diving has been experienced by 80.7% of the sample, more or less aligning with previous reports: 23.9–52.1% of recreational divers have reported similar symptoms in other surveys,[27,28] and a prevalence of 36.5–72.0% has been reported on multiday-diving courses,[22,29] and even after a single dive[21] (although these numbers mostly reflect otoscopic barotrauma, not necessarily symptomatic MEBt). As the present study examined the *lifetime* (to date) prevalence of MEBt in diving, the numbers not surprisingly surpass those of the studies spanning shorter time periods.

The findings relating to conditions in which the symptoms arose (diving phase, depth) or how they manifested (symptoms, laterality, duration) have not been previously investigated, making this the first publication to comprehensively present such findings. Moreover, to the best of our knowledge, no studies on the health effects of MEBt while diving have been published.

STRENGTHS AND LIMITATIONS

The external validity of the results is certainly the study's largest limitation. As our study population did not consist of the entire target population (i.e., all recreational and professional divers in Finland) and the study sample was comprised of only 26.6% of the study population, the results can hardly be considered representative of all divers operating in Finland. However, the study is (by far) the

largest survey on ME barotraumas while diving to date, and therefore a valuable contribution to the diving and medical communities.

Regarding internal validity, the data describing the frequency, clinical characteristics, and health effects of MEBt can be considered reliable, whereas the data relating to possible risk factors are vulnerable to several biases, predominantly confounding. To minimise this, multivariate logistic regression analyses were carried out, in which poor success in Valsalva and Toynbee manoeuvres was associated with the frequency of symptoms. These associations are further supported by application of the Bradford Hill guidelines for observational data, presented in Appendix 3*.

The main strengths of the study are its large sample size and level of detail regarding the questions submitted to the respondents: no previous studies have examined MEBt while diving on such a large scale and a detailed level. Moreover, the anonymity of the questionnaire is an additional strength: with no risk of identification, the reason for dishonesty disappears when submitting one's response.

Limitations include the use of diver-reported data with its inherent subjectivity. While this is a limitation, many of the outcomes the study was designed to investigate were in themselves subjective, so such a limitation could not be avoided. Moreover, the health effects of MEBt covered by our questionnaire failed to include their long-term effects, such as any permanent ear and hearing disorders resulting from the repeated MEBt. In addition, there is a possibility that a proportion of the symptoms, here attributed to MEBt, were in fact the result of other pathologies such as inner ear barotrauma or inner ear decompression sickness. Finally, given the ~27% response rate, the possibility of a reporting bias among respondents cannot be excluded.

Given the high prevalence and sometimes hazardous sequelae of MEBt while diving, a reliable method for evaluating a diver's middle ear equalisation is needed. While predictors such as tympanometry,[23,24] the 9-step-test[20,30,31] and mastoid pneumatization volume[20,32] have been studied previously, most of these studies have either had relatively small sample sizes or have mainly focused on otoscopic (instead of symptomatic) barotraumas. Moreover, previous studies examining success in Valsalva or Toynbee manoeuvres have demonstrated poor predictive value, albeit these studies have also investigated the ability to predict otoscopic barotraumas, not symptomatic ones.

The diving community would most benefit from a predictive tool that is quick and easy to perform in a wide range of environments (e.g., on a multi-day boat dive) and which can reliably predict symptomatic MEBt (or its absence) while diving. While future research should focus on discovering such a tool, most MEBt seems to arise in relatively shallow depths and therefore, in some instances, the diver can simply test his/her ET function at the start of the dive, and discontinue if problems in middle ear equalisation arise. The association of MEBt to one's success in pressure equalisation could suggest that practicing these techniques might be an effective way of preventing MEBt while diving.

Conclusions

MEBt is common in both recreational and professional divers affecting 80.7% of a sample of over 1,800 divers, and is most often manifest when descending at relatively shallow depths. MEBt is most strongly associated with poor success in pressure equalisation techniques, but women and those with a high number of URTIs per year also seem to be at an increased risk. More research is needed to establish whether the practicing of pressure equalisation techniques could prevent MEBt while diving. This could offer an effective way to reduce the burden of MEBt to both recreational and professional divers around the world.

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Conflicts of interest and funding

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List of appendices

Appendix 1. English translation of the questionnaire
Appendix 2. Details of data acquisition
Appendix 3. Application of the Bradford Hill guidelines for observational data: middle ear barotraumas while diving and the condition's possible risk factors

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Fatalities involving divers using surface-supplied breathing apparatus in Australia, 1965 to 2019

John Lippmann[1,2]

[1] *Australasian Diving Safety Foundation, Canterbury, Victoria, Australia*
[2] *Department of Public Health and Preventive Medicine, Monash University, Victoria, Australia*

Corresponding author: *Dr John Lippmann, Australasian Diving Safety Foundation, P.O. Box 478 Canterbury, VIC 3126, Australia*
johnl@adsf.org.au

Key words
Carbon monoxide; Chain of events analysis; Diving compressors; Diving deaths; Fitness to dive; Occupational diving

Abstract
(Lippmann J. Fatalities involving divers using surface-supplied breathing apparatus in Australia, 1965 to 2019. Diving and Hyperbaric Medicine. 2021 March 31;51(1):53–62. doi: 10.28920/dhm51.1.53-62. PMID: 33761541.)
Introduction: This study identified characteristics and diving practices of victims of fatal surface supplied breathing apparatus (SSBA) incidents in Australia from 1965–2019 to determine underlying factors and risks associated with these activities, better educate the diving community and prevent such deaths.
Methods: A hand search was made of 'Project Stickybeak' reports from 1965–2000 and SSBA fatality data were compared to the Australasian Diving Safety Foundation fatality database. The National Coronial Information System was searched to identify SSBA diving deaths for 2001–2019. Extracted data were collated and analysed using descriptive statistics and Poisson Regression. A chain of events analysis was used to determine the likely sequence of events.
Results: There were 84 identified SSBA-related deaths during the study period. Most victims were relatively young, healthy males (median age 33 years). At least 50% of victims were undertaking work-related diving, and 37% were recreational diving. Equipment issues, mainly compressor-related, were the main contributor, identified as a predisposing factor in 48% of incidents and as triggers in 24%.
Conclusions: Preventable surface-supplied diving deaths still occur in both occupational and recreational diving, often from poor equipment maintenance and oversight. Incorrect configuration of the SSBA and lack of training remain on-going problems in recreational users. These could be addressed by improved education, and, failing this, regulatory oversight. The increase in health-related incidents in older participants may be controlled to some extent by greater medical oversight, especially in recreational and non-certified occupational divers who should be encouraged to undergo regular diving medical assessments.

Introduction

Surface-supplied breathing apparatus (SSBA) diving involves breathing gas underwater, usually compressed air (unless in deeper commercial diving) supplied from the surface through a long hose to a demand valve. The term 'hookah' is frequently used in Australasia to describe a minimalist version of SSBA. This equipment supplies air from a simple, usually petrol-driven, compressor which feeds a small reservoir and in turn delivers the air along a hose to a demand valve regulator. If the demand valve is not of the 'upstream' type, the system should incorporate a non-return valve to prevent potential injury to the diver from a gas supply failure, such as a hose rupture. It is essential that such compressors are fit-for-purpose, well-maintained, appropriately configured to provide a steady and plentiful supply to the number of divers using it at the target depth. They also need to be positioned securely in a well-ventilated area to prevent overheating, while also ensuring that any exhaust fumes cannot contaminate the breathing gas.

The diver wears a mask (or helmet in commercial diving), fins and thermal protection, weights, and ideally, a buoyancy compensator device (BCD). An emergency gas supply in the form of a bail-out cylinder is highly desirable to enable the diver to reach the surface in the event of an interruption to the breathing gas supply. Commercial divers may also have a communication system to liaise with the surface tender.

In Australia, SSBA is used for a variety of purposes. These may include generally well-trained and experienced commercial divers undertaking underwater work; commercial seafood harvesters such as abalone divers and fish farmers; pearl divers; research divers; recreational divers, often hunting or harvesting seafood; and rank novices participating in an underwater experience. Unlike in the past,[1] commercial diving operations have generally become better organised and are under the oversight of workplace regulators, and subject to relevant Codes of Practice. However, serious incidents involving systemic failures still occur.[2,3]

Unlike scuba, where a certification is supposed to be checked prior to the filling of a cylinder, there are currently no restrictions on who can obtain and use a SSBA for recreational use. In addition, some users convert compressors designed for non-diving purposes or fit their compressors out inappropriately, sometimes with dire consequences.[4]

An earlier summary of SSBA-related deaths in Australia indicated that many deaths were the result of equipment-related issues and lack of training or experience.[5] The aim of this current and more detailed study is to examine all Australian SSBA diving-related deaths recorded on the Australasian Diving Safety Foundation (ADSF) database[4] to determine the likely chain of events, examine trends, and to better educate the diving and medical communities and prevent such deaths.

Methods

Approval for the study was received from the human research ethics committees of the Victorian Department of Justice, the Royal Prince Alfred Hospital, Sydney; the Coroner's Court of Western Australia; and the Queensland Office of the State Coroner.

SEARCH

This was a complete case series of known SSBA diving-related fatalities that occurred in Australian waters from 1965 to 2019, inclusive. A hand search was made of the relevant 'Project Stickybeak' reports published in the diving medical literature.[5–7] The data obtained were compared to that recorded on the Australasian Diving Safety Foundation (ADSF) fatality database[4] and adjustments made where necessary. In addition, a comprehensive key word search was made of the National Coronial Information System (NCIS)[8] to identify SSBA diving deaths reported to various State Coronial Services for the years 2001–2019, inclusive. Key words included compressed air, compressed gas, or surface supply, or hookah and diving. Cases identified were matched with cases collected by the investigator via the media or the diving community to minimise the risk of over- or under-reporting.

The review procedure involved review of the published case reports and database entries for cases from 1965 to 2000, to investigate any discrepancies and enter relevant data in a specially designed Excel spreadsheet. The coronial data were also reviewed for cases from 2001 to 2019 and relevant data were also entered into the spreadsheet.

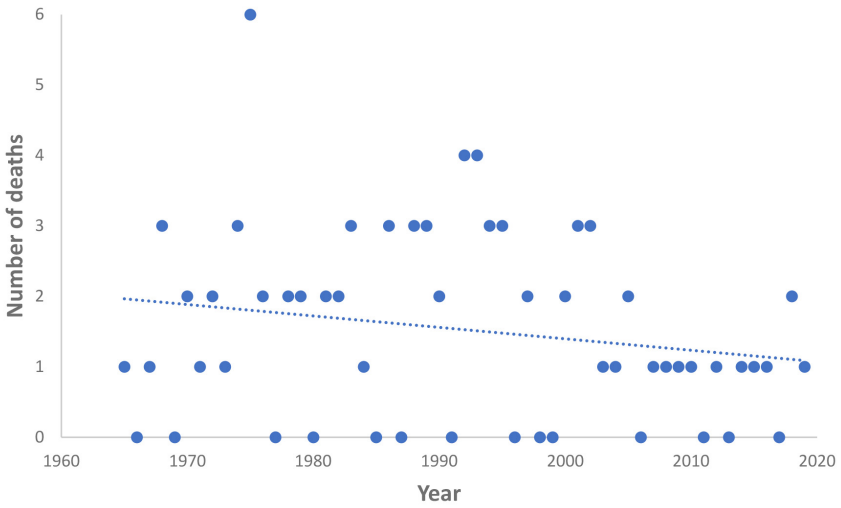
PREDICTORS

A range of potential predictors was extracted, including diver demographics, certifications and experience; dive location; buddy and supervision circumstances; dive purpose and depth; equipment used and any associated problems; incident description.

CHAIN OF EVENTS ANALYSIS

A chain of events analysis (CEA) was conducted to identify predisposing factors and outcome measures including trigger, disabling agent, disabling condition, and cause of death. The CEA was based on the criteria and templates for scuba fatalities previously published.[9] The investigator applied the published templates (with the term 'Disabling Condition' replacing 'Disabling Injury') to these data and obtained the results reported below. An example of the application of such a template is: A faulty compressor (predisposing factor) stalls and interrupts the diver's air supply (trigger), causing the diver to make an emergency

Figure 1
Australian surface-supplied breathing apparatus-related diving fatalities by year, 1965 to 2019 (n = 84)



ascent (disabling agent). He suffers a cerebral arterial gas embolism (disabling condition), becomes unconscious and subsequently drowns (cause of death).

STATISTICAL ANALYSIS

Descriptive analyses based on means and standard deviations or medians and ranges as appropriate was conducted using SPSS Version 25 (IBM® Armonk, NY; 2017). Poisson regression was used to analyse possible trends. All models were fitted using the Stata 16 software (StataCorp 2019).

Results

There were 84 recorded fatalities in divers using SSBA during the 54-year study period.

ANNUAL COUNTS

The median (IQR) number of annual deaths was 1 (0.5, 2) with a maximum of six. The annual deaths generally reduced until 2002 and then remained relatively steady, although the trend was not significant ($P = 0.064$) (Figure 1).

DEMOGRAPHICS

The median (IQR) age of the victims over the study period was 33 (26, 40) years; range 16–72 years). There was a small albeit statistically significant ($P < 0.001$) rise in the age of victims over the period of approximately 3.1 years per decade. The median (IQR) age of victims over the final 20 years was 36.0 (28, 47) years. All but one of the 84 victims were male. Non-occupational divers were older than occupational divers (with means 38 and 32 years, respectively), although this difference was not significant ($P = 0.053$).

LOCATION

The distribution of deaths between the various states and territories was: Victoria (16), Western Australia (17), Tasmania (15), Queensland (15), South Australia (12), New South Wales (8), and Northern Territory (2).

TRAINING AND EXPERIENCE

In 36 of the 84 cases the victims were reported to have undergone some form of training, although the level of training was unspecified in 26 of these. At least seven victims were scuba certified, and at least three had undergone commercial diver training. Twenty-one victims were untrained, and the level of training was unreported in 24 cases. Significantly more of the occupational divers had undergone some type of training ($P = 0.03$).

Although all but 11 of the reports included a description of the diver's experience, or lack thereof, there was no detailed quantification of this experience, so the following summary is somewhat arbitrary. Fifty-two of the victims were described as experienced, 11 had some experience (in some cases very little), and 10 had no experience at all.

Table 1
Diving activity during the fatal incident in 84 SSBA divers. *Construction, oil rig, maintenance, salvage

Activity	Deaths
Occupational	
Commercial*	16
Abalone collecting	10
Pearling	9
Aquarium fish collecting	3
Sea cucumber collecting	2
Crayfish collecting	1
Scallop collecting	1
Research	1
Recreational	
Crayfish collecting	14
Sightseeing	6
Scallop collecting	5
Spearfishing	4
Abalone collecting	1
Self-training	1
Miscellaneous	
Other	4
Unknown	6

ACTIVITY

Forty-three of the 84 victims were undertaking work-related diving, and 31 were diving for recreation, whether harvesting seafood or sightseeing. One untrained and inexperienced victim was trying out the second SSBA demand valve while his friend was underwater. One victim was involved in an underwater survey for his studies (Table 1).

BUDDY/SUPERVISION CIRCUMSTANCES

Solo diving was three times more prevalent in occupational divers than in others. In 64 of the 84 incidents, the victim had either set out alone, or had already separated from his buddy before the fatal incident had occurred. However, in at least 47 of these incidents there was an observer present. Overall, a surface observer was present in 58 cases, and there was no observer in 16 cases. The presence or absence of surface support was unknown in 10 cases.

DEPTH

The dive depth was not reported in eight cases. For the remaining 76 incidents, the median (IQR) dive depth was 11.25 (7.5, 6.75) metres' seawater (msw), range 3–74 msw. Only nine fatal dives were reported to have been deeper than 30 msw. Seven of the 42 occupational divers and only one of the others were known to have been diving deeper than 30 msw. The depth of the incidents shown in Table 2.

Table 2
Depth of fatal incident in 84 SSBA divers. NR = not reported.

Incident depth (msw)	*n* (%)
Surface	15 (18)
Ascent	17 (20)
0–10	22 (26)
11–20	12 (14)
21–30	4 (5)
> 30	4 (5)
NR	10 (12)

WEIGHTS

One victim was known not to have worn weights for his dive, and the weighting circumstances were unreported in 16 incidents. Of the 67 victims who were known to have been wearing weights, 61 were still wearing these weights when rescued or recovered.

BUOYANCY COMPENSATOR DEVICES (BCDS)

There was no indication whether a BCD was worn in 23 of the incidents. However, at least 56 of victims were not wearing a BCD. Five victims were reported to have been wearing a BCD and none of these were inflated when found. One was found to have been faulty.

Chain of events analysis

PREDISPOSING FACTORS

One hundred and seventeen predisposing factors (PFs) were identified as possible or likely contributors to the 84 deaths (Table 3). There were insufficient data to suggest any PFs in 18 cases, and no obvious PF in one incident, despite detailed information. Multiple factors were identified in 42 incidents: 22 incidents with one identified PF, 31 incidents with two, and 11 incidents with three PFs. The main PFs identified were related to equipment, planning, and health issues.

Absence of appropriate equipment or use of defective equipment

Defects, sometimes multiple, were found in a variety of equipment, mainly compressors which were poorly maintained and/or defective (19). Defects included poor hose connections, inappropriate hoses which were overly long, kinked and/or melted, and the absence of intake hoses. Defective demand valves (2), or full-face mask (1) were also implicated. At least two deaths resulted from the compressors' inability to supply sufficient air to multiple divers at their operating depth. Loss of unsecured demand valves (3), obvious overweighting (2) and a very tight wetsuit were also implicated. Of note, the reports rarely mentioned the carrying of a bail-out gas cylinder which would generally be considered as appropriate equipment for such diving.

Planning

Poor planning decisions were often made, usually immediately before the dive. The most common of these was inappropriate placement of the compressors, which was a factor in 13 deaths, mainly due to exhaust gas entering the air intake. Failure to properly assess and plan for environmental dangers contributed to six deaths. These dangers included strong water flows in water supply containment areas, toxic gas in an enclosed space, an active cleaner pump, an engaged boat propellor and the increased risk of local large shark activity. Three divers, who died from decompression sickness (DCS) had not followed any decompression guidance. Two divers were diving solo without surface oversight (one at night), and another two had failed to ensure their working platform was properly secured.

Health

Health-related factors contributed to at least 15 deaths, including eight of 61 deaths occurring before the year 2000, and at least seven of the 23 subsequent deaths. Half of these deaths were in occupational divers. The most common health condition was pre-existing, albeit apparently undiagnosed, ischaemic heart disease (IHD). Pulmonary blebs, pleural scarring after pleurisy, and asthma were implicated in three deaths (one also involving IHD), seizures in three (two associated with congenital brain abnormalities and one idiopathic) and severe obesity in one. Alcohol and heroin appeared contributory to two incidents. There were insufficient data in most cases to determine whether the victims had undergone a recent diving medical examination.

Training, skills, experience

There were at least 13 cases where lack of any or sufficient training, inexperience, and associated lack of skills likely contributed to the outcome. Although six of the victims had scuba certification, in four it was recent, and they had little experience. One very experienced scuba diver died on his first SSBA dive though lack of familiarity with the equipment. Two untrained but experienced SSBA divers were unable to read decompression tables and died from DCS after omitting large decompression obligations. One untrained and inexperienced victim drowned during his first use of SSBA while his friend hunted fish below.

Supervision

Poor supervision likely contributed to at least 14 incidents. Eight of these involved inadequate oversights of the compressor and resulted in contamination of the breathing gas by compressor or boat exhaust, a burning air intake hose, or overheating. In two cases, the surface tender failed

Table 3
Predisposing factors associated with 65 of 84 SSBA fatalities. Some deaths involved multiple predisposing factors, hence the number predisposing factors exceeds the number of cases

Predisposing factor	Subgroup	*n*
Absence of appropriate equipment or use of faulty equipment		27 (32%)
	Defects in equipment	19
	No demand valve security	3
	Insufficient supply capability	2
	Overweighting	2
	Tight wetsuit	1
Planning		28 (33%)
	Poor compressor setup	13
	Environmental danger	6
	Absent decompression planning	3
	Unsupervised solo diving	2
	Unsecured dive platform	2
	Other	2
Health		15 (18%)
	Significant medical history	13
	Alcohol and drug intake	2
Training/experience/skills		13 (15%)
	Scuba trained, novice SSBA	6
	Inexperience-related anxiety/panic	4
	Untrained – no understanding of decompression tables	2
	Untrained, no experience	1
Supervision		14 (17%)
	Compressor oversight failure	8
	Inexperienced working diver	3
	Hose endangerment	3
Activity		9 (11%)
	Seafood collection	6
	Deep commercial dives	3
Organisational		8 (9%)
	Lack of appropriate procedures or equipment	4
	Poor equipment maintenance	4
Poor communication/ co-ordination		3 (4%)
	Failure to communicate site dangers	3

to act promptly on compressor failure and consequent loss of air supply. Another two cases of loss of supply involved the diver's hose being cut by the propeller or entangled around the stern. Both problems were not noticed by the lookouts until too late. There were two incidents involving poor oversight of inexperienced divers in commercial operations, one where a surface tender failed to confirm that the propellor was disengaged when a diver entered the water nearby, and another where a commercial operator failed to have an effective rescue plan, which lead to a delayed, and ultimately unsuccessful, rescue.

Activity

Six of these incidents involved divers who were collecting or harvesting sea life in areas likely to be frequented by large sharks due to whale and seal activity. Two involved deep commercial dives on an oil rig, and one incident occurred during a deep commercial dive in a cold dam with zero visibility and snagging hazards.

Organisational

Poor or absent protocols or procedures for managing commercial diving activities contributed to four incidents. These included inadequate induction procedures and oversight of new divers in commercial operations (seafood/ pearling). Poor organisational equipment maintenance protocols contributed to four deaths, and a lack of appropriate diver rescue procedures to another. A further death would have been prevented by the fitting of a propeller guard on a diving work barge.

Table 4
Triggers identified in 63 of 84 SSBA fatalities. Some deaths involved multiple triggers

Trigger	Subgroup	*n*
Equipment		20 (24%)
	Compressor problem	9
	Hose disconnection	4
	Air hose entanglement	3
	Separation of unsecured mouthpiece	3
	Loss of fin	1
Environmental		20 (24%)
	Marine creature	7
	Entrapment	4
	Immersion effects	3
	Conditions	3
	Boat	3
Gas		14 (17%)
	Contamination	10
	Interrupted/reduced supply	3
	Incorrect gas	1
Exertion associated with…		8 (10%)
	Pre-existing ischaemic heart disease	6
	Epilepsy	1
	Severe asthma and overweighting	1
Diver error		4 (5%)
Anxiety		1 (1%)

Communication

Failure to communicate to the divers the presence and potential site-associated dangers of fast-flowing dam or pipeline waters was instrumental in three deaths.

TRIGGERS

No triggers were identified in 21 incidents due to lack of sufficient information. Sixty-seven possible or likely triggers were identified in the 63 remaining incidents, the main ones related to equipment and environmental factors (Table 4).

Equipment

The main equipment-related triggers arose from problems with compressors, including overheating, valve failures, a blown gasket and the falling over of a poorly secured compressor. Other problems were hose separation due to poor connectors and hose entanglement with work equipment or platforms. Mouthpiece separation from the demand valve due to the absence of securing ties featured in three incidents. All of these affected the supply of breathing gas to the diver.

Environmental

Seven of the fatal incidents were triggered by the presence of dangerous marine creatures, six of these large sharks and the other a crocodile. Another two events were triggered by moving boats, one of which was the diver's working platform. The other diver was hit by a passing speedboat, despite the display of a 'Diver Below' flag. The incidents involving entrapment included three divers entangled in kelp, and one was trapped when his BCD strap was sucked into a hull scrubber. Three incidents involved adverse water conditions, including current and high water flows. Three deaths in divers with pre-existing IHD are thought to have been triggered by the circulatory effects of immersion.

Gas supply

Gas supply-related triggers were largely due to contamination of the air supply due to poorly maintained, functioning, or positioned compressors. One of the incidents triggered by interruption or reduction of the gas supply was due to a kinked hose. Another occurred when the (inexperienced) diver's air supply was interrupted while surface cylinders were changed, and he failed to transfer to his bail-out bottle. Another occurred when the surface team mistakenly changed the supply from an air cylinder to an oxygen cylinder.

Exertion

Exertion unrelated to sea conditions, was implicated as a trigger in nine incidents. Most of these involved performing heavy work underwater. Six of the victims had significant IHD, one a history of seizures, and a severe asthmatic was very over-weighted and working in adverse conditions.

Figure 2
Disabling agents associated with 72 of 84 SSBA fatalities. PBT/CAGE – pulmonary barotrauma/arterial gas embolism; IHD – ischaemic heart disease; DCS – decompression sickness; SAH – subarachnoid haemorrhage

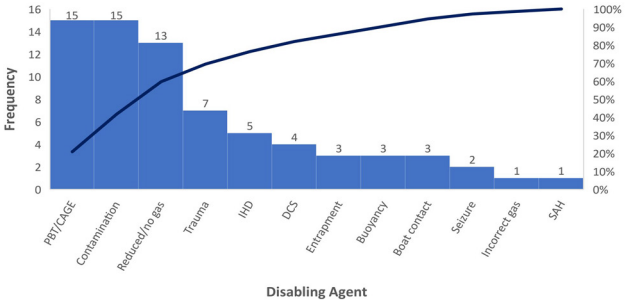
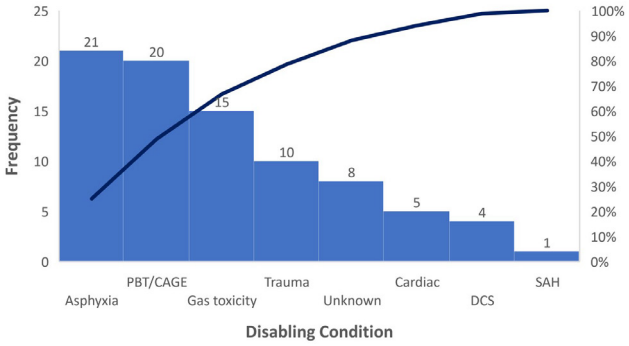


Figure 3
Disabling conditions associated with 84 SSBA fatalities. PBT/CAGE – pulmonary barotrauma/arterial gas embolism; DCS – decompression sickness; SAH – subarachnoid haemorrhage



Primary diver error

Three cases involved failure to adequately plan and follow any decompression guidance and all resulted in fatal DCS. The other involved the diver failing to disengage his boat's engine before entering the water.

Anxiety

Although anxiety and consequent panic are often associated with diving incidents, there was only one case where it was specifically mentioned in the reports. This very inexperienced diver aborted an earlier dive due to anxiety and, after calming down somewhat, attempted another dive where he was seen to suddenly ascend rapidly to the surface. When checked, the equipment was found to be working correctly.

DISABLING AGENTS

Likely disabling agents (DA) were identified in 72 of the incidents. These were related to the gas supply (28), ascent (19), medical conditions (9), environmental circumstances (13), and buoyancy (3). No DAs were identified in 12 cases due to insufficient information (Figure 2).

DISABLING CONDITION

The predominant disabling conditions were asphyxia, CAGE with or without evidence of PBT, and gas toxicities (Figure 3). Of note, the victims with cardiac disabling conditions were considerably older (median 43 y).

Discussion

SSBA diving-related fatalities have reduced over time with a current frequency of around one per year. The victims were predominantly relatively young, healthy males who were experienced SSBA divers. One half of the victims were diving for work and more than one third for recreation, often harvesting seafood. Equipment faults and poor planning predisposed to many of the fatal incidents.

DEMOGRAPHICS

Although the age of SSBA victims increased over the study period, with the median age increasing to 36 years beyond the year 2000, these victims were considerably younger than those of scuba (median 45 y) and snorkelling (median 51 y) victims post-2000.[10,11] Of interest, certification records from the Australian Diver Accreditation Scheme (ADAS), the certification agency for occupational divers in Australia since 2003, show a median age of 45 years for currently certified ('active') SSBA divers. (A Sordes, personal communication, 15 July 2020). This may indicate that the younger cohort of SSBA divers are more at risk of an accident. ADAS records also reveal that 99% percent of certificants are male, consistent with the proportion of male victims.

EQUIPMENT PROBLEMS

Equipment-related issues were the major contributor to these fatalities. Absence of, or faulty equipment predisposed to one third of the deaths. Equipment problems were also identified as the triggers in one-quarter of cases. The main source of problems were compressors which were often poorly maintained, poorly positioned, or inappropriately configured. This led to interruption of the breathing gas supply or breathing gas contamination by carbon monoxide (CO), among other contaminants. Adequate separation of the engine exhaust from the compressor air intake is important to reduce the risk of contamination.[12]

The compressor must be placed in a secure and well-ventilated position to prevent overheating or toppling. Although electric compressors are available, most of the SSBA compressors are driven by a petrol or diesel motor, so it is essential that exhaust fumes are prevented from entering the air intake, which should be positioned in an elevated position upwind of the compressor. The compressor

and intake should be constantly observed throughout use for correct function and the effect of any wind changes. Appropriate filters to remove dust, water and odour must be fitted and replaced when needed. Additional sensors/converters to detect or remove contaminants such as CO can be fitted but are relatively uncommon. Correct lubricants must be used to ensure smooth operation and avoid gas contamination from lubricant breakdown. Regular checking and maintenance are essential to maximise the likelihood of smooth and safe operation. It is also important to ensure, in advance, that a compressor can provide a steady and plentiful supply of air to the number of divers who will use it, at the target depth, and their likely level of exertion.

It is also important to use and maintain appropriate non-kink hoses and secure fasteners for hoses and demand valve mouthpieces. The demand valve should be adequately secured so it is not inadvertently pulled from the diver's mouth. BCDs were seldom used but may have saved several lives had they been available and inflated, especially with victims who were over-weighted or had failed to ditch their weights when needed.

The number of victims who failed to ditch their weight belt is alarming and reflective of the high incidence in scuba diving victims.[10] However, with SSBA divers, it may also be associated with securing the belt and air hose together, a practice that should be discouraged. Some victims wore multiple weight belts, and several belts were not fitted with quick-release buckles. Both factors would have made it difficult to ditch weights when needed. The carrying of a bail-out bottle, rarely evident in this series, is a very important safety measure that needs to be strongly encouraged.

TRAINING, EXPERIENCE AND SKILLS

Inadequate training, experience and skills were identified as contributors to at least 16% of the deaths. Traditionally, if SSBA divers had any formal training, they were either trained as commercial divers to work in the offshore industry or trained to use scuba. Less than half of the victims were known to have undergone any formal related training and, in many cases, the training was not sufficient to address the particular equipment and skills required to safely perform SSBA diving.

Over more recent times, a variety of courses have been available through the Australian Nationally Recognised Training System and taught by registered training organisations. These range from introductory programs offered by recreational scuba diver training agencies, to comprehensive commercial diving courses certified by ADAS. However, despite the increased availability of pertinent training, some divers still take untrained friends or workmates on SSBA dives, occasionally with serious consequences.[13] In addition, some training programmes need ongoing monitoring and improvement to better equip divers for the tasks they need to perform and problems they might encounter.[2]

Experience is valuable in reducing diving risks, but it can sometimes cause complacency. Many of the more than 60 percent of victims who were described as experienced, were not heedful enough of the risks associated with poor planning, equipment maintenance and configuration.

ORGANISATIONAL, PLANNING AND ACTIVITY RISK MANAGEMENT

The fact that half of the victims were undertaking work-related diving is a cause for concern and reflection on the need for safe working practices to be adopted, adhered to, and continually monitored. Many of the victims were self-employed and dived solo, often with an observer on the boat or another nearby platform. Good planning and vigilant surface supervision are essential for safe SSBA diving in such circumstances. Some industries, such as the Tasmanian abalone industry, have created codes of practice to try to improve standards and practices, although this is far from universal.

Poor organisational procedures and practices predisposed to several preventable deaths. Improved induction procedures and oversight of new divers, more rigorous maintenance protocols, and careful planning would have reduced the likelihood of problems in these occupational settings. Relevant Australian standards and regulations pertaining to occupational diving exist and workplace regulators have an important role to play in monitoring compliance.[14,15]

The incidents in the recreational setting were mainly associated with seafood harvesting by relatively inexperienced divers. They often involved the victim becoming overwhelmed by the environment or circumstances while distracted by the 'thrill of the chase'.

Some of the activities undertaken involved increased risk, such as working at substantial depths, near very fast-flowing water, and harvesting seafood in areas and at times where large sharks are likely to be present. Such activities require heightened planning and vigilance, and back-up in the event of a problem.

BUDDY/SUPERVISION CIRCUMSTANCES

As with scuba fatalities,[10] a high proportion of SSBA victims, in this case three-quarters, had either set out alone or had already separated from their buddy before the incident. Unlike many recreational scuba situations where the diver was solo or separated, there was often a surface observer present, theoretically increasing the opportunity for rescue. However, in many of these cases, the observer missed opportunities to prevent or reduce the severity of the

incident by inadequate supervision of the compressor, air hoses or environmental hazards and failing to identify and act on a problem. Although working without visual contact is common in some occupational settings, the combination of communications with an in-water or a surface stand-by diver is invaluable in preventing or managing life-threatening incidents.

PRE-EXISTING MEDICAL CONDITIONS

Over the entire study period, health-related factors, predominantly undiagnosed IHD, contributed to one fifth of the incidents and were identified as disabling agents in more than ten percent. However, the increased involvement of health factors in later years is consistent with more recent reports for scuba diving and snorkelling victims where health issues were contributory to over 40% of deaths.[10,11] This increasing incidence likely reflects an older cohort of participants with the associated higher prevalence of medical conditions. In the recreational setting, all divers and prospective divers aged 45 years or more are advised to have a diving medical examination, irrespective if they have any known health conditions.[16] Among other things, such an examination would assess cardiovascular health and might prevent some cardiac-related diving deaths. In addition to immersion *per se*, diving involves a variety of other cardiac stressors, such as the exertion often associated with underwater work and seafood harvesting, whether recreational or occupational.

In Australia, occupational divers are supposed to have an initial, and subsequent annual, fitness-to-dive examination by a doctor with relevant training.[15] ADAS requires evidence of this for certification and recertification. Such examinations are designed to determine potentially problematic health conditions so that they can be addressed to reduce perceived associated risk. However, recreational SSBA divers are not subject to this requirement, and not all occupational operators comply or monitor compliance, especially in small owner-operator businesses where certification may not be sought.

DISABLING CONDITIONS

When comparing the disabling conditions in these SSBA deaths to those in a cohort of scuba deaths in Australia[17], several differences become apparent. These include lower proportions of deaths from primary asphyxia (25% vs. 37%) and cardiac factors (6% vs. 25%), and higher proportions from PBT/CAGE (24% vs. 15%) and gas toxicity (18% vs. 0%).

The lower prevalence of cardiac-related deaths likely reflects the comparatively younger age of the SSBA cohort and, in some cases possibly a higher level of fitness and the requirement for regular dive medicals or monitoring in the occupational sector. The greater prevalence of PBT/CAGE is very likely a direct consequence of the higher incidence of problems with cessation or reduction in breathing gas, a relatively common occurrence in this series. Severe gas contamination is, fortunately, a relatively uncommon occurrence in the scuba arena due to more rigorous protocols around the filling of scuba cylinders, especially in a commercial recreational setting. However, as evidenced in this report, it remains a serious concern with SSBA. Finally, the lower proportion of deaths from primary asphyxia is likely a direct result of the other higher attributions. There were additional cases where drowning was determined to be the cause of death, but this was secondary to unconsciousness from CO toxicity or PBT/CAGE.

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. Many incidents were not directly witnessed, so assertions in the reports are sometimes speculative. Important information was not available in some cases, especially in the earlier reporting years, which rendered COE data incomplete, thus limiting the conclusions that can be drawn. Even with the use of a template, classification of cases into a sequence of five events in the COE 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

SSBA diving-related fatalities have reduced over time. This is likely because of improvements in education, training, equipment, and regulation, predominantly in the occupational sector. However, the recreational sector remains problematic with little or no oversight of who can use such equipment, and generally poor education and training in its use.

Preventable fatalities still occur in both sectors, often because of poor equipment maintenance and oversight. Incorrect configuration of the SSBA unit and lack of training remain on-going problems in recreational users, and could be addressed by improved education, and, failing this, regulatory oversight.

The increase in health-related incidents in older participants may be controlled to some extent by greater medical oversight, especially in recreational and non-certified occupational divers who should be encouraged to undergo regular diving medical assessments. Commercial organisations should periodically assess their protocols and

practices to identify and address potential shortcomings regarding safety.

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Does persistent (patent) foramen ovale closure reduce the risk of recurrent decompression sickness in scuba divers?

Björn Edvinsson[1], Ulf Thilén[1], Niels Erik Nielsen[2], Christina Christersson[3], Mikael Dellborg[4], Peter Eriksson[4], Joanna Hlebowicz[1]

[1] *Department of Cardiology, Department of Clinical Science, Skåne University Hospital, Lund University, Lund, Sweden*
[2] *Department of Cardiology, Department of Clinical Science, University Hospital Linköping, Linköping University, Linköping, Sweden*
[3] *Department of Medical Sciences, Cardiology Uppsala University, Uppsala, Sweden*
[4] *Department of Cardiology, Department of Clinical Science, Sahlgrenska University Hospital, University of Gothenburg, Gothenburg, Sweden*

Corresponding author: *Dr Joanna Hlebowicz, Department of Cardiology, Skåne University Hospital, Lund University, SE-221, 85 Lund, Sweden*
joanna.hlebowicz@med.lu.se

Key words
Decompression illness; Right-to-left shunt; Risk; Scuba diving; Trimix;Venous gas embolism

Abstract
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Introduction: Interatrial communication is associated with an increased risk of decompression sickness (DCS) in scuba diving. It has been proposed that there would be a decreased risk of DCS after closure of the interatrial communication, i.e., persistent (patent) foramen ovale (PFO). However, the clinical evidence supporting this is limited.
Methods: Medical records were reviewed to identify Swedish scuba divers with a history of DCS and catheter closure of an interatrial communication. Thereafter, phone interviews were conducted with questions regarding diving and DCS. All Swedish divers who had had catheter-based PFO-closure because of DCS were followed up, assessing post-closure diving habits and recurrent DCS.
Results: Nine divers, all with a PFO, were included. Eight were diving post-closure. These divers had performed 6,835 dives (median 410, range 140–2,200) before closure, and 4,708 dives (median 413, range 11–2,000) after closure. Seven cases with mild and 10 with serious DCS symptoms were reported before the PFO closure. One diver with a small residual shunt suffered serious DCS post-closure; however, that dive was performed with a provocative diving profile.
Conclusion: Divers with PFO and DCS continue to dive after PFO closure and this seems to be fairly safe. Our study suggests a conservative diving profile when there is a residual shunt after PFO closure, to prevent recurrent DCS events.

Introduction

Divers use different mixtures of breathing gas depending on the depth and duration of the activity. The most common are: air, with approximately 78% nitrogen and 21% oxygen; nitrox which is oxygen-enriched air; and trimix, which is a mix of oxygen, nitrogen and helium (gas mixtures containing helium are used at greater depths). Nitrogen and helium are inert gases not involved in physiological processes, and when breathing compressed gas underwater, the inert gas dissolves at higher partial pressure in the tissues and blood vessels.[1] With reduction in ambient pressure gas can come out of solution causing bubble formation in the blood and extravascular tissues, and this can result in decompression sickness (DCS).[2,3] The underlying causes of DCS symptoms are principally local effects and pressure exerted by the bubbles, manifested for instance as cutaneous itching, marbled skin and joint pain, complex biochemical reactions in the brain and the spinal cord affecting neurological function.[2–5] The optimal treatment for DCS is 100% oxygen therapy as soon as possible, in combination with intravenous fluids and hyperbaric oxygen treatment (HBOT) in a recompression chamber.[6]

By making stops at certain depths determined by a decompression table or dive computer during the ascent from great depths or after a long dive duration, divers try to minimise the risk of DCS. However, even when diving in line with recommendations for safe diving profiles with decompression stops, studies have shown bubble formation in the venous circulation.[7,8] Normally, these bubbles are filtered and exhaled by the lungs without causing DCS.[3] Both atrial septal defects[9] and persistent (patent) foramen ovale (PFO)[1,10–12] have been associated with an increased risk of DCS due to a right-to-left shunt of venous decompression bubbles into the arterial circulation. Moreover, it has been suggested that divers with PFO are more likely to suffer severe neurological forms of DCS and require longer

treatment with hyperbaric oxygen therapy (HBOT).[12] The Valsalva manoeuvre used to equalise the middle ear pressure, and resistance lifting of heavy diving equipment, have been proposed to cause an increased pressure in the right atrium, which can facilitate the shunting of bubbles.[13]

There are a limited number of studies on diving habits and DCS incidence following PFO closure,[14-16] and such a study had not been performed in Sweden previously. The main aims were to investigate whether patients who had suffered DCS events that led to catheter-based closure of a PFO continued to dive after the closure and if there were any DCS events after the closure.

Methods

The study was approved by the local ethical committee (Dnr 2017/572). All participants were informed about the study in writing before being interviewed, and signed a written informed consent form. The study was registered at clinicaltrials.gov, NCT03997084.

All five centres performing PFO closure in Sweden were asked to participate in the study, however one of them was not able to provide data. All patients who had had a catheter-based closure of PFO or an atrial septal defect (ASD) following DCS at these four centres were identified in SWEDCON, a national registry on congenital heart disease also covering catheter interventions including PFO. In accordance with international consensus,[17] DCS symptoms considered 'mild' were musculoskeletal pain, patchy non-dermatomal paraesthesiae, rash, lymphatic swelling, and constitutional symptoms such as fatigue. 'Serious' symptoms were objective neurological deficits and cardiopulmonary symptoms. Based on information in medical charts, patients who had had the closure because of DCS were selected according to the following criteria: at least one DCS event that led to investigation for a PFO or ASD; PFO or ASD verified with contrast echocardiography; and a completed catheter-based intervention to close the PFO or ASD with the indication being DCS prevention.

A letter with information on the purpose of the study and a consent form was sent to the potential subjects. Subsequently, a phone interview was conducted. The interview was based on a questionnaire with eight main questions focusing on the dive habits and DCS event(s):

- Type of diving certificate: for recreational diving or for professional diving;
- The total number of dives before closure separated into the breathing gas used: compressed air, nitrox or trimix;
- The date of the DCS event/events prior to the closure, together with additional facts regarding each event: breathing gas used, depth, if oxygen therapy was used, if recompression therapy was used, remaining symptoms after the therapy, and symptoms of DCS. The symptoms were divided into mild and serious as described above.
- The date and location of catheter-based closure.
- The total number of dives after closure categorised by the breathing gas used, and the maximum dive depth post-closure;
- The date of DCS events after closure, together with the additional facts stated in question 3;
- The number of dives performed during the last year.
- Optional comments from the participants regarding their diving history.

Data about the treatment and DCS symptoms were collected at each centre in medical records. All DCS cases were diagnosed medically. None of the subjects had any evidence of barotrauma. Information about the result of the closure together with data about the dives that lead to DCS events, including breathing gas and diving depth were compiled. The SWEDCON registry and medical records provided information about the date of closure, the patient's height and weight at the time of intervention, the size of the defect measured with a sizing balloon, the type of closure device, complications post-intervention and the result of the closure. The result was measured by the number of agitated NaCl contrast bubbles found in the left heart, when provoked with Valsalva manoeuvre that was visible on echocardiography 24 hours and one year after the closure. Results were categorized as either no detected bubbles, 1–10 bubbles, more than 10 bubbles or an incalculable amount at each of the follow-ups respectively.

There were no patients with ASD that met the criteria after the medical record review. Hence, the following data pertain only to PFO patients.

Table 1

Defect size (mean SD) and closing device. Echo result after closure based on the number of agitated saline contrast bubbles found in the left heart when provoked with a Valsalva manoeuvre. * One patient did not have a follow-up after one year. In that case no residual shunt was detected with echocardiography after 24 h and after seven days

Parameter	
Initial defect size (mm)	7 (SD 3)
Complications after closure	Nil
Closing device (*n*)	
Amplatzer PFO Occluder 25 mm	6
Gore Septal Occluder 25 mm	2
Noble Stitch	1
Echo at 24 h (*n*)	
1–10 bubbles	3
No residual shunt	6
Echo at one year*(*n*)	
1–10 bubbles	2
No residual shunt	6

Table 2
The total number of dives and median number of dives, separated by the breathing gas used, performed by the nine divers before closure, and by the eight divers that dived post-closure

Period	Total, median (range)	Compressed air *n* (%)	Nitrox *n* (%)	Trimix *n* (%)
Dives before closure	6,835, 410 (140–2,200)	4,950 (72)	1,320 (19)	565 (8)
Dives after closure	4,708, 413 (11–2,000)	2,639 (56)	1,507 (32)	562 (12)
Dives in the last year	539, 19 (0–250)	269 (50)	250 (46)	20 (4)

Table 3
The number of decompression sickness (DCS) events and depths of incident dives before and after closure, separated by the breathing gas used. * = Median (range). ** = Range. Six of the seven incident dives during use of trimix were performed by the same diver, and depth data could only be provided for one of these six dives (76 m)

Parameter	Total	Air	Nitrox	Trimix
DCS before closure	17 1 (1–6)*	9 (53%)	1 (6%)	7 (41%)
DCS after closure	1	0	1	0
Depth (m) of incident dive before closure		31 (15–49)*	36	73–76 **
Depth (m) of incident dive after closure		–	19	–

Results

From 1997 up until the end of 2017, a total of 603 PFO were closed; 13 of these were performed because of previous DCS. Out of these 13 PFO patients, four chose not to participate in the phone interviews, leaving nine persons who agreed to participate. Five of them were professional divers and four were recreational divers (one female). The subjects had a mean age of 29 (SD 4) years and a mean BMI 26 (3) $kg \cdot m^{-2}$ at the time of PFO closure. A sizing balloon was positioned in the PFO in all cases to measure the stretched diameter of the defect. The PFO defects were mean 7 (SD 3, range 4–10) mm (Table 1).

There were no complications associated with the closure. At one-year follow-up six patients demonstrated complete closure while two patients had a residual shunt of 1–10 bubbles (Table 1). The median time after closure at which interview for this study was conducted was seven years (range 2–18). A total of 6,835 dives were performed before PFO closure (median per subject 410, range 140–2,200). One diver did not dive after the closing procedure. The other eight divers performed a total of 4,708 dives after closure (median 413, range 11–2,000) ranging in depth from 20–100 metres (m) (Table 2). One stopped diving six years after the PFO closure, and one diver stopped after another incident of DCS. Six divers were still diving at the time of the interview.

In total, 17 DCS events (seven mild, ten serious) were reported before closure (median 1, range 1–6) (Table 3). Oxygen therapy was used in 15 out of 18 events, the one post closure DCS included. HBOT was used in 10/18 cases. The reported diving depths for the dives that caused DCS varied between 15 and 76 m and are summarised in Table 3. One DCS event was reported after the closure procedure (Table 3). The affected diver was the only female included in the study. She was one of two patients who had a residual shunt one year after closure (Table 1). She performed 300 dives with compressed air before the closure, and had suffered three DCS events. After the intervention, she performed nine dives with compressed air and two with nitrox. The dive that caused DCS was performed with nitrox to 19 msw. She experienced a serious DCS after the PFO closure but was successfully treated with hyperbaric oxygen with complete symptomatic relief. However, the incident dive was performed with a provocative diving profile that potentially could increase the risk of DCS. It is notable that the information about the provocative diving profile was added by the diver voluntary at the end of the interview, and not as an answer to our predetermined questions. The second diver who had a residual shunt one year after closure did not suffer DCS in 125 dives post-closure.

Discussion

Divers with PFO and previous DCS events are currently recommended to dive more conservatively to reduce the risk of recurrent DCS.[18] PFO screening is generally recommended when DCS occurs after a non-provocative dive, after neurological or repetitive DCS events.[19] Several authors have suggested that PFO closure would abolish the increased risk of DCS events associated with PFO.[15,16,20–22]

This proposal is supported by the finding that chamber dives producing venous gas emboli in the majority of subjects resulted in arterial bubbles in some divers with PFO but in no divers with a catheter-based PFO closure.[23] However, 'dry' divers have been shown to produce fewer venous gas emboli than submerged dives.[19]

To our knowledge there are five studies of DCS outcomes after PFO closure. In two studies,[14,24] no episodes of major DCS were reported after PFO closure in 11 and 20 divers respectively. Another showed a decreased DCS incidence after PFO/ASD closure.[16] In the study by Billinger et al., there was one case of serious DCS after PFO closure and this occurred in the only diver out of 26 who had a residual shunt.[20] In the latest study by Honek et al. PFO closure was shown to prevent DCS.[25] In the present study one subject out of nine experienced a DCS event after the closing procedure but this subject undertook a provocative diving profile and had a residual shunt. A possible conclusion from this is the importance of the follow-up echo examination after the intervention. If a residual shunt is detected, we suggest it would be wise to recommend conservative diving profiles.

Estimates of the DCS risk per dive is 0.095% for commercial divers and 0.01–0.019% for recreational divers.[4] It has previously been proposed by two studies that PFO increases the DCS risk 2.5–5 times.[10,26] Based on the DCS risks described above, 0.01-0.095%, combined with these increased risk estimates, the expected number of DCS events pre closure in our cohort (6,835 dives), would be 2–32 events, we report 17. If the DCS risk post closure (4,708 dives) is estimated on the numbers above but without the increased risks associated with PFO 0.5–4 events would have been expected, we report one. Hence, our results correspond to the risks previously described in the literature.

In the questionnaire, we chose to include maximum diving depth after the closure. AGE can occur even after ascent from shallow diving.[4] Results from a study where divers performed saturation dives to certain depths and then ascended without decompression stops, indicate that DCS is uncommon at depths shallower than 6–9 metres.[27] All participants in our study, except one that had not performed any dives after the closure, had been diving deeper than 20 metres after the closure. Thereby, they had exposed themselves to conditions that theoretically could cause both DCS and AGE. This exposure to dives carrying a risk of DCS strengthens our conclusion that PFO-closure protects against recurrence. Among this small group of Swedish divers a large majority continued to dive after the PFO closure. This is important because if the divers would not attempt to dive again due to fear of recurrent DCS, the benefit of the intervention would have been called into question.

In deciding whether to undertake PFO closure after DCS the risks of PFO closure must be taken into account. In a study including 825 patients, overall device implantation failed in 0.2% of the interventions. Complication rate was 2.2% and most common were embolisation of the device in 0.6% of the cases.[28] The PFO closing procedure seems reasonably safe and feasible, but the risks should still be considered before recommending the intervention.

Our study cohort was small, and the number of dives performed made calculations of DCS risk precarious. However, there are no very large studies so the combined findings of these studies, including ours, could be of importance in the future recommendations regarding diving for patients with a closed PFO. Since retrospective reviews were conducted on a large number of medical charts, another potential weakness is that we cannot be entirely sure that all relevant patients were identified. There is a potential selection bias among the included patients. One could speculate that some divers, especially recreational divers, with a PFO who suffer DCS prefer to stop diving rather than have the PFO closed. We have not focused on the diving profiles in this study, because we did not do a logbook review and it is possible that divers dived more conservatively after PFO closure. A logbook review would have been time-consuming and potentially unfeasible in a retrospective study like this. In a future study it would be ideal to only include dives where the diver had strictly followed decompression tables.

Conclusions

Divers who suffer certain forms of DCS may be recommended to undergo investigation for a PFO and, if a large PFO is discovered, to close it to reduce the risk of recurrent DCS before resuming diving. After PFO closure it is important to check for residual shunting as this may be associated with a persistent increased risk of DCS. If there is a residual shunt and the diver wishes to continue diving, conservative profiles are recommended. These results suggest that divers with PFO who have experienced DCS and undergone PFO closure, don't need to cease diving after the intervention.

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Hyperbaric oxygen for sudden hearing loss: Influence of international guidelines on practice in Australia and New Zealand

Susannah Sherlock[1,2], Sharon Kelly[3], Michael H Bennett[4,5]

[1] *Hyperbaric Medicine, Royal Brisbane and Women's Hospital, Brisbane, Australia*
[2] *Senior Clinical Lecturer, University of Queensland, Brisbane, Australia*
[3] *Department of Ear Nose and Throat Surgery, Royal Brisbane and Women's Hospital, Brisbane, Australia*
[4] *Department of Diving and Hyperbaric Medicine, Prince of Wales Hospital, Sydney Australia*
[5] *Prince of Wales Clinical School, University of New South Wales, Sydney, Australia*

Corresponding author: *Dr Susannah Sherlock, Royal Brisbane and Women's Hospital, Butterfield St, Herston QLD 4029, Brisbane, Australia*
susannah.sherlock@health.qld.gov.au

Key words
ENT; General interest; Hearing loss, sudden; Hyperbaric facilities; Hyperbaric oxygen therapy; Medical society

Abstract
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Introduction: Idiopathic sudden sensorineural hearing loss (ISSHL) is an otolaryngologic emergency. The Undersea and Hyperbaric Medicine Society (UHMS) revised practice guidelines in 2014 adding ISSHL to approved indications. This study investigated whether the UHMS guidelines influenced referral and practice in Australia and New Zealand.

Methods: Retrospective review of 319 patient referrals in two time periods (five years prior to addition of ISSHL to indications (T-PRE) and three years post (T-POST)).

Results: Seven of eight participating hyperbaric facilities provided data down to the level of the indication for HBOT for analysis. In T-PRE 136 patients were treated with HBOT for ISSHL, representing between 0% and 18% of the total cases to each facility. In the T-POST period 183 patients were treated for ISSHL, representing from 0.35% to 24.8% of the total patients in each facility. Comparison between the two periods shows the proportion of patients treated with ISSHL among all indications increased from 3.2% to 12.1% ($P < 0.0009$). One facility accounted for 74% (101/136) of ISSHL patients receiving HBOT in T-PRE and 63% (116/183) in T-POST. ISSHL case load at that facility increased from 18% to 24.8% ($P = 0.009$) after the UHMS guideline publication. Three of the seven units had a significant increase in referrals after the guideline change.

Conclusion: There remains equipoise regarding HBOT in the management of ISSHL. Only three out of seven units had a significant increase in ISSHL patients after the UHMS guidelines publication. Without well controlled RCTs to develop guidelines based on good evidence this is unlikely to change and practice variation will continue.

Introduction

Idiopathic sudden sensorineural hearing loss (ISSHL) is considered an otolaryngologic emergency. The clinical practice guidelines published in 2012 and updated in 2019 by the American Academy of Otolaryngology and Head and Neck Surgeons (AAOHNS) suggest consideration of hyperbaric oxygen treatment (HBOT) within two weeks of symptom onset.[1,2] The Undersea and Hyperbaric Medicine Society (UHMS) revised their guidelines in 2014 with the addition of ISSHL to the approved list of indications. The society also recommend treatment within two weeks of symptom onset for initial treatment or within four weeks if used as salvage treatment.[3] This study was designed to ascertain whether the publication of the UHMS guidelines influenced referral patterns and practices in Australia and New Zealand (A/NZ).

Methods

HREC (ethics) exemption was provided as a quality assurance project by the Royal Brisbane and Women's Ethics Committee (LNR/2019/QRBW/60494).

This was a retrospective cohort study of 319 patients with ISSHL who received HBOT in A/NZ facilities during two defined time periods before and after ISSHL was added to the UHMS guidelines. ISSHL was defined using criteria described by the National Institute on Deafness and Other Communications Disorder for consideration of HBOT.[4] Data were collected over a five-year period (Jan 2010 to Dec 2014) from eight participating units collaborating on a previously published study and compared to a data set collected over a 3-year period after the UHMS added ISSHL to their indication list.[5]

Data for the second time period (July 2016 to June 2019) was provided by the Hyperbaric Technicians and Nurses Association (HTNA) and is collected annually for their scientific meeting. The first time period is designated T- PRE and the second time period is designated T- POST. An e-survey was sent to participating units to follow up on reasons for practice variation in 2018. A total of 6,284 patients received HBOT during the study periods. One unit treated enough patients to be independently analysed and the other units' data were combined, due to small numbers, to allow comparison between the two time periods.

Comparison between groups was made using Chi-square analysis of difference in proportions, or Fischer's exact methods if any cell contained fewer than five individuals. A *P*-value of < 0.05 indicated a statistically significant result.

Results

Seven of the eight participating hyperbaric facilities were able to provide data down to the level of the indication for HBOT for analysis (facility six was excluded from analysis, (Table 1)). In the T-PRE period 136 patients were treated with HBOT for ISSHL, representing between 0% and 18% of the total cases to each facility (Table 1). Two facilities reported no patients treated with ISSHL. In the T-POST period 183 patients were treated with ISSHL, representing from 0.35% to 24.8% of the total patients in each facility. Three facilities did not provide full data for the calendar year 2017 (facilities 3, 4, 5). The comparison between the two periods suggests the overall proportion of patients treated with ISSHL increased from 3.2% to 12.1% (Chi-sq = 128.9, $P < 0.0009$).

One facility dominated the figures accounting for 74% (101/136) of all ISSHL patients receiving HBOT in A/NZ in T-PRE and 63% (116/183) in T-POST. Data from that facility showed a statistically significant increase in case load after the UHMS guideline was introduced from 18% to 25% ($P = 0.009$). The comparison for other individual units is shown in Table 1. Three out of seven units had a significant increase in referrals over the period. Two of these units were in the same Australian state.

There was wide variation between facilities in the dose of oxygen used, both in terms of treatment pressure and duration. The pressures used were 202.6 (one facility), 243.1 (six facilities) and 283.6 kPa (one facility), for periods between 90 and 120 minutes for each session. Many units were unable to provide data concerning the actual number of HBOT sessions each patient received as this is not routinely collected for HTNA datasets. Three facilities treated only Monday to Friday, whilst three treated their patients without interruption over weekends.

There were very few referrals in some states but large numbers in others. The frequency of referrals varied greatly between locations when followed up by eSurvey, with 6 units responding. One hospital received more than one per week, one more than one per month, one less than one per month, and three less than two per year.

Table 1

Treatment data from participating hyperbaric units pre- and post-publication of the UHMS guideline accepting ISSHL as an indication for HBOT. * Data for 2017 missing. † Fisher's exact test

Facility	T-PRE (2010–2015)			T-POST (2017–2019)			Chi-square (*P*-value)
	Patients receiving HBOT	Patients with ISSHL	Proportion ISSHL (%)	Patients receiving HBOT	Patients with ISSHL	Proportion ISSHL (%)	
1	558	101	18.1	467	116	24.8	6.92 ($P = 0.009$)
2	1,225	20	1.6	939	25	2.7	2.5 ($P = 0.12$)
3	275	0	0	64*	0	0	–
4	972	3	0.3	253 *	9	3.5	18.6 ($P < 0.001$)†
5	228	3	1.3	50 *	0	0	–
6		Not reported			Not reported		–
7	515	9	1.7	347	32	9.2	25.6 ($P < 0.001$)
8	473	0	0	285	1	0.4	0.06 ($P = 0.38$)†
Total	4,246	136	3.2%	1,571	183	11.6%	128.9 ($P < 0.001$)

Discussion

The adoption of guidelines into clinical practice can be variable. In Australia there has been a tendency to adopt the UHMS guidelines for HBOT indications as they are regularly published and are evidence based. With the amalgamation of the South Pacific Underwater Medical Society (SPUMS) and the European Underwater Baromedical Society (EUBS) as co-publishers of this journal, this may change and some units may refer to the European Committee for Hyperbaric Medicine (ECHM) Consensus Statement from 2016 which also recommends HBOT for ISSHL.

Whichever guideline is more popular, in Australia and New Zealand the majority of units do not receive referrals from otolaryngologists and this continues to be the case. This is similar to the UK experience which showed that 96% of otolaryngologists in 2014 did not use HBOT to manage ISSHL despite the EUBS recommending HBOT for ISSHL in 1994.[6,7] This may reflect both the quality of the evidence and the behaviours of both patients and clinicians.[8] Patient preferences have been shown to be a barrier for general practitioners following guidelines.[9]

ISSHL management remains controversial. The definition, spontaneous resolution rate, best drug therapy and best outcomes to measure response have all been disputed.[10] This has hampered research in the area and made meta-analysis difficult as trial protocols comparing steroids and HBOT vary widely in dose of both steroids and oxygen, and for the route of administration of steroids. Many studies describe the steroid protocol in detail but provide no detail on the HBOT protocol.

A particular problem is the reporting of outcome measures across the many small outcome studies published to date. While many studies employ the pure tone audiogram (PTA) thresholds over different frequencies (PTA4 or PTA6), they inconsistently report the changes as 'mean threshold', 'absolute improvement in threshold' or 'proportional improvement in threshold', none of which can be combined without access to the raw data. There is little or no reporting of any patient–centred outcomes such as functional ability, quality of life or speech discrimination scores.

Evidence-based clinical practice guidelines by both otolaryngologists and hyperbaric groups agree HBOT should be started within two weeks of onset for initial management, and this has helped reduce some of the practice variation. However, the adoption of guidelines is not universal and considerable local differences in practice persist. Either otolaryngologists do not refer such patients, hyperbaric physicians do not accept them or both. Non-acceptance of guidelines often occurs due to a poor evidence base and the resulting vague and unhelpful guidelines for practice. Unfortunately the literature on ISSHL is generally of poor statistical quality.

A 2012 Cochrane Review analysed seven studies on HBOT for ISSHL and concluded HBOT probably improved outcomes, but the clinical significance of the improvement remains unclear due to small patient numbers and poor methodology.[11] A more recent review in 2018 concluded no significant difference between studies comparing steroids to steroids plus HBOT other than in patients with severe to profound loss. The review included 16 studies with various methodologies. Only two studies, contributing 117 patients in total, were randomised controlled trials, out of the 1295 patients included in the analysis.[12] The evidence for steroids in ISSHL is similarly contradictory and of poor quality.[13]

Facilities providing HBOT require a referral from a specialist who is managing the patient with ISSHL. The unit with the largest number of referrals usually only accepts referrals which are within the AAOHNS guidelines. Patients are not accepted if a patient has actively lobbied for a general practitioner to refer them without specialist input. While the AAOHNS advise those managing ISSHL to consider HBOT if within two weeks of onset, it seems the referral rate remains very low in A/NZ. While this may reflect a reluctance to consider HBOT as a viable alternative for geographical or financial reasons, it is possible the low referral rates reflect either late presentation to an otolaryngologist or a reluctance to refer to HBOT until a failure to respond to steroids is clear. The most active hyperbaric facility in this area confirms a high rate of late referral where the patient is unlikely to derive benefit from HBOT.[14]

As is the case for other indications, the differences in HBOT protocols probably reflects the historical treatment protocols used in different facilities. Any treatment involving 100% oxygen breathing between 202.6 kPa and 253.3 kPa, for 90 minutes and repeated 10 to 20 times is within the UHMS guideline. There is no guidance on the frequency of these sessions – daily or twice daily, or even whether they should be consecutive (including weekends), or only Monday to Friday. There was extensive variation in the number, timing and duration of air breaks for those units using a 243.1 kPa table. While air breaks were historically introduced to reduce pulmonary oxygen toxicity, many units now use them in the belief they may reduce central nervous system toxicity. A recent study did not support this supposition,[5] though another does.[15] The majority of units did not collect any meaningful quantitative outcome data.

Conclusion

There is considerable clinical equipoise remaining in the management of ISSHL and the place of HBOT. Only 3 out of 7 units had a significant increase in patients treated with HBOT after the UHMS guidelines were published. One State accounted for the majority of patients who received HBOT. Without well controlled RCTs to develop guidelines based on good evidence this is unlikely to change and practice variation will continue.

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Patient knowledge and experience of hyperbaric oxygen treatment

Louise MacInnes[1], Carol Baines[1], Alexandra Bishop[2], Karen Ford[3]

[1] *Department of Diving and Hyperbaric Medicine, Royal Hobart Hospital, Hobart, Tasmania, Australia*
[2] *DDRC Healthcare, Hyperbaric Medical Centre, Plymouth, Devon, UK*
[3] *Tasmanian Health Service South, Tasmania and University of Tasmania, Hobart, Tasmania, Australia*

Corresponding author: *Dr Carol Baines, Department of Diving and Hyperbaric Medicine, Royal Hobart Hospital, Tasmania 7000, Australia*
carol.baines@ths.tas.gov.au

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Abstract
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Introduction: This paper presents a quantitative and qualitative study exploring patients' knowledge and experience of hyperbaric oxygen treatment (HBOT).
Methods: Participants included 29 patients with appropriate indications who were undertaking HBOT at facilities in two different locations: Hobart, Australia, and Plymouth, United Kingdom. Participants completed surveys prior to commencing HBOT, after five sessions, and on completion of HBOT. Semi-structured one-to-one interviews were conducted with each individual on conclusion of their course. Data were analysed using descriptive statistics and interpretive description.
Results: Prior to referral, 15/29 (52%) of participants knew HBOT was used to treat divers, and of these, 9/15 (60%) were familiar with its use for non-divers. Only one third sought additional information about the process between referral for HBOT and attending their medical assessment. Anxiety was a pre-treatment concern amongst participants. However, when re-measured after five sessions and upon completion of the HBOT course, anxiety was reduced. The interview data revealed themes based around the physical, emotional and social aspects of HBOT: (1) anxiety within self; (2) naivety to normalisation; (3) enjoyment being a 'diver'; and (4) burdens of HBOT.
Conclusions: Many patients experienced anxiety prior to commencing HBOT but, with support, quickly adjusted to treatment, transitioning from a state of naivety to normalisation in their experience of the hyperbaric chamber. They enjoyed feeling like a 'diver' and considered aspects of the burdens of treatment, such as finances or logistics, a minor inconvenience. These results highlight the need for psychosocial support during treatment by identifying gaps in patient preparation for HBOT.

Introduction

Hyperbaric oxygen treatment (HBOT) is a systemic medical intervention in which the patient inhales 100% oxygen at greater than one atmosphere pressure within the confines of a purpose-built hyperbaric chamber.[1] HBOT is prescribed by specialist medical practitioners for a number of acute medical conditions, including decompression illness, and is also utilised as part of the medical management for patients with chronic conditions, such as hypoxic wounds, soft tissue radionecrosis or osteonecrosis.[2]

HBOT takes place in either a mono-place (single person) or multi-place chamber; the latter can seat multiple patients as well as a healthcare professional (often a registered nurse (RN) or a member of staff with specific medical skills).[3,4] HBOT is usually administered daily, five days per week, with 30–40 consecutive treatments typically considered a full course of treatment for wound and radionecrosis indications. The multi-place chamber provides a unique situation in healthcare, with the inside attendant and the patient together sharing many components of the HBOT experience, including being compressed to a prescribed increased ambient pressure.

Despite these shared features, understanding the patient's own experience of HBOT could inform improvements in patient-centred care and specifically patient's care needs when undergoing HBOT.[5] A thorough exploration and understanding of the burdens, such as anxiety and ear pain, associated with HBOT as experienced by patients may help drive practice innovations.

The aims of this study were to explore individual patient knowledge of HBOT, identify the resources that improve patient knowledge, and to explore and compare patient experiences of HBOT. Also to assess how the treatment impacts the daily life of the patient. The study was conducted at two centres in two different countries.

Methods

This study was approved by the Tasmanian Human Research Ethics Committee (UTAS HREC No: H0016784) and

conducted in accordance with National Health and Medical Research Council (NHMRC) guidelines and relevant institutional governance procedures. Ethical approval was not required in the United Kingdom (UK) as the study was considered an evaluation of the service as confirmed with the Research and Development Department of the Plymouth facility.

SETTING

The Australian study setting was the Department of Diving and Hyperbaric Medicine at the Royal Hobart Hospital, a tertiary hospital in Hobart in the state of Tasmania. Data were collected from February to September 2018. HBOT was undertaken in a monoplace chamber or a cylindrical multiplace chamber.

The study setting in the UK was a private hyperbaric medical centre, the Diving Diseases Research Centre (DDRC Healthcare), Plymouth. Data were collected from February 2018 to April 2019. HBOT was delivered in a monoplace chamber or cylindrical multiplace chamber.

RESEARCH TEAM

Interviews were conducted by two RNs in Hobart and three in Plymouth, all with substantial clinical experience in the specialist field of hyperbaric and diving medicine and all of whom were involved in the direct care of the study participants. Reflexivity, through deliberate and open discussion between researchers, allowed for identification of preconceptions, and to distinguish between intuitive knowledge and new emerging knowledge from data analysis. Each research centre reviewed the data and identified codes and developing themes. The Hobart research team included an independent researcher who was not directly involved in patient care.

RECRUITMENT

Adult patients who were undertaking their first course of HBOT for either a chronic hypoxic wound, soft-tissue radiation injury or osteoradionecrosis were invited to participate. Patients who had previously received a course of HBOT and those who were due to commence HBOT > 4 weeks post-initial assessment were excluded. Similarly, patients receiving HBOT under a 'Marx Protocol' (prophylactic course of hyperbaric oxygen treatment undertaken prior and post-surgical/dental procedure) were excluded.

PROCEDURE

The same study methods, which combined quantitative and qualitative components, were employed at both the DDRC and the Royal Hobart Hospital. Data were obtained via surveys administered at three pre-set time points, and a semi-structured interview with each participant. The pre-set time points were immediately prior to commencing their first HBOT, after the fifth HBOT and after their final HBOT.

The survey was paper/computer-based and consisted of multiple choice. Likert scale and open-ended questions, which all participants were invited to complete. The questions were designed to explore individual knowledge of HBOT, the experience of undertaking HBOT and the impact of HBOT on participant's lives. Descriptive data, including age and total number of HBOT treatments undertaken during this study, were also collated. The descriptive statistics utilised in this study were not designed to determine statistical significance but to summarise data and provide a richer context.[6]

The semi-structured, one-to-one interview (phone or face-to-face) took place at the completion of the course of HBOT. Each interview was audio-recorded, transcribed verbatim and analysed by two members of the research team.

The study was underpinned by the principles of interpretive description; a constructivist and naturalistic orientation to enquiry utilising inductive analytic approaches. This method allows researchers to illuminate the characteristics, patterns and structure of clinical phenomena in order to generate knowledge relevant for the clinical context.[7–9] Thematic analysis was undertaken as described by Braun and Clarke.[10]

Each participant provided written consent, which was confirmed verbally at each interaction with the research team. Whilst no participants withdrew from the study, not all participants completed all four elements.

Results

Thirty-one patients were initially recruited, but two were excluded following consent as they did not go on to start HBOT within four weeks of assessment. Of the remaining 29 participants, 20 were based in Hobart (HBT) and nine in Plymouth (PLY). Hobart participants included 12 males

Table 1
Demographics of study participants. DW – diabetic wound; NDW – non-diabetic wound; ORN – osteoradionecrosis; RC – radiation cystitis; RP – radiation proctitis; STRN – soft tissue radiation necrosis

Characteristic	Hobart	Plymouth
Participants (*n*)	20	9
Sex (male / female)	12 / 8	6 / 3
Age range (years)	31–84	58–78
Mean HBOT sessions per participant	32	41
Reason for HBOT (*n*)	DW (12) RP (4) RC (4)	NDW (1) RP (2) ORN (5) STRN (1)

and eight females, with an age-range of 31 to 84 years. The average number of HBOT sessions per person was 32, and the predominate diagnosis was a hypoxic wound secondary to diabetes mellitus. The nine participants in Plymouth comprised six males and three females, with an age range of 58–78 years. The average number of HBOT sessions per person was 41, and the predominant diagnosis was osteoradionecrosis (Table 1).

SURVEY DATA

The survey questionnaires and interview results were combined in order to interpret responses as a whole. Duration of the interviews was between 4–21 minutes across both sites. Prior to referral for treatment, some participants (15/29, 52%) had some knowledge or awareness of HBOT. Of these, all knew it was used to treat divers, but some (9/15, 60%) were also aware that HBOT was used to treat other conditions such as wounds and radiation injury. Only two Hobart participants knew what either a multiplace or monoplace chamber looked like and none knew what a treatment course involved. A minority of participants (9/29, 31%) sought additional information about the process between referral and attending their medical assessment with eight (HBT = 3, PLY = 5) utilising the internet and eight (HBT = 4, PLY = 4) speaking to a health professional.

Participants had little difficulty dealing with the physical aspects of HBOT, with 18/29 (62%) finding it easy to equalise pressure in their ears. These findings remained consistent across five treatments and at completion of HBOT, across both sites. There were reports of tiredness/fatigue from five Hobart participants after completing five treatments of HBOT, and this increased to 10 participants at the completion of HBOT. Fatigue was not in Plymouth.

Of the Hobart participants, 12 indicated they had experienced changes in their vision, five had no concerns about their vision, and two expressed difficulty in dealing with these changes. Four participants from Plymouth reported visual changes that were 'manageable'.

Data from both sites indicated the majority (n = 20) of participants considered that HBOT did not take up too much of their time or impair their ability to work or undertake social activities. Participants did not find it financially difficult to attend HBOT as treatment was offered at no cost to patients at both sites and some support was available to assist with transport and accommodation through non-government organisations. Participants from both sites reported logistical considerations, such as travel and car parking which was arranged by participants, to be a self manageable burden.

IDENTIFICATION OF THEMES

Four key themes, outlined below, were identified. These were: anxiety within self; moving from naivety to normalisation; enjoying being a 'diver'; and burdens of HBOT are a 'minor inconvenience'.

Anxiety within self

Anxiety within the participant group was measured at three separate time-points using a five-point Likert scale. The same question was asked at the conclusion of HBOT, and results showed a reduction in participant anxiety after five treatments and throughout HBOT (Table 2).

Moving from naivety to normalisation

Participants had little overall knowledge of HBOT prior to treatment. However, they quickly moved from a naïve emotional response to a sense of normalisation and acclimatisation to the (previously) unfamiliar environment of HBOT.

Participants at both sites expressed initial anxiety. One had "*visions of tubes in the mouth*" (participant 16, Hobart) and another said, "*I thought they* [the mono-place chambers] *looked like coffins*" (participant 1, Plymouth). One participant explained: "*You're closed in, you go into a pressurised environment. The first day I found it strange … but after that, I didn't find any impost on my body at all*" (participant 20, Hobart).

Some participants reported a quick reduction in anxiety after the initial treatment, with one explaining, "*I think at the beginning I was maybe a bit anxious, a bit worried, not knowing what to expect to happen. But then it went very smoothly… It felt like it was secure and safe …so I was just anxious because it is new…you have to experience it to feel*

Table 2

Participant response to "*I feel anxious about going into the hyperbaric chamber*". Data are number of patients responding per category. * incomplete

Survey time	Strongly disagree	Disagree	Neutral	Agree	Strongly agree
Pre-treatment	7	3	10	5	4
Post five treatments	18	4	2	1	1
On completion*	19	5	0	1	2

Table 3
HBOT delivery method. Data are number of patients

Chamber type	Hobart	Plymouth
Monoplace only	3	0
Multiplace only wearing Amron Hood ™	2	4
Treatment in both chambers	15	5
Total number of participants	20	9

better about it, I think" (participant 14, Hobart) and another reported, "*As soon as I had done it once,* [I found it] *quite relaxing*" (participant 16, Hobart).

Most participants experienced HBOT in both mono- and multiplace chambers (Table 3). A number of participants attributed their familiarisation with this new HBOT environment to interactions with the staff member inside the multiplace chamber on their first dive: "*Having the first dive with somebody else – it gave me the ability to ask questions, have questions answered, not be at all concerned about, you know, ears popping and so on and so forth, it was great…she made me feel really comfortable*" (participant 18, Hobart) and "*having company in there made it okay*" (participant 7, Plymouth).

For some, receiving treatment in a hyperbaric chamber of their choice was important. All but one ("*I sort of enjoyed the interaction with the chamber assistants*" (participant 11, Hobart)) of the participants who experienced both chamber types preferred the monoplace chambers. Lying in the monoplace chamber was described as "*more comfortable*" (participant 12, Hobart) and "*far more civilised*" (participant 5, Hobart). The movies in the monoplace were "*a good distraction*" and "*made the time pass faster*" (participant 9, Hobart). In Plymouth, patients could watch a film in the multiplace as well as the monoplace chambers, so this factor did not influence hyperbaric chamber choice. The Amron ™ hoods (Amron, California, USA), which are the usual method used to administer oxygen in the multiplace chamber, were described as "*annoying and uncomfortable*" by several participants (participants 5, 7, 12, 14, 19, Hobart; participant 5, Plymouth).

Being in the "*right mindset*" was seen to be important (participant 6, Hobart). As explained by participant 8, Hobart: "*You just had to tell yourself that it was alright*", and, "*I had to go in there and I knew I had to, so I just trusted myself and that's all there is*" (participant 3, Hobart). This view was similarly expressed by others: "*It's just a matter of convincing myself that it is doing me good*" (participant 18, Hobart), and, "*by reading a book the time seemed to pass more quickly*" (participant 9, Plymouth).

By the completion of their course, 13/29 (45%) of participants described the experience of having HBOT as "*normal*" and many were dismissive of any specific physical or psychological experience associated with being in the hyperbaric chamber: "*Whilst I'm inside the chamber itself, it feels normal*" (participant 18, Hobart).

Enjoying being a 'diver'

The machinations of the hyperbaric chamber and unique social experience quickly led to participants assuming the identity of a 'diver'. This was evidenced by the participant's use of language and diving-specific jargon, both within the hyperbaric facilities and in the community with family and friends. Several participants spoke enthusiastically about sharing their experience with others: "*They are all interested …* [and] *seem to be very keen on knowing what it is all about*" (participant 19, Hobart). "*I talk to everyone about it…It's been a joke all along…in the sense: 'where are you going today?', 'You know where I'm going – diving!*'" (participant 4, Hobart) "[They] *think you are really diving, but you are not diving ...now I have a picture of me in it so I can actually show them what it is!*" (participant 17, Hobart).

The delight in having photographs both of the hyperbaric chambers and participants themselves receiving treatment was a way of sharing "*something you have never experienced in everyday life*" (participant 17, Hobart) and involving family and friends in care. Recording the experience with photographs seemed more in keeping with an adventure or recreational activity than a medical procedure, which in turn contributed to the experience of hyperbaric oxygen treatment as enjoyable.

Interactions with healthcare professionals alleviated potential difficulties associated with HBOT. "*It was pleasant… I come in every day, watch a movie, talk to* [the technician] *and everybody else, it's all good fun*" (participant 6, Hobart) and "*Support from all the staff, absolutely brilliant*" (participant 5, Plymouth). The technicians (who operate the hyperbaric chambers) were particularly identified as creating a positive atmosphere: "*They tease, particularly one… it makes the day*" (participant 1, Hobart).

Burdens of HBOT are a 'minor inconvenience'

Participants described the logistical considerations and impact of attending a course of HBOT as burdensome. However, these difficulties were largely accepted in light of their positive outcomes: "*I just considered it a minor inconvenience for the benefit*" (participant 18, Hobart), and, "*If it's going to prove successful I think that it is worth doing*" (participant 3, Plymouth).

Others felt they were an encumbrance: "*I did feel guilty that I had to rely on other people... I felt I was a bit of a burden and an imposition*" (participant 7, Hobart). The strain on

relationships with family increased over time and was most evident in those who needed to relocate for the duration of HBOT away from their relational networks "[The] *only problem for me was that I've been away from home for eight weeks*" (participant 8, Plymouth).

Fatigue was another burden of a long treatment course. Responses to fatigue varied, however, with some participants finding it frustrating and others relishing the opportunity for additional naps or to improve their sleep pattern. Some acknowledged that fatigue was not just related to the treatment itself or the length of the course, but the cumulative effect of a long medical treatment journey, one component of which was HBOT.

Participants in Plymouth raised boredom as a factor that they needed to overcome and recommended to others to address this issue by "*bringing a book to read*" (participants 3 and 9). This was not noted by participants in Hobart.

Oxygen-induced myopia was a troubling medical side effect of their treatment. This impacted on participants' everyday lives: "*The vision thing has been quite difficult to get used to* [but it's a] *small price to pay*" (participant 18, Hobart). Another stated "*I hate it, I really hate it but what can you do? You have just got to put up with it*" (participant 19, Hobart). Two participants in Plymouth reported improvement in vision, "*So that's a plus"* (participant 9).

Despite burdens, participants all wished to continue with treatment, "*There are the downsides and everything, but I mean, if it's making me better, I've got no problems with it. I've just got to live with the other parts of it*" (participant 17, Hobart). Burdens were described as a "*small price to pay... benefits far outweigh the negatives*" (participant 18, Hobart). Advice for other patients attending for treatment included, "*Don't worry about it, go for it*" (participant 5, Plymouth).

Although some participants were pleased to finish their course of treatment, levels of engagement remained high throughout their HBOT course, some expressing sentiments of loss upon completion, and many were willing to revisit HBOT in the future. The general feeling was: "*I'm glad I've done it and I wouldn't hesitate to do it again if I had to*" (participant 8, Hobart).

Discussion

The range of feelings and emotions presented by participants across both study sites are similar to findings by Chalmers et al, who reported that "*treatment uncertainty can subsequently provoke feelings of anticipatory apprehension and anxiety based on fear of the unknown*".[11] This phenomenon is recognised in interventions such as magnetic resonance imaging (MRI), but there is limited commentary concerning anxiety experienced by patients undertaking HBOT.[12,13] Studies have shown that anxiety typically abates following initial HBOT as treatment becomes familiar and is perceived as unproblematic.[14,15] Our research showed that the experience of anxiety prior to initial HBOT was considerable. The unfamiliarity of the new treatment, having to trust a new and highly technical environment and a fear of the unknown could be impediments to commencing or continuing treatment. These issues must be sensitively but proactively addressed by clinicians, particularly at the beginning of the patient journey.

The sense of belonging created by technical and other staff appears to be instrumental in the development of the patient identity as a 'diver'. Perhaps uniquely within healthcare, HBOT offers the opportunity to reframe identity given the culturally appealing connotations of diving and staff who may themselves be divers, and a willingness to confer membership of this social group to patients. Consistent with social identity theory and self-categorisation theory, this self-identification appears to move patients out of the traditional sick role and provides an alternative self-concept, with associated pride and self-esteem derived through belonging to and identifying with a social group utilising jargon and slang to cement membership, denote status and provide social capital.[16,17]

Despite these positive connotations, HBOT remains a medical treatment requiring participation and adherence to regulations by patients. Studies indicate that patients with chronic conditions experience burden not only from their chronic disease but also from the impact and workload of treatment regimens, which in turn affects patients' experience and links to their self-management strategies.[18–23] This study shows that perceived burdens were not an impediment to participants initially engaging with treatment. Whilst the experience of burden somewhat changed over the duration of the HBOT, participants and their relational networks had sufficient capacity and resilience to enable them to maintain attendance.

Conclusions

This study has identified key components of the patient experience of HBOT. Whilst there has previously been little research to guide practitioners, this study suggests that opportunities for improved practice could focus on addressing the initial anxiety felt by participants, supporting them to transition from a state of naivety to normalisation within the hyperbaric chamber, celebrating the experience of being a HBOT 'diver' and acknowledging the willingness of patients to accept burdens as a minor inconvenience whilst supporting them to minimise any impact. This has the potential to improve the lived experience of patients undertaking this unique health care treatment.

Findings from two sites in different countries have highlighted many common experiences for patients. It is recommended that the patient experience of HBOT be further

explored at multiple geographical sites with varied chamber styles and include a wide range of patient cohorts.

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Short communication

Provision of emergency hyperbaric oxygen treatment for a patient during the COVID-19 pandemic

Michelle L Lim[1], Soo J Kim[2], Meng K Tan[2], Kim H Lim[2], Hooi G See[2]

[1] *Department of Surgical Intensive Care, Division of Anaesthesiology and Perioperative Medicine, Singapore General Hospital, Singapore*
[2] *Hyperbaric and Diving Medicine Centre, Singapore General Hospital, Singapore*

Corresponding author: *Dr Michelle Lim, Department of Surgical Intensive Care, Division of Anaesthesiology and Perioperative Medicine, Singapore General Hospital, 1 Outram Road Singapore 169608, Singapore*
michelle.leanne.lim@singhealth.com.sg

Key words
Carbon monoxide; Infectious diseases; Intensive care medicine; Logistics

Abstract

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The experience of managing a critically ill severe carbon monoxide poisoning patient suspected of possibly also suffering COVID-19 and requiring emergency hyperbaric oxygen treatment is described. Strategies used to minimise infection risk, modifications to practice and lessons learnt are described. All aerosol generating procedures such as endotracheal tube manipulation and suctioning should be undertaken in a negative pressure room. In the absence of in-chamber aerosol generating procedures, an intubated patient presents less risk than that of a non-intubated, symptomatically coughing patient. Strict infection control practices, contact precautions, hospital workflows and teamwork are required for the successful HBOT administration to an intubated COVID-19 suspect patient.

Introduction

In March 2020, the World Health Organization (WHO) declared a novel coronavirus disease SARS-CoV-2 (COVID-19) a global pandemic.[1] During the pandemic, the Singapore General Hospital (SGH) Hyperbaric and Diving Medicine Centre (HDMC) had a policy on providing hyperbaric oxygen treatment (HBOT) as an intervention for COVID-19 confluent with major hyperbaric committees worldwide.[2–4] The centre did not treat any COVID-19 positive patients. However, as in other clinical services, there was always the possibility that a COVID-19 infected patient, or a patient suspected at high risk of being infected, might require HBOT for another indication. This short communication describes the emergency management of a carbon monoxide poisoning patient whose COVID-19 status was uncertain prior to treatment. The report takes the form of an evolving clinical case description. The patient provided written consent for publication of case information.

Case description

HDMC received this case on 19 June 2020. A 21 year-old female was found unconscious in an enclosed room with burning charcoal after a suicide attempt. She was last seen 14 hours before that. She was conveyed via ambulance to a district hospital and was found to have a carboxyhemoglobin (COHb) level of 23%. She also sustained third degree 5% total body surface area burns on her limbs. She was intubated for airway protection as she was drowsy. In view of the severity of the CO poisoning (high COHb level, altered mental state, and end organ involvement), HDMC was contacted. The patient had a single episode of fever at 38.1 degrees Celsius on arrival at the district hospital emergency department (ED) and one of her family members was an asymptomatic frontline worker involved in screening travellers in the airport.

PREVENTING TRANSMISSION OF COVID-19: PATIENT SCREENING

All patients entering SGH are assessed for COVID-19 risk using temperature monitoring and a standard hospital screening questionnaire, created by the SGH Campus Disease Outbreak Task Force. The questionnaire is based on the Ministry of Health criteria for suspected COVID-19 infections. Patients with respiratory symptoms, and risk factors such as close contact with COVID-19 cases or significant travel history are not allowed into the HDMC for treatment.

INTERHOSPITAL TRANSFER

Inter-hospital transfers are allowed only for cases requiring urgent clinical expertise that is not available in the referring hospital. SGH hospital workflow requires the infectious diseases (ID) physician on-call to perform a COVID-19 risk assessment prior to receiving inter-hospital transfers. Based on the screening questions and investigations such as a chest x-ray, patients are stratified into low, moderate and high risk for COVID-19 infection. Patients with moderate and high risk for infection are admitted to a single or a negative pressure room. A minimum of two negative COVID-19 PCR swab tests done at least 24 hours apart are required for clearance and de-isolation.

Case description, continued

Despite having a normal chest X-ray, this patient was deemed to have a moderate risk for COVID-19 infection as she was febrile, had increased airway secretions and had been intubated. This was a logistical challenge as she required an interhospital transfer, and HBOT, which was not previously offered to COVID-19 suspect patients.

IMPLEMENTING EMERGENCY HBOT WORKFLOW FOR A COVID-19 SUSPECT CASE

The ID physician visited the HDMC to understand the HBOT process and work area to identify areas of increased risks and ways to mitigate it. Once both the ID and HDMC teams were satisfied that the HDMC staff would not be exposed to an unnecessarily high risk of infection, HBOT was offered to the patient.

COORDINATION AND PATIENT TRANSFER WITHIN THE HOSPITAL

Pre-planning of the transport route was done, and the shortest route with minimal human traffic was selected. Transfer commenced only when the receiving location and staff were ready, and the transfer route cordoned off with help from the security department. To minimise risk of cross-infections to other patients, the patient was treated in isolation.

STAFF PROTECTION

Frontline healthcare staff involved in high risk aerosol generating procedures (AGP) such as intubations, endotracheal tube manipulations (i.e., replacing air in the ETT cuff with saline), procedures involving ventilator disconnections, and open suctioning had undergone personal protective equipment (PPE) training and competency tests on the use of National Institute of Occupational Safety and Health (NIOSH)-certified N95 respirators. All staff working at the HDMC were mask fitted.

During the treatment, staff present within the compound was minimised, and the entrance into the HDMC was locked to prevent unnecessary staff movement. Prominent signage indicating that an infective case treatment was in progress were placed to alert other staff. As the HDMC does not have a negative pressure room, all staff present within the vicinity wore N95 masks for the entire treatment duration.

The hyperbaric physician and in-chamber nurse, who were in direct contact with the patient, wore full PPE[5] – including goggles or face shield, N95 mask, cap, gown and gloves.

IN-CHAMBER STAFF

As this was a high-risk case, an experienced in-chamber nurse accompanied the patient. The in-chamber nurse performed a single breath hold while changing from the N95 mask to the built-in-breathing-system (BIBS) mask for the end of treatment nitrogen off-gassing. In this case, the N95 mask did not impede the staff from performing Valsalva manoeuver. An alternative method which does not require mask removal, such as the voluntary tubal opening technique can be employed for equalisation of the middle ears.

For hospital staff who failed N95 mask fit, SGH stocks alternative systems such as the CleanSpaceR HALO™, and the 3M™Jupiter™ Personal Air Purifying Respirator. Such systems cannot be used in the hyperbaric chamber as they are not pressure tested. They rely on lithium batteries, which pose a fire risk.

Although N95 masks are recommended by WHO, the masks have not been tested under hyperbaric conditions, which may potentially affect the seal and efficacy. Nonetheless, it is worthwhile to remember that the air flow in the chamber is not static. The multiplace chamber utilises a continuous high air flow ventilation system with an optional flushing system to maintain the chamber pressure. Furthermore, the risk of aerosol generation in a patient who is sedated, intubated, and ventilated using a closed system ventilator in the hyperbaric chamber is lower than in an un-intubated patient.

Potential alternatives to the N95 for the in-chamber staff would be personalised air delivery systems via hoods or BIBS.[6] However, the staff movement within the chamber would be restricted by the length of the gas delivery tubing.

Fire safety of the polypropylene PPE gown needs to be taken into account especially in chambers which use 100% oxygen for compression as there is a risk of static electricity generation. In the multiplace chamber which uses air for compression, continuous in-chamber oxygen level monitoring was done to minimise fire risk. The risk of fire from the PPE gown needs to be weighed against the risk of in-chamber staff infection and psychological well-being.

HDMC uses washable shoes for chamber work, thus shoe covers are not required. The shoes were washed in sodium hypochlorite after use. SGH infectious disease protocol requires all HDMC staff in contact with COVID-19 suspect patients to shower within the HDMC compound, and change into clean clothes prior to leaving HDMC.

MANAGEMENT OF A POTENTIALLY INFECTIOUS INTUBATED AND VENTILATED PATIENT FOR HBOT

As HBOT treatments are time critical, the usual HBOT workflow in SGH would be for an intubated patient to be transferred directly from the ambulance bay in the emergency department (ED) to the HDMC for treatment. During the COVID-19 outbreak, the ideal location to review a COVID-19 suspect patient would be in a negative pressure isolation room, which is only available in the intensive care units (ICU). The workflow was modified to transfer the patient from the ambulance bay directly into the burns ICU (BICU), bypassing the ED. In BICU, the patient was stabilised and prepared for HBOT by the hyperbaric physician.

High risk AGPs[7] should be done within the negative pressure ICU room. The patient should be kept deeply sedated or paralysed to prevent coughing. During ventilator change, clamping the ETT with a large artery forceps after full expiration, switching off the ventilator prior to disconnection minimises aerosol generation.[8] In the absence of ETT manipulation, open suctioning or circuit disconnection, the risk of aerosol generation is minimal in a closed ventilator circuit.

To avoid environmental pollution within the HDMC complex, the transport ventilator was changed to the in-chamber ventilator within the hyperbaric chamber. Unless absolutely necessary, ETT manipulation and ETT in-line suctioning was avoided. Ventilator tubing and connections should be tightened to minimise circuit leaks. As per usual hospital practice, expired air from the patient passed through two (2) high efficiency particulate air (HEPA) filters – a low dead space filter (Portex® Thermovent® HEPA Low Deadspace Heat and Moisture Exchange Filter) was placed at the end of the ETT, and the second filter (MAQUET Servo Duo Guard) between the expiratory limb and the machine. An additional HEPA filter (MAQUET Servo Duo Guard) was placed between the ventilator outflow tubing and the chamber outflow port to filter exhaust to minimise environmental contamination within the hyperbaric chamber (Figure 1).

Post-procedure, the HBOT ventilator (Maquet Servo 900C Ventilator, Siemens) parts were soaked in ethanol 70% for one hour according to the manufacturer's instruction, and the external surface of the ventilator was wiped down.

Figure 1
Ventilator set up indicating one low dead space HEPA filter at the end of the ETT, one HEPA filter at the gas inflow to the ventilator and one HEPA filter before the chamber outflow port

Although the usual practice is to rapidly wean off sedation to assess GCS score during the HBOT session, this patient was kept sedated until she was transferred back to BICU to minimise coughing and risk of circuit disconnections.

ENVIRONMENT AND EQUIPMENT

One method to minimise contamination for suspect COVID-19 is to remove unnecessary equipment and to cover non-removable objects or surfaces with plastic sheets or wraps. As part of our usual practice, only essential items are kept in the chamber.

It is important to ensure that chemicals used for disinfection are safe to use on acrylic surfaces - this should be checked with the chamber manufacturer.[9] In SGH, the chamber and equipment are wiped down with Mikrozid® sensitive wipes (Schülke & Mayr GmbH, Vienna, Austria). As an added precaution, all surfaces within the hyperbaric chamber are cleaned with sodium hypochlorite 1000PPM, with a contact time of five minutes on top of regular cleaning. To prevent prolonged contact of sodium hypochlorite with the metal surfaces which could cause rusting, a second wipe down with water after 5 minutes was done.

In SGH wards, ultraviolet (UV) light irradiation is used for terminal cleaning. It was not used in this case as UV light sterilisation is only effective for surface areas exposed to the light and may not be effective in our multiplace chamber which has fixed seating and thus many shadow-generating obstructions. It is important to cover UV sensitive acrylic viewports if UV light is used.[10] Simple rubber gloves have been used for this purpose.[11]

Footnote: * Appendix1 is available on DHM Journal's website: https://www.dhmjournal.com/index.php/journals?id=84

Case description continued

The patient underwent two HBOT sessions within 24 hours, and the first HBOT session was started within six hours of presentation. Two samples of endotracheal aspirates, each collected 24 hours apart were sent for COVID-19 PCR testing. Both samples tested negative for COVID-19. The patient subsequently received surgical treatment for her burns and she recovered well.

Lesson learned and future improvements

The successful management of this case could be attributed to three main factors. First, hospital wide infectious control precautions/practices were in place, and had become a part of the daily routine for all staff since the start of the pandemic. Second, the ID physician visited the HDMC personally to walk through the HBOT process and helped to identify potential issues and ways to minimise infection risk. Third, the experienced HDMC team who were trained and comfortable in managing critically ill and intubated patients were key in caring for the patient whilst in full PPE.

An area for improvement would be to employ the use of simulation for a dry run prior to the actual case. Simulation was employed for emergency operation cases in SGH major operating theatres when the COVID-19 outbreak started.[8] Although simulation is resource intensive, one of the main benefits is the ability to identify latent threats not previously anticipated, while concurrently training staff. We recommend that simulation training be carried out if manpower permits and expertise is available. Appendix 1* can serve as a guide for units providing emergency HBOT during the COVID-19 pandemic.

Evidence for HBOT for CO poisoning is controversial at best,[12] and during a pandemic we recommend a risk-benefit evaluation for every case. As COVID-19 is expected to remain endemic, continued vigilance and strict infection control measures are required to prevent disease transmission.

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Appendix 1. Suggested workflow for the provision of emergency hyperbaric oxygen treatment during the COVID-19 pandemic

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Case reports

Investigation of a cluster of decompression sickness cases following a high-altitude chamber flight

Nazim Ata[1], Erkan Karaca[1]

[1] *Aeromedical Research and Training Center, Eskisehir, Turkey*

Corresponding author: *Dr Nazim Ata, Ucucu Sagliği Arastirma ve Egitim Merkezi, Eski Hava Hastanesi, Eskisehir, Turkey 26010*
doktornazim@yahoo.com

Key words
Ascent; Aviation; Barometric pressure; Case reports; Hyperbaric oxygen; Hypoxia training

Abstract

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Although relatively safe, hypoxia exposure is a mandatory training requirement for aircrew that carries the risk of decompression sickness (DCS). Usually DCS affects only one individual at a time. Here, a cluster of three simultaneous cases is reported. Since these numbers were well in excess of the usually encountered incidence rate, the purpose of this work was to identify the most likely reasons using the epidemic DCS investigation framework which involves four main considerations: time; place; population; and environment. Based on time and place observations, this cluster clearly falls into the individual-based classification, where the environment is a primary concern. Indeed, equipment analysis allowed us to identify the most likely reason for two out of three cases (perforations in the oro-nasal oxygen masks worn during training). It led to replacement of damaged equipment and modification of teaching to prevent such damage. It is recommended that this investigative template may be used for any future occurrences of DCS in clusters.

Introduction

Hypoxia training in the high-altitude chamber is a part of the physiological training of aircrew, where trainees experience the symptoms of hypoxia and the changes of volume of gas-filled cavities within the body, akin to what can occur during actual flight. The aim of such training is to make them aware of the problem of hypoxia and their respective hypoxic signatures.[1,2] In theory, should an in-flight hypoxic event occur, it may prepare aircrew members to take necessary remedial measures and exercise better control over the aircraft, as required in actual flight conditions.

Although relatively safe, sometimes decompression sickness (DCS) can occur during this training. This is a condition arising from dissolved gases coming out of solution to form bubbles inside the body on depressurisation, which is the case when flying an unpressurised aircraft at altitude. Usually, DCS affects aircrew on an individual basis. This is consistent with the experience of our centre as we only record one or two DCS cases every year, always happening as single case.

Rarely, however, DCS may occur in clusters, affecting more than one person at a time. When four or more individuals are affected, this has been called "*epidemic DCS*".[3] According to Butler, who first coined the term, there must be an exposure compatible with DCS and the incidence of the event must be higher than normal baseline incidence.

This report presents three simultaneous cases, which were evaluated for mild ('Type I') DCS after a high-altitude chamber exposure. Although, this does not correspond to the exact definition of DCS epidemic, we decided to investigate this cluster within the epidemic DCS investigation framework. Indeed, by definition an epidemic of a disease is an outbreak that exceeds the normal incidence of that disease, which is the case here. Therefore, the purpose of this work was to search for and identify the most likely reasons for those cases through a well-coordinated, thorough and systematic approach.

Cases

All cases discussed in this report gave written consent to use of their medical data for this purpose.

Hypoxia training is a standardised procedure (Table 1). A group of trainees (typically 10) are exposed to a pressure equivalent of 25,000 feet above sea level. Training starts with ascent to altitude and finishes with descent to ground level. An inside observer/instructor officer (IO), acting as safety officer, also participated in the chamber flight. All participants underwent a medical examination, including

Table 1
Standard altitude hypoxia training profile

Phase	Procedure	Altitude (ft)	Ascent and descent rate (ft·min^{-1})	Pressure (mmHg)	Pressure (kPa)	Mask position	Inspired oxygen %
1	Denitrogenation	2,000	–	706.6	94.2	On	100
2	Sinus check	2,000–7,500–2,000	5,000	–	–	On	100
3	Ascent	2,000–25,000	5,000	–	–	On	100
4	Hypoxia training	25,000	–	288.6	38.4	Off	20.9 (Air)
5	Descent	25,000–22,000	5,000	–	–	On	100
6	Descent	22,000–2,000	2,500	–	–	On	100

Table 2
Epidemic DCS investigation framework. IO – instructor officer

	Focus	Factors	Sub-factor
1	Time	NA	–
2	Place (location)	NA	–
3	Population (person)	Trainee and IO	Medical checks DCS risk factors
4	Environment	Training programme	Profile (alterations of procedures)
		Equipment	Maintenance (failure) Oxygen system

a detailed otorhinolaryngology (ear, nose, and throat) examination the day before the altitude chamber training. They were further checked by the flight surgeon in relation to current health status, particularly with respect to the upper respiratory tract, just before entering the chamber. Following the mandatory safety briefing, the training began.

During the descent three individuals developed symptoms: the IO (aged 31) experienced right wrist pain; one of the trainees (aged 24), whose time of useful consciousness during hypoxia was 201 seconds, developed left wrist pain and another trainee (aged 29),whose time of useful consciousness was 224 seconds, noted right knee pain. The flight surgeon overseeing the hypoxia training examined each individual and diagnosed Type I DCS. The IO and both trainees, now patients, were treated in accordance with US Navy Treatment Table 5 within 15 minutes of reaching ground level. All three patients recovered completely after treatment and were completely symptom free.

Follow-up of all the three affected personnel did not reveal any sequelae of DCS. The trainees were routed back to their units and the IO resumed his attendance at altitude chamber training.

Investigation

In our centre, approximately 1,500 trainees are exposed to a total of 200 altitude chamber flights per year, with a DCS incidence of 0.067%. Here, there were three cases from one exposure of a total of 11 individuals (27%). Symptoms were noticed following an altitude exposure plausibly consistent with causing DCS. Subsequent symptom resolution with hyperbaric oxygen reinforced the diagnosis. Since these numbers were well in excess of the usually encountered altitude chamber DCS incidence rate, this event was considered a cluster that needed proper investigation. It was considered that the epidemic DCS investigation methodology was the right approach and would, in all probability, reveal the cause thereby enabling the authors to prevent recurrences.

Epidemic DCS falls into two classes, individual-based (Epi-I) and population-based (Epi-P). Epi-I is defined as four or more DCS patients as a result of a solitary exposure, and Epi-P is defined as four or more DCS patients over an extended time frame.[3,4] In classical infectious disease outbreak/epidemic investigations, the time, place, population and environment must be examined. As pointed out by Butler, this methodology is not altogether useful for epidemic DCS, in particular Epi-I.[3] Therefore, a new template was created by adapting the classical framework to investigate our cluster of DCS. This template is named 'Epidemic DCS Investigation Framework' (Table 2) and includes four main foci: time; place; population; and environment. In this cluster of DCS cases, the most relevant foci were population and environment. 'Population' relates to the affected patients and factors such as medical status

Figure 1
Oronasal mask used during hypoxia training. The tear in the mask can be seen in the right nasal finger recess

Figure 2
Close up of the tear in the right nasal finger recess in one of the training masks

and DCS risk factors, including hydration, in-chamber exercise, injury, fatigue, obesity, lack of pre-oxygenation and pre-chamber exercise. 'Environment' relates to equipment (maintenance and oxygen systems [hose, mask, breathing gas etc.]) and the training program (flight profile).

After creating the Epidemic DCS Investigation Framework the investigation was initiated according to this template.

Time and place: In a situation like this, where a cluster of DCS cases occurs with one altitude exposure, time and location are not generally helpful. This was the situation with the present cluster where all the cases occurred at a single location. However, based on these observations, our cluster clearly falls into the Epi-I classification, where the environment is generally a primary concern.[3]

Population: Analysis of the susceptible population often reveals some tell-tale evidence. The various DCS-specific risk factors, including hydration, in-chamber exercise, injury, fatigue, obesity, lack of pre-oxygenation and pre-chamber exercise were looked into.[5–11] However, nothing significant was discovered in these physically well-conditioned young men. Both trainees underwent a thorough medical examination the day before the training and the IO, an instructor with 14 years' experience in the altitude chamber, passed his yearly medical examination. None of them reported any medical problem before the training.

Environment: Although no procedural discrepancies before, during or after the altitude chamber training were identified, the operational procedures were reviewed in detail. This review revealed no untoward practices. Trainees were under close supervision of two outside operators and one IO. So it was not possible to remove their masks in the first (denitrogenation) phase (Table 1).

The focus then fell on equipment issues. Although, periodic maintenance requirements were met, close scrutiny of the equipment revealed holes (Figure 1 and Figure 2) in the trainees' masks (MBU-12/P Oxygen Mask). Further investigation revealed that during their initial training both trainees had performed Valsalva manoeuvers and pinched their noses by pushing their fingernails into their mask, leading to tears in the mask material. When discussed with the IO, it was learned that he checked all masks before the training and there were no problems with the masks. However, during phase 1, the Valsalva manoeuvre was used. The demand valve and hose were also checked with no failure found. Analysis of the respired gases occurs before they are breathed in the chamber. If the oxygen level is less than 99.8% or any toxic gas detected, the system is alarmed. The system didn't give any alarm the day of the event. It was felt that these tears were likely an important causal factor in the trainee's DCS. Inward leakage of ambient chamber air through the tears during inhalation may have diluted the oxygen content of the inhaled gas, and resulted in insufficient negative pressure inside the mask to fully trigger delivery of 100% oxygen from the demand valve, thus compromising denitrogenation.

Two different types of masks are used in our centre. These holes were formed in only one type of masks. These masks were replaced with newer ones of a sturdier design. In addition, Valsalva manoeuvre training in training lectures was revised. At the same time, the safety teams were advised to be more cautious while inspecting masks, hoses, and related equipment before commencement of altitude chamber training.

Since the replacement of the defective masks and revision of training protocols, no instances of DCS, above the incidence rate, have occurred during our high-altitude chamber

exposures. As to the IO, the authors were unable to discern a specific aetiology for his DCS.

Conclusion

Although this cluster of DCS cases did not meet the definition of epidemic DCS, using the epidemic DCS investigative framework allowed identification of the most likely reason for two out of three cases. It also led to replacement of damaged equipment and improvement of training. It is recommended that this investigative template may be used for any cluster of DCS cases encountered henceforth.

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Hyperbaric oxygen treatment: Results in seven patients with severe bacterial postoperative central nervous system infections and refractory mucormycosis

Pedro Valente Aguiar[1,2], Bruno Carvalho[1,2], Pedro Monteiro[1,2], Paulo Linhares[1,2,3], Óscar Camacho[4], Rui Vaz[1,2,3]

[1] *Neurosurgery Department, Centro Hospitalar Universitário São João, Oporto, Portugal*
[2] *Faculty of Medicine, Oporto University, Oporto, Portugal*
[3] *Neurosciences Centre, Hospital CUF, Oporto, Portugal*
[4] *Hyperbaric Medical Unit, Unidade Local de Saúde de Matosinhos, Portugal*

Corresponding author: *Dr Pedro D Valente Aguiar, Department of Neurosurgery, Centro Hospitalar Universitário São João, Alameda Professor Hernâni Monteiro, 4200-319, Porto, Portugal*
pedrovalenteaguiar@protonmail.com

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Brain; Mucormycosis; Neurosurgery; Refractory infections; Spine

Abstract
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Introduction: Resistant bacterial infections following brain and spine surgery and spontaneous mucormycosis with central nervous system (CNS) involvement represent a serious treatment challenge and more efficient therapeutic approaches ought to be considered. Hyperbaric oxygen treatment (HBOT) has shown promise as a complementary therapy. This case series evaluated whether HBOT contributed to infection resolution in seven patients with refractory CNS infectious conditions.
Methods: Clinical results for seven patients referred for HBOT between 2010 to 2018 to treat refractory postoperative brain and spine infections or spontaneously developing mucormycosis were retrospectively analysed. The patients' clinical files and follow-up consultations were reviewed to assess evolution and outcome.
Results: Seven patients were referred with a median age of 56 years. The median follow-up was 20 months. Four patients had postoperative infections and three had rhino-orbital-cerebral mucormycosis (ROCM). HBOT was used as an adjunctive treatment to antimicrobial therapy in all patients. Prior to HBOT, all patients had undergone an average of four operations due to infection refractoriness and had completed an average of five months of antimicrobial therapy. After HBOT, infection resolution was obtained in six patients without additional operations, while one patient with ROCM stopped HBOT after the third session due to intolerance. Three patients stopped antimicrobial therapy while four were maintained on prophylactic treatment.
Conclusions: Infection resolution was reached in the six patients that completed HBOT as prescribed. HBOT may serve as an effective complementary treatment in CNS refractory postoperative and spontaneous infections.

Introduction

Resistant infections remain a challenge to neurosurgical and neurological care. The use of less conventional techniques can be an adjuvant option to consider when standard treatments are ineffective. Albeit seldom used in complex cases, there is evidence to support hyperbaric oxygen treatment (HBOT) as a complementary therapy.

The 2016 European Committee for Hyperbaric Medicine (ECHM) guidelines[1] strongly recommend HBOT use in anaerobic or mixed bacterial infections and the 2013 European Society for Clinical Microbiology and Infectious Diseases (ESCMID) and European Confederation of Medical Mycology (ECMM)[2] joint guidelines for the diagnosis and management of mucormycosis express marginal support for HBOT in refractory infections.

Previous studies have also shown HBOT can be of value in complicated brain and spine infections.[3–6]

HBOT provides beneficial pathophysiological changes in the context of infection and inflammation, such as the correction of tissue and cellular hypoxia and the enhancement of polymorphonuclear leukocyte activity.[7,8] Concomitantly, a release of anti-inflammatory cytokines occurs, microvascular perfusion is enhanced and wound healing improves. Hyperbaric oxygen has also bacteriostatic and bactericidal properties.[8–10]

Larsson et al. reported fewer reinterventions for infection control following HBOT in complicated postoperative neurosurgical infections.[6] However, evidence remains scarce regarding hyperbaric oxygen impact on chronic central nervous system (CNS) infection morbidity. Therefore, the present study evaluated the impact of a complementary therapy that has been somewhat overlooked by the medical community, despite its potential effectiveness and safety, in achieving infection resolution in four patients with complicated postoperative infections and three patients with rhino-orbital-cerebral mucormycosis (ROCM) that had proved refractory to conventional treatment.

Methods

This study was approved by the institutional Ethics Committee of the Centro Hospitalar Universitário São João.

The patient database of the Hyperbaric Medical Unit (HMU) at Pedro Hispano Hospital (PHH) in Matosinhos, Portugal was accessed to identify patients referred by the Department of Neurosciences and Mental Health of Centro Hospitalar Universitário São João (CHUSJ) since 2006 (when the HMU started its activity) and those cases in which the hyperbaric department had acted as consultant due to CNS involvement.

Demographic and clinical data were collected including: age and gender; past medical history; active disease; HBOT clinical indication; number of HBOT sessions; number of operations before and after HBOT; presence or absence of surgical heterologous material; microbiologic results; and antibiotic treatment. Patients and physicians were contacted to assess clinical improvement. Serum inflammatory markers, temperature charts, computed tomography (CT) and magnetic resonance imaging (MRI) scans were evaluated to gauge whether infection resolution had been achieved.

The primary endpoint was infection resolution. Infection resolution was defined by clinical improvement, normalisation of elevated inflammatory markers when applicable, and by the lack of active infection on MRI imaging. All cases of infection resolution were confirmed by infectious diseases specialists.

HYPERBARIC OXYGEN THERAPY

All patients were treated at the multiplace hyperbaric chamber of the HMU of Pedro Hispano Hospital. The sessions' duration was 90 minutes and took place on consecutive weekdays. Treatment was given at 243.1 kPa (2.4 atmospheres absolute) pressure breathing 100% oxygen.

Before treatment all patients were examined by an anaesthesiologist to rule out relative contraindications such as Eustachian tube dysfunction, uncontrolled epilepsy and pulmonary conditions like pneumothorax.

ANTIBIOTIC AND ANTIFUNGAL TREATMENT

Prior to and throughout HBOT, patients with infections were treated with antibiotics and/or antifungal therapy based on the susceptibility of the isolated agents. When no microbial pathogen could be isolated patients were treated empirically taking into account the most likely pathogen and local resistance patterns. The antimicrobial treatment was optimised by infectious diseases specialists in all cases.

Results

A total of seven patients (median age 56) with relevant infectious pathology were consecutively treated with HBOT from 2011 to 2018 inclusive. The median follow-up was 20 months. Key patient and pathology characteristics are provided in Table 1 and the history of surgical intervention and selected biochemistry results in Table 2.

Patients one to four had refractory postoperative infections following neurosurgical intervention at our department while patients five to seven had complex ROCM that had not been controlled with antifungal treatment.

Previous to HBOT, all patients had undergone an average of four operations and had completed an average of five months of antimicrobial therapy indicating the refractory nature of the infections. Addition of HBOT was associated with infection resolution in six patients without additional subsequent operations, while one patient with ROCM stopped HBOT after the third session due to intolerance. Three patients stopped all antimicrobial therapy while four were maintained on prophylactic treatment.

CASE SUMMARIES

Patient 1

A 65-year-old female patient with no relevant past medical history underwent craniotomy for drainage of a chronic subdural hematoma.

One month later she underwent surgical site debridement due to infection and two years later she underwent bone flap, cranioplasty and surgical drainage of a subdural empyema. Because of infection persistence she underwent two additional operations for refractory empyema drainage and superficial wound infection revision and cleansing.

Before HBOT initiation she had completed five months of multiple antibiotic regimens while sulfamethoxazole and trimethoprim were added before HBOT. The patient completed 60 sessions of HBOT and has remained on prophylactical sulfamethoxazole and trimethoprim during the follow-up period of 20 months.

Following HBOT complete wound closure and infection resolution were obtained.

Table 1

Relevant clinical data for seven patients treated with HBOT for refractory CNS infections. F – female; m – months; M – male; MR – multidrug resistant; MRSA – methicillin-resistant *Staphyloccocus aureus*, MSSA – methicillin-sensitive *Staphyloccocus aureus*; NSES – nasosinusal endoscopic surgery; PLF – posterior lumbar fusion; RA – rheumatoid arthritis; ROCM – rhino-orbital-cerebral mucormycosis; T1DM – type 1 diabetes mellitis; T2DM – type 2 diabetes mellitus, TLIF – transforaminal lumbar interbody fusion; TPF – transpedicular fixation

Case	Sex	Age	Condition	Past history	Antibiotic treatment pre-HBOT	Antimicrobials during HBOT	HBO#	Microbe	Operations prior to HBOT
1	F	65	Subdural empyema	Chronic hypochromic microcytic anemia	5 m vancomycin, meropenem, metronidazole, ceftriaxonesulfamethoxazole + trimethoprim	Sulfamethoxazole + Trimethoprim	60	Could not isolate	4
2	M	56	Brain abscess	Glioblastoma (Grade IV)	6 m vancomycin, ceftazidime, flucloxacillin, ceftriaxone, cefepime and meropenem	Ceftriaxone	40	*Enterobacter aerogenes* (surgical site)	5
3	F	51	L4 – L5 osteomyelitis + soft tissue infection + paravertebral muscles abscesses	T2DM + RA Lumbar TLIF, PLF, TPF	5 m levofloxacin, ceftazidime, vancomycin, ciprofloxacin, ceftriaxone and daptomycin	Flucloxacillin Rifampicin	40	*MSSA* (surgical site) *MRSA* (surgical site)	4
4	M	69	Sepsis and meningitis + L4-5 osteomyelitis and spondylodiscitis	T2DM	3 m meropenem, vancomycin, linezolid and rifampycin	Vancomycin	40	*MRSA* (blood culture) *MSSA* (surgical site)	2
5	M	73	ROCM	Diffuse large B cell lymphoma	1 m liposomal amphotericin B, meropenem, vancomycin and posaconazole	Vancomycin Posaconazole	40	All surgical site *Aspergillus fumigates Mucor sp.,* MRSA, *MR Corynebacterium Tuberculostearicum*	2
6	F	20	ROCM	T1DM	3 m ceftriaxone, clindamycin, meropenem, liposomal amphotericin B, ceftriaxone, ampicillin, metronidazole, cefepime and posaconazole	Liposomal amphotericin B Ceftriaxone, Cefepime Isavuconazole	40	MSSA *Serratia marcensens MR – S. epidermidis*	5
7	M	14	ROCM	Anaplastic T-cell lymphoma	6 m vancomycin, posaconazole, linezolid and meropenem	Liposomal Amphotericin B and isavuconazole	3	*Rhizopus sp.* (Left NSES) *Staph. warneri* (Surgical site dura)	3

Table 2

Case surgical timelines and procedures and pre- post-HBOT C-reactive protein and white cell counts. CRP – C-reactive protein; CSDH – chronic subdural hematoma; GBM – glioblastoma; L – lumbar; NSES – nasosinusal endoscopic surgery; PEEK – polyetheretherketone; PLF – posterior lumbar fusion; ROCM – rhino-orbital-cerebral mucormycosis; TLIF – transforaminal lumbar interbody fusion

Case	Initial surgery/ diagnosis	1st operation	2nd operation	3rd operation	4th operation	5th operation	Pre-HBOT Post-HBOT CRP / WBC $mg{\cdot}L^{-1}$ / L^{-1}
1	April 2016 CSDH drainage with craniotomy	May 2016 Surgical site debridement	May 2018 Empyema drainage	July 2018 Empyema drainage	September 2018 Surgical debridement	–	9.9 / 5,820 2.2 / 7,000
2	February 2015 Right frontal GBM removal	June 2015 Reopening of craniotomy with abscess drainage	July 2015 Reopening of craniotomy with surgical cleansing of right frontal abscess	September 2015 Surgical site cleansing and debridement	December 2015 Surgical right frontal abscess drainage	February 2016 Surgical site cleansing	3.0 / 4,900 8.2 / 5,300
3	August 2011 L4-5 left synovial cyst excision + L5 laminectomy	September 2011 Surgical subcutaneous and subfascial cleaning	November 2011 L2-4 PEEK bars removal and L2-3 left screws removal TLIF L4-5, PLF L5-S1	January 2012 Purulent epidural hematoma drainage Subfascial abscesses drainage	February 2012 Infected seroma drainage and L5 epidural cyst removal	–	101.2 / 7,740 30 / 8,850
4	January 2012 TLIF L4 -5	February 2012 Subcutaneous and paravertebral abscesses drainage	July 2012 Surgical site cleansing with paravertebral muscles abscesses drainage and cage removal	–	–	–	77.7 / 8,290 28.8 / 7,260
5	March 2017 ROCM diagnosis	March 2017 NSES– right unciformectomy and right anterior and posterior ethmoidectomy	April 2017 Orbital apex biopsy and sphenoidotomy	–	–	–	17 / 1,770 2.8 / 6,310
6	August 2018 ROCM diagnosis	August 2018 Right NSES with unciformectomy and right maxillary sinus antrostomy	September 2018 Right NSES Anterior and posterior ethmoidectomy	October 2018 Right orbitotomy for right orbit abscess drainage	October 2018 Right orbitotomy revision with drainage of small orbital abscess	October 2018 Right orbit exenteration	18.8 / 4,840 2.3 / 3,620
7	November 2018 ROCM diagnosis	November 2018 Left NSES with ethmoidectomy and sphenoidectomy	November 2018 Microsurgical removal of intracranial infectious component Left superior and lateral orbitotomy. Left temporal pole abscess drainage	November 2018 Total left orbit exenteration	–	–	NA

Patient 2

A 56-year-old male had a right frontal glioblastoma removed via craniotomy. In the following weeks he started chemoradiotherapy, but treatment had to be stopped once the diagnosis of brain abscess at the surgical site was made (Figure 1).

Subsequently, the patient underwent four operations for abscess drainage and surgical locus cleansing and completed 10 months of varied antibiotics.

He underwent 40 HBOT sessions while maintaining the antibiotic regimen that had previously failed to achieve infection control. Infection resolution was reached following HBOT (Figure 1) and the patient was kept on prophylactic levofloxacin. He remained infection-free during the follow-up period of six months but passed away due to tumour progression.

Patient 3

A 51-year-old female with past history of type II diabetes, rheumatoid arthritis treated with steroid sparing agents and previous operations for spine instrumentation underwent L4-5 left synovial cyst excision and L5 laminectomy.

Due to surgical locus infection and osteomyelitis she was kept on wide spectrum antibiotics for five months and underwent four operations with removal of previous spine instrumentation hardware and new instrumentation performed at adjacent levels. Methicillin-resistant *Staphylococcus aureus* (MRSA) was isolated from the surgical site and rifampicin was introduced prior to HBOT.

Owing to refractory infection she was prescribed hyperbaric oxygen therapy and completed 40 sessions that led to infection resolution. No further surgical or antibiotic treatment was necessary.

Patient 4

A 69-year-old male with past history of type II diabetes mellitus underwent a L4-5 transforaminal lumbar interbody fusion (TLIF). He developed a postoperative infection with formation of paravertebral abscesses and subcutaneous tissue empyema.

He underwent two operations for surgical site cleansing and hardware removal and completed six months of multiple courses of different antibiotics, without successfully achieving infection control.

The patient underwent HBOT while on antibiotics that had previously failed to resolve the infection and upon completing 40 sessions of hyperbaric oxygen, the infection was successfully cured with no further need for surgical operation or antimicrobial treatment.

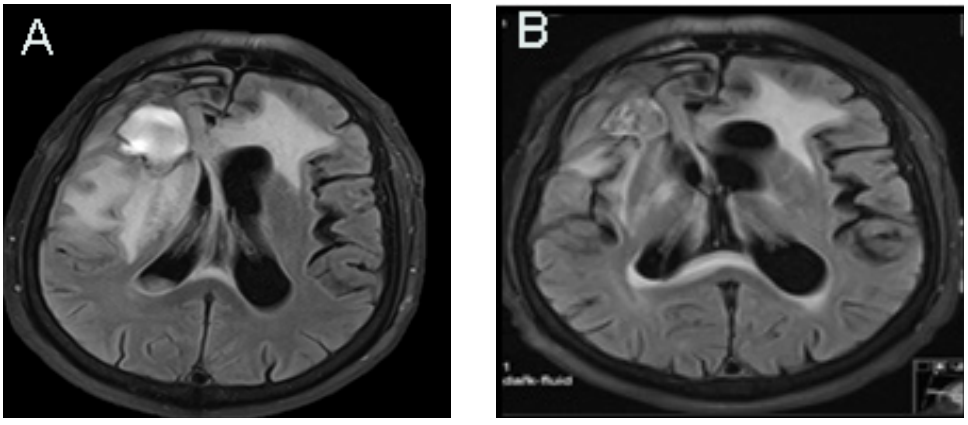
Patient 5

A 73-year-old male with a year long history of diffuse large B-cell lymphoma developed sudden onset of right-sided vision loss, extreme ocular pain and proptosis. Orbital and brain MRI demonstrated a diffuse infiltrate of the right optic nerve, sphenoid and ethmoidal sinuses and discreet cerebral invasion (Figure 2). Endonasal endoscopic biopsy confirmed the diagnosis of mucormycosis with isolation of *Aspergillus fumigates* and *Mucor spp*.

The patient underwent two operations for infection control and had completed one month of antifungal and antibiotic treatment due to bacterial superinfection prior to HBOT initiation.

Infection resolution was obtained after 40 HBOT exposures and the patient maintained prophylactic antifungal therapy with oral posaconazole.

Figure 1
Patient 2. Brain abscess at glioblastoma excision site. A – final MRI before HBOT; B – first MRI post HBOT (FLAIR sequence)



Patient 6

A 20-year-old female with poorly controlled type I diabetes mellitus presented with a two week history of right sided facial pain, hypoesthesia, oedema and purulent drainage from the right superior dental arch, with right ocular pain and ptosis. Brain and orbital MRI demonstrated a diffuse infiltrate arising from the paranasal sinuses with ocular and cavernous sinus invasion on the right side.

She had urgent surgery with nasosinusal endoscopic unciformectomy and right maxillary sinus antrostomy for diagnostic and therapeutic purposes. Due to the refractory infection pattern the patient underwent four subsequent operations, including right orbital exenteration.

Polymerase chain reaction (PCR) was run on a surgical microbial isolate that was positive for *Mucor spp* and, due to suspected bone osteomyelitis, she was treated with antibiotics alongside multiple antifungal medication during a three-month period.

The patient completed 40 HBOT sessions and was considered infection-free upon treatment completion. She was kept on prophylactic isavuconazole for 12 months.

Patient 7

A 14-year-old male with past history of anaplastic T-cell lymphoma presented with rapid onset of left-sided ophthalmoplegia, complete amaurosis and proptosis. He underwent three operations, including brain abscess drainage due to intracerebral invasion from a rhinosinusal fungal infection that had also invaded the left orbit and later required left ocular exenteration. *Rhizopus spp*. was isolated from the first endoscopic nasosinusal intervention.

After referral for HBOT, the patient could only complete three sessions and sadly was not able to accomplish treatment as prescribed due to nausea, vomiting and generalised discomfort while in the hyperbaric chamber.

Up to the end of the current follow-up period of 20 months, the patient had undergone two further operations for encephalocele and cerebrospinal fluid (CSF) fistula correction and remained on amphotericin B and isavuconazole for ROCM treatment.

Discussion

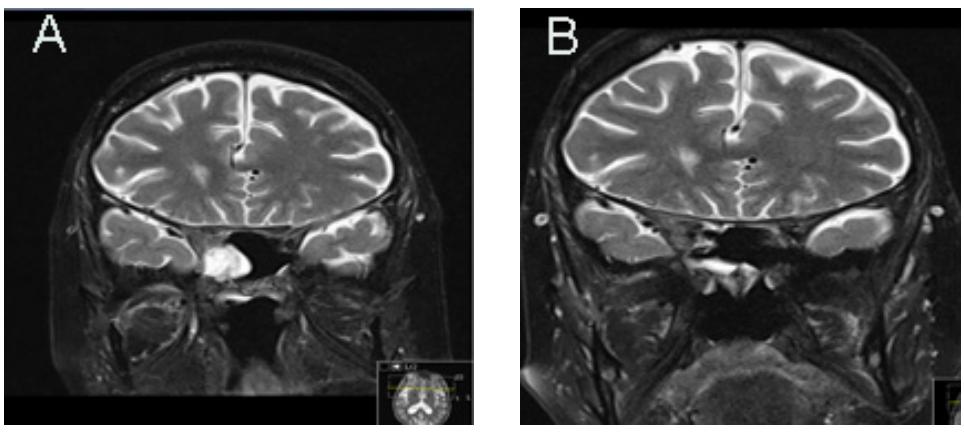
The aim of this case series was to evaluate the impact of HBOT in resolving complex refractory postoperative or spontaneous infections with CNS involvement. The cohort is small and lacks a control group; therefore, caution is needed when interpreting the results. However, with resistant infections following neurosurgical interventions and mucormycosis with CNS involvement representing rare events and thus rendering randomised controlled trials difficult to undertake, case series of this nature are likely to provide the best evidence for potential efficacy of HBOT.

As previously mentioned, there are multiple potentially relevant physiological effects of HBOT. In patients with a compromised immune system, HBOT may enhance elements of immune system activity and also lead to improvement in tissue oxygenation, wound healing and neovascularization.[7,10,11] Hypoxia leads to deficient neutrophil activity and by inducing hyperoxia, HBOT seems to optimise neutrophil antimicrobial activity through enhancing their release of reactive oxygen species.[7,11] In addition, animal studies have shown hyperbaric oxygen to dampen inflammation through cytokine downregulation. HBOT also seems to increase the efficacy of certain antimicrobial agents.[11–15]

Notwithstanding its relative safety, HBOT is not devoid of shortcomings. Besides side effects that include middle ear barotrauma, transient visual acuity changes and pulmonary

Figure 2

Patient 5. Mucormycosis in right sphenoid sinus. A – MRI taken a few days after starting HBOT; B – first MRI post HBOT (T2 sequence)



oxygen toxicity[16] it may be burdensome to obtain for patients whose local hospital or clinic does not possess a hyperbaric chamber. In this series HBOT proved safe and tolerable to all but patient seven. None of the other patients reported symptoms from HBOT.

POSTOPERATIVE INFECTIONS

The four postoperative infection patients reported here were cured after completing HBOT despite long histories of being refractory to multiple interventions. These results are similar to those previously reported in the literature[3–6] and were supportive of the ECHM type I recommendation for HBOT use in anaerobic and mixed anaerobic infections.

Larsson et al.[6] evaluated the results of HBOT both in post-craniotomy and post-spine instrumentation surgery infection with 35 out of a total of 38 patients achieving infection resolution and 23 being able to reach the primary goal of avoiding reoperation for bone flap or spine fixation material removal. Similarly, Bartek et al.[4] set out to assess the efficacy of adjuvant HBOT in resolving deep brain stimulation (DBS) hardware related infections and avoiding material extraction. All 12 patients were cured of their infection and 10 could keep their implants. In a retrospective analysis of two groups of brain abscess patients Bartek et al.[5] showed that those treated with HBOT in addition to surgery and antibiotics had fewer recurrences when compared to surgery and antibiotics alone. Finally, in a series of six patients with spine osteomyelitis treated with HBOT Ahmed et al.[3] reported infection control in one of two patients with spontaneous infection and in four patients with previous spine surgery. All six patients had risk factors for poor infection control.

In complicated postoperative and spontaneous infections, HBOT may serve as an efficient supplementary treatment. Identifying patients with risk factors for developing refractory infections, who may benefit from HBOT, may contribute not only to an earlier cure but also lead to a reduction of antibiotic burden and avoidance of repeated operations, with benefits both to patient well-being and in terms of cost reduction. We believe identifying the correct timing of HBOT application in such complex infections should also be the focus of future studies.

MUCORMYCOSIS

Mucormycosis mostly occurs in immunosuppressed patients, and has high mortality despite antifungal drugs. It follows that alternative supplemental treatments should be sought. HBOT stops fungal growth *in vitro*, augments the efficacy of amphotericin B and through hypoxia reversal and angiogenesis helps to revert the highly hypoxic and hypoperfused infection locus environment. Nevertheless, in spite of a solid pathophysiological case supporting its application[17–21] HBOT use in mucormycosis has been infrequent. The present series, however, adds to a growing number of positive results[17,22–24] that highlight the complementary potential of HBOT in this highly complex infection.

A previous study reported two cases of refractory mucormycosis in diabetic patients that were cured once HBOT was added to the therapeutic armamentarium.[17] Another presented a successful case of mucormycosis control once HBOT was initiated in a child with B-cell precursor acute lymphoblastic leukaemia (ALL).[22] A 60% survival rate in mucormycosis has been reported when HBOT complemented surgical debridement and amphotericin B therapy,[23] representing a significant survival increment when compared to surgical debridement and antifungal therapy alone. Finally, a review of 28 cases of mucormycosis secondary to various immunosupressive states treated with HBOT reported a significant survival benefit for diabetic patients.[24]

Two of three patients in the present series were cured after the completion of HBOT and remarkably patient number six has ceased prophylactic antibiotic treatment. Unfortunately, patient seven could not complete treatment as initially prescribed and has thus far not been able to scale down treatment.

Taking into account the aforementioned results, an earlier introduction of HBOT in mucormycosis treatment might prove beneficial by potentially avoiding the devastating effects of this infection, especially when there is CNS involvement. As recommended for post-op infections, emphasis on the optimal timing for HBOT initiation should be a focus of future studies.

Conclusion

Seven consecutive cases of complex refractory infections with CNS involvement have been reported, of which six were successfully resolved after introduction of HBOT. Previous to HBOT these patients had unsuccessfully undergone prolonged antimicrobial therapy and multiple operations for infection control. After HBOT further reinterventions were not needed and antimicrobial therapy was scaled down or stopped. Moreover, three out of seven patients who achieved infection resolution did not require alteration of their antimicrobial treatment during HBOT, further suggesting hyperbaric oxygen therapy had a positive impact on disease control.

Nonetheless, prospective randomised controlled trials or larger case series are needed to consolidate our findings and more emphasis should be directed towards establishing the correct timing of HBOT initiation in patients with refractory post-operative or spontaneous infections.

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Saturation diver fatality due to hydrogen sulphide while working on a subsea pipe line

Ajit C Kulkarni[1]

[1] *Hyperbaric Solutions, Mumbai, India*

Corresponding author: *Dr Ajit C Kulkarni, Hyperbaric Solutions, 3 A, Siddhivinayak Chambers, Bandra East, Mumbai 400051, India*
drackulkarni@gmail.com

Key words
Gas solubility; Hydrogen sulphide; Hyperbaric oxygen; Pulmonary oedema; Saturation diving

Abstract

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In the offshore oil industry, Multipurpose Support Vessels with extensive diving capability are used for inspection, maintenance and repair of subsea pipelines. The diving industry has developed systemic safety checks and strict regulatory control after a number of fatal accidents in early years. However, accidents do continue to occur and, when involving divers in the water, are often fatal. Hydrogen sulphide (H_2S), called 'sour gas' in an oil field, is produced by the action of anaerobic bacteria on sulphate containing organic matter. A highly toxic gas, it remains a constant danger for offshore oil industry workers who must remain vigilant. Crude oil and gas produced in these oilfields is called 'sour crude' and pipelines carry this crude with varying content of dissolved H_2S to shore for processing. Divers are routinely called to attend to leaking pipelines and come in contact with this crude. Their hot water suits and umbilical lines are often covered with crude containing dissolved H_2S. There is always a possibility that these may enter and contaminate the bell environment. Such a case leading to fatality is reported here.

Introduction

Saturation divers play a major role in the development and production of an offshore oil field. During the construction and developmental phase of an oil field, they dive extensively from construction and pipe-lay barges often working against time. Once production has commenced, pipelines laid at the bottom of the sea and platforms need constant inspection, maintenance and repair. A number of diving vessels are often deployed in the oil field for this reason. The shallowest 20 metres' seawater (msw) of the platform legs is generally attended to by a surface supplied air diving team. If the depth of the oil field is 100 msw, structures deeper than 20 msw would typically be attended by teams of saturation divers; two in each team at different storage depths called split levels. Upward and downward excursion from these depths is limited to 10–20 msw but the entire length of the platform underwater including cross members can be attended to by the saturation diving teams stored at the appropriate depths. One or two teams are stored at each depth depending on the quantum of work.

Case report

The incident happened on board a Multipurpose Support Vessel (MSV) operating in an offshore oil field. The vessel was a purpose-built MSV with accommodation for 12 divers in saturation and a single three-man bell of 4.5 m^3 volume. The diving system was certified to 300 msw and was well maintained. Although it was a three-man bell, a two-man bell run was the norm followed by the client and the contractor. The MSV was on a long charter for carrying out inspection, maintenance and repair duties for the client oil company. On the eventful day, her crew was informed by the charterers about an oil leak in one of the main 36-inch diameter subsea lines carrying sour crude. The vessel was directed to proceed to the site and carry out pipeline repair on an emergency basis.

The MSV had carried out similar operations on 'sour crude' pipelines and a standard operating procedure (SOP) was in place. Salient features included the following. First, the diver shall wear a disposable coverall over the hot water suit which is to be discarded before entering the bell. Second, the diver helmet and umbilical shall be cleaned properly before it is brought inside the bell. Third, both divers shall be on built in breathing system (BIBS) masks when the bell is launched, and the bell man shall be on BIBS throughout. Standard oro-nasal BIBS were fitted in the bell. Fourth, the bell is to be flushed continuously preventing ingress of toxic gas inside the bell.

The MSV left for the indicated location and on arrival, observed a large oil film and gas bubbles on the sea surface.

The pipeline was at 74 msw depth. The MSV was positioned and bell diving started. The bell was at 65 m depth a short distance away from the pipeline, not directly above it. On reaching the seabed, the diver reported oil and gas gushing out from the pipe line with great force and as a result was unable to approach the leak location for closer inspection. This was immediately reported to the oil company and a request was made to reduce the pressure of product flow in the pipeline.

Once the product flow was reasonably reduced, the diver approached the leaking pipeline to locate the rupture. The diver reported that a big trench had formed in the seabed, gouged by the force of the gushing oil. The probable position of the leak was at the bottom of the pipe. The size of the rupture could not be ascertained. The diver was unable to get closer to the leak position as the seabed all around the trench was fully covered with oil sludge.

On complete stoppage of gas and oil flow and consequent cessation of the leak, the diver approached the location and did a close survey of the leak area and the pipeline. After getting preliminary details, he was asked to return to the bell. The bell man was informed of this.

The bell man assisted the diver to enter the bell and removed his helmet. He also had to remove his BIBS for a short time to assist the diver. They exchanged pleasantries. The diver started cleaning his umbilical after sitting down in bell. He had not put on the BIBS and had not secured his safety harness to an 'eye' in the bell. Shortly after, the bell man stopped reacting to the diving supervisor's instructions, collapsed and laid down on the diver's seat.

Within a few seconds of this, the unfortunate incident occurred; the diver, who was sitting in the bell, fell down all of a sudden. Part of the umbilical was still outside the bell awaiting cleaning and the bell bottom door was not closed. Not having secured the safety harness, he fell into the water and was carried away by the current. The bell man was on BIBS and was still unconscious.

As the bottom door of the bell was open, the bell could not be lifted out of the water. The bell was being flushed continuously.

An emergency situation was declared, and a medical officer (the author) was informed by the MSV owner's representative that his presence on board was required urgently. A helicopter sortie was arranged immediately.

Another MSV working in adjoining field, about 40–50 nautical miles away, was requested to proceed to the area for help as quickly as possible. After reaching the helibase, the author established communication with the diving superintendent of the affected MSV and was appraised of the situation. He suggested to the diver superintendents of both the MSVs that a bell-to-bell rescue was the best option available and that they should start preparing a dive plan accordingly. It was then decided that the medical officer should proceed to the rescue MSV and not the affected MSV. The rescue vessel was still underway when the helicopter landed with the medical officer on board. A job specific safety checklist based on risk analysis was prepared jointly with the diving superintendent. The masters of both vessels had worked out a protocol of operation as regards dynamic positioning reference systems and thruster, hull interaction, wind, current and other considerations.

When the rescue MSV arrived at the dive site a rescue diver and bell man were identified and briefed by dive superintendent. All equipment for the rescue diver and unconscious diver was checked and rechecked. Bell diving was carried out from the rescue MSV and the rescue diver reached the unconscious bell man. He was disrobed and new gear was donned on him. He was wet-transferred to the rescue vessel bell. After the rescue, the rescue diver and bellman were instructed to disrobe completely in the bell and not bring any personal gear into the saturation system trunking and transfer under pressure areas of the rescue MSV to avoid the contamination of the living chambers.

In the meantime, the medical officer accompanied by one diver, with resuscitation drugs and equipment were pressurised to 65 msw on heliox into the rescue MSV's saturation living chambers.

The patient, when received was unconscious. His pulse was 60 per min, regular, slow volume. Blood pressure was 106/64 mmHg. Respiration was shallow. His oral cavity was cleared using a foot operated suction machine and a Guedel airway inserted. An intravenous line was secured and 5% glucose infusion was started. The BIBS supply was connected to an Ambu bag and the diver was ventilated using treatment mix (265 kPa PO_2). After approximately 15 min the patient regained consciousness but was disoriented. After 20 min, the BIBS supply was disconnected and the patient was ventilated using chamber gas containing 50 kPa PO_2 for five minutes. During a second ventilation period with treatment mixture, his condition improved. He would obey verbal commands but was not talking. He was disoriented, finding himself with divers who were not there in the chamber earlier and was surprised to see the medical officer treating him inside the living chamber of another MSV. He was eventually able to take deep breaths and ventilation support using the Ambu bag was discontinued. He was switched over to BIBS and an additional four cycles of 20 min breathing treatment mix was completed with five minute breaks. A total of six cycles of treatment mix was given.

Furosemide 20 mg was injected slowly over a minute through the intravenous line to treat potential pulmonary oedema, and repeated eight hourly. Nitrite solution

was not available. The 5% glucose was followed by 500 ml Ringers lactate. No additional intravenous fluids were administered as the diver had recovered consciousness and oral feeds could be started. Breath sounds were normal. There was no clinical evidence of pulmonary oedema. His BP was 110/68 mm and he had passed urine. Adequate oral fluid intake was ensured. He had no recollection of past events other than the diver locking out of the bell. After a 24-hour hold, standard saturation decompression was started which was uneventful. He remained alert but quiet.

Once he was evacuated ashore, he underwent a thorough medical evaluation. A magnetic resonance imaging brain scan, a high resolution computed tomography scan of the lungs and a complete blood work up was carried out. He was evaluated by a neurologist and a psychiatrist. He was declared fit to dive after a month and resumed his work offshore.

The day after the rescue, the body of the diseased diver floated a few meters away from the vessel and was recovered.

Discussion

The presence of hydrogen sulphide (H_2S) in the bell was the root cause of the accident resulting in the fatality. The permissible exposure limit of H_2S is 15 parts per million (ppm) for 15 min at normal temperature and pressure (NTP). At 10 ppm it has a 'rotten egg' smell but at concentrations above 200 ppm, the olfactory nerve becomes paralysed immediately. At concentrations above 500 ppm often the sense of equilibrium is lost and the affected person can become unconscious. Beyond 1000 ppm death is almost instantaneous.[1]

H_2S is transferred easily across the alveoli into blood where it affects cytochrome oxidase causing cellular anoxia and oxygen transport by haemoglobin is affected. The effect is same as oxygen deprivation or asphyxiation but rather more quickly. It is a strong pulmonary irritant causing pulmonary oedema.

Treatment of H_2S poisoning is complicated as its mechanism of toxicity is similar to that of cyanide. H_2S poisoning is commonly treated in the emergency room with intravenous sodium nitrite along with supportive therapy. Hyperbaric oxygen has been used as supportive therapy although it is not available routinely.[2] Given this case occurred in a diving system with the patient under increased ambient pressure, resuscitation was carried out using hyperbaric oxygen.

The deceased diver had reported that a trench had formed along the pipeline where the rupture had occurred and was filled with oil sludge. While working he had dislodged the sludge which resulted in release of dissolved H_2S. Although the diving bell was not directly above the leak, there is a possibility that some H_2S entered the diving bell. Electronic continuous gas monitoring system was not fitted in the diving bell nor was a handheld detection unit carried in the bell. The diving supervisor would not have had any indication of H_2S in bell.

Another possibility is that although the diver had discarded his coverall per standard practice, a considerable amount of oil was on his diving suit. His umbilical was also covered with oil sludge. Rising from seabed to the bell, the pressure decreased by almost 100 kPa (one bar), reducing solubility of H_2S in oil and excess gas was released from solution and entered the bell. The bell man was affected first. As soon as the diver removed his helmet and started breathing bell heliox mixture, he was affected, became unconscious and slipped out of the bell. He probably had no time to hook his harness to an 'eye' in the bell.

The bell man was breathing a heliox mixture containing 70 kPa PO_2 and on BIBS. This is probably the reason he survived a high H_2S content in bell brought in by the diver with his umbilical and diving suit. Aside from the short period of exposure with BIBS off when both exchanged pleasantries he did not continue to breathe from the contaminated bell environment. The diver on the other hand, was sitting and as soon as he removed his helmet, was exposed to high H_2S content at high pressure (750 kPa) and lost consciousness.

During treatment, the patient was given 35% HeO_2 mixture, called 'treatment mix'. At 65 msw he was breathing approximately 265 kPa PO_2. Six cycles of 20 min interspaced with five minute 'chamber mix' (50 kPa PO_2) was sufficient to neutralize the effects of hydrogen sulphide. Intravenous furosemide ensured pulmonary oedema was treated although not detected clinically. There was also no indication for inserting an endotracheal tube.

Ideally, a remotely operated vehicle (ROV) could have carried out a pipeline survey at zero risk. When this accident happened, ROVs were not routinely available. Today, work ROVs are present on MSVs and carry out pipeline surveys, marine growth removal, etc. Divers continue to work on pipelines but a similar accident has not recurred. The International Marine Contractor's Association (IMCA), an industry trade association representing offshore, marine and underwater engineering companies, has also revised its guidelines on diving in contaminated waters subsequently.[3]

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Echocardiography – techniques and pitfalls whilst diagnosing persistent (patent) foramen ovale as a risk factor in divers with a history of decompression sickness

Charles P Azzopardi[1], Kurt Magri[1], Alex Borg[2], Jake Schembri[3], Jonathan Sammut[3]

[1] *Baromedicine Department, Hyperbaric Unit, Mater Dei Hospital, Malta*
[2] *Cardiology Department, Mater Dei Hospital, Malta*
[3] *Mater Dei Hospital, Malta*

Corresponding author: *Dr Charles P Azzopardi, Baromedicine Department, Hyperbaric Unit, Mater Dei Hospital, Triq id-Donaturi tad-Demm, Tal-Qroqq, Msida, Malta*
charles.a.azzopardi@gov.mt

Key words
Bubbles; Decompression illness; Right-to-left shunt

Abstract

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The case of a diver with a history of decompression sickness (DCS) after recreational scuba diving is presented. Cutis marmorata, a subtype of cutaneous DCS, has been consistently associated with the presence of a persistent (patent) foramen ovale (PFO) as a risk factor. Diagnostic uncertainty arose when transthoracic echocardiography with antecubital injection of agitated saline bubbles (ASBs) did not show any significant shunt, but the presence of a large Eustachian valve was counteracted by intra-femoral injection of ASBs, showing a large PFO with spontaneous shunting. The importance of proper echocardiography techniques prior to resorting to intra-femoral injection of ASBs to counteract the haemodynamic effects of the Eustachian valve is emphasised.

Introduction

The case of a diver with a history of decompression sickness (DCS) after recreational scuba diving is presented. Cutis marmorata, a subtype of cutaneous decompression sickness, has been consistently associated with the presence of a persistent (patent) foramen ovale (PFO) as a risk factor for DCS. The case highlights an interesting point in relation to echocardiography for investigation for PFO.

Case history

The patient gave written consent for details and images pertaining to this case to be published.

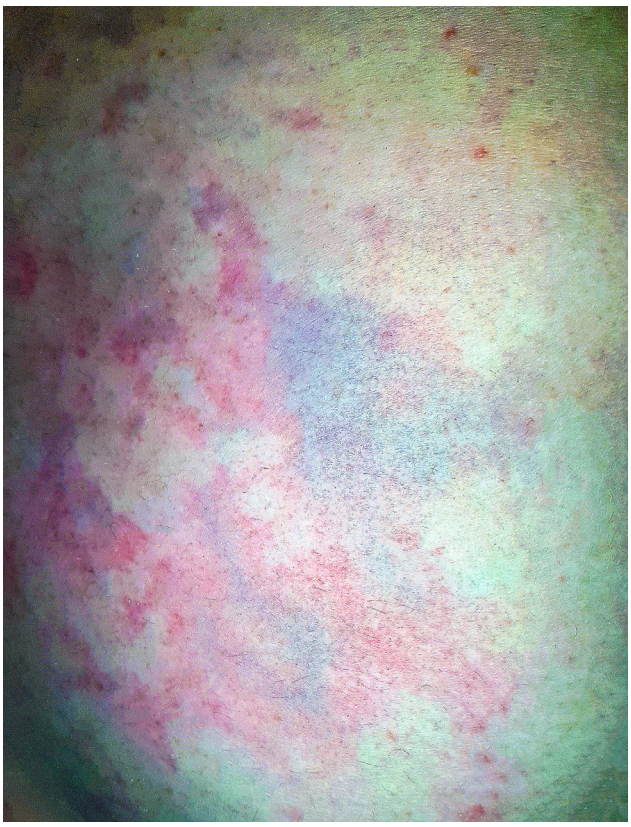
A 38-year-old female certified diving instructor was brought to the emergency department. She complained of an itchy rash over her abdomen 30 minutes after surfacing from her single dive of the day. She had a history of 3,996 logged lifetime dives, as well as three prior episodes of DCS, three years (lymphatic), two years (cutaneous) and one year (cutaneous) prior to this presentation. The maximum depth of the dive was 34 metres' sea water (msw) for an absolute bottom time of 85 minutes on open circuit 31% nitrox, with a total decompression time of 35 minutes, with a gas switch to 50% nitrox at 21 msw and 80% nitrox at 9 msw for accelerated decompression purposes. On the day prior to presentation, she had performed a single dive to 60 msw on open circuit trimix with a total dive time of 67 minutes.

On arrival at the emergency department, the patient was alert and orientated, eupnoeic, afebrile and normotensive. She had a normal full neurological examination. Her body mass index was 24.5 $kg \cdot m^{-2}$ and lab tests showed blood glucose of 5.6 $mmol \cdot L^{-1}$ and oxygen saturation of 99% on room air. A macular, pruritic non-blanching rash was present over all quadrants of her abdomen (Figure 1).

She was administered two litres of intravenous 0.9% sodium chloride prior to being transferred urgently to the hyperbaric unit for recompression therapy. She was treated with a United States Navy Treatment Table 5 with 100% oxygen at a treatment pressure of 282 kPa, with a total therapeutic table time of 135 minutes, excluding descent. This began within 35 minutes from the onset of her symptoms post surfacing. She exhibited full resolution of the rash within 10 minutes of compression to 282 kPa.

Trans-oesophageal echocardiography (TOE) incorporating a bubble contrast study performed in Hungary after the second episode of DCS had shown no evidence of a right-to-left shunt at pulmonary or atrial level. During this

Figure 1
Cutis marmorata rash post-diving



procedure contrast had been injected antecubitally, but no provocation measures were performed as the patient was sedated. Trans-thoracic echocardiography (TTE), this time performed in Malta, with antecubital vein injection of two separate boluses of bubble contrast, one at rest and one with provocation manoeuvres after the third episode of DCS, had shown an atrial level shunt with minimal spontaneous shunting. Abdominal compression and prolonged Valsalva augmented flow across the shunt. The diver elected not to change her diving practices seeing the small documented size of the atrial level shunt at this point. TTE performed in Malta eight weeks following the fourth episode of DCS, with intra-femoral injection of two boluses of bubble contrast to avoid the effect of the Eustachian valve, showed a large PFO with manifest spontaneous right-to-left shunting. The diver elected to proceed with percutaneous PFO closure.

Discussion

The foramen ovale is an inter-atrial connection that enables rapid flow of umbilical blood to the brain and vital organs during intra-uterine foetal life. At birth, the foramen ovale flap, the septum primum, is physiologically closed onto the septum secundum when pulmonary vascular resistance and right atrium pressure drop. Fusion, which begins with contact, is completed in the first two years of life, but may remain incomplete in up to 25–30% of the general population.[1] While individuals with PFO are generally discovered incidentally during autopsies performed for other indications, antemortem diagnosis is made during the diagnostic workup of clinical scenarios associated with PFO, such as cryptogenic stroke, migraine, sleep apnoea, platypnea-orthopnoea syndrome and DCS. In an autopsy study consisting of 965 people, PFO diameters were measured at between 1–19 mm (4.9 mm on average) and the mean size was 3.4 mm in the first decade and was 5.8 mm in the tenth decade.[1] One may thus hypothesise that small PFOs are closed over time and that large ones may remain open. When this process fails, the foramen remains patent and hence allows blood flow across it, while another mechanism which might be at play is dehiscence of a previously fused foramen ovale flap.

The combining hypothesis for the association of PFO with the numerous clinical scenarios mentioned previously is based on the passage of particulate emboli, bubbles or chemical substances from the venous circulation to the systemic circulation, bypassing the lungs through a right-to-left shunt. The left atrial pressure is higher than the pressure in the right atrium, which normally prevents passage by holding down the septum primum flap opposed to the septum secundum. However, situations may arise in which changes in intrathoracic pressure (e.g., during knee bends, straining post-dive, forceful Valsalva manoeuvre, or cough) may result in spikes in right atrial blood loading, increasing the risk of an embolisation process via a PFO.[2]

Another issue about PFO-mediated shunting and its implications during the diagnostic process are the blood flow dynamics in the right atrium and their relationship with the fossa ovalis. In the right atrium, the currents from the caval veins do not collide head-to-head, but turn forward and contribute to the rotation of the blood in a clockwise direction. This filling pattern, associated with directing the atrial volume towards the tricuspid valve entry, is extremely important in maintaining the continuous activity of the heart with minimal energy. The 'semi-lunar groove', lying next to the fossa ovalis where the PFO is located, needs to be appreciated as the source of turbulent blood flow which may impact the passage of blood through the PFO.[2] Venous bubbles injected into a large blood vessel antecubitally may be swept away from the inter-atrial septum by this turbulence and thus be prevented from becoming 'paradoxical gas emboli'. This may make the detection of a PFO by bubble contrast echocardiography a challenging task and contribute to a 'false-negative' result. Knowledgeable cardiologists are aware of this possibility and coach the subject to perform respiratory provocation (a sharp inspiratory sniff), Valsalva manoeuvres and abdominal compression manoeuvres to reliably diagnose a PFO.[3]

The PFO-mediated shunt can be determined by different echocardiographic techniques, namely TTE, TOE and transcranial Doppler (TCD). TOE has superior image resolution, and is able to define morphology, as well as the presence, number and size of these accompanying

Figure 2
Contrast-free zone peri-septally on the right side of the inter-atrial septum following antecubital bubble contrast injection

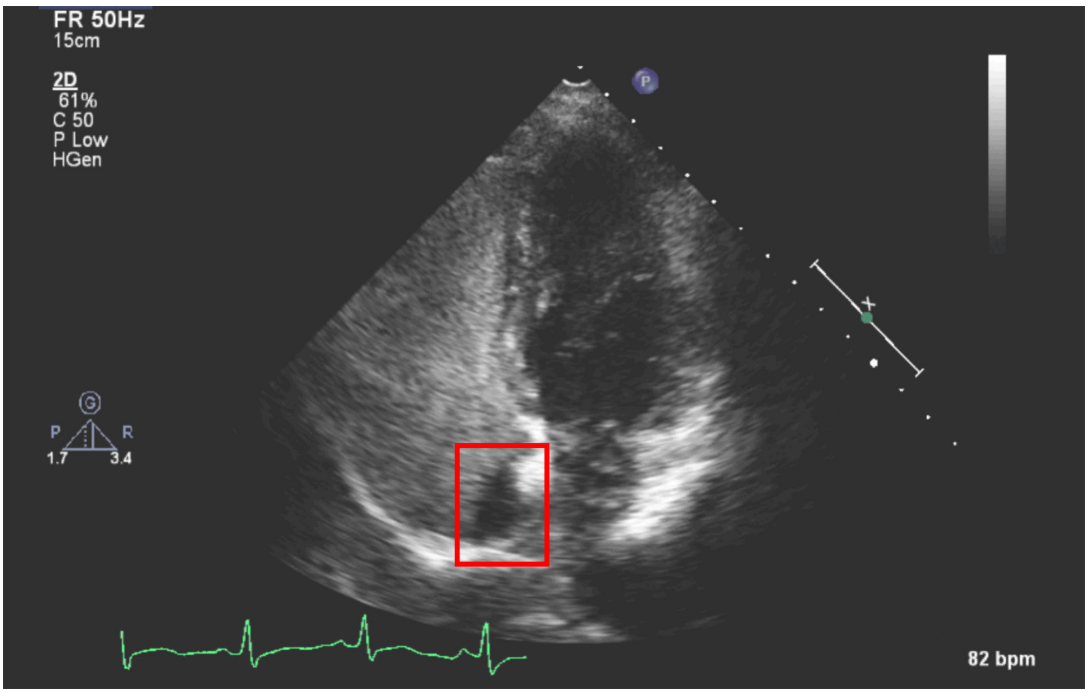
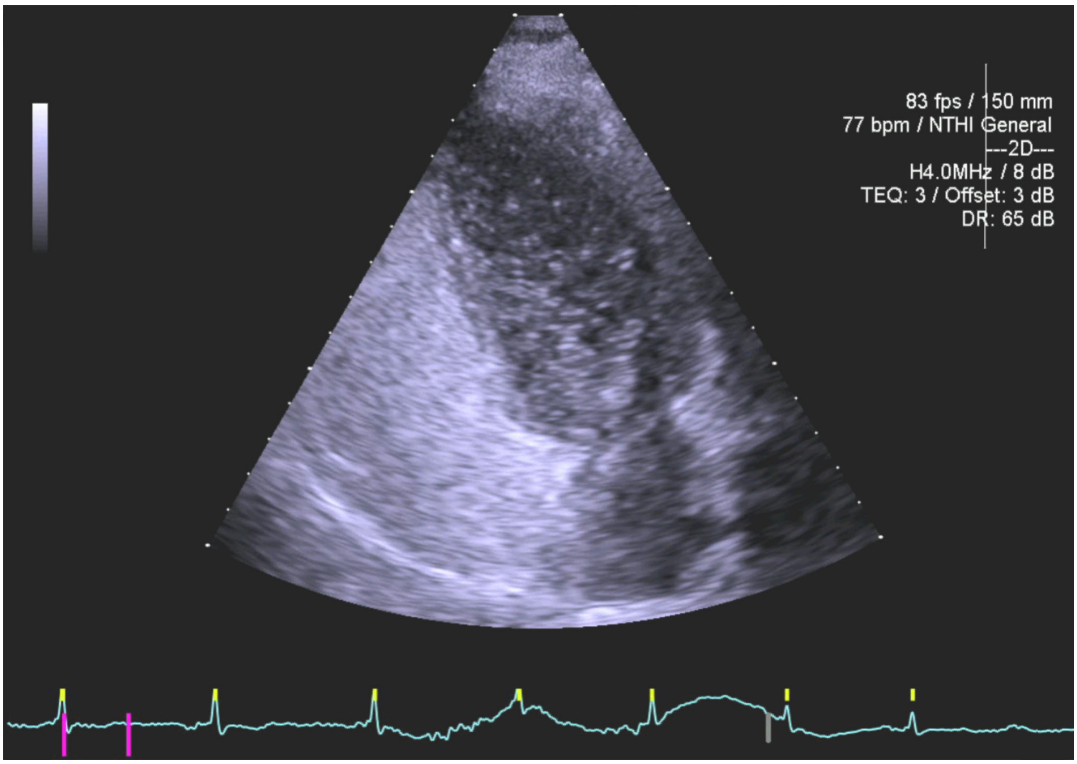


Figure 3
Transfemoral bubble contrast injection in same patient with no contrast-free zone on the right side of the inter-atrial septum; marked right-to-left bubble contrast shunting within 3 cardiac cycles



defects. It also allows assessment of the completeness of the septum apart from the defect and the presence of anatomic structures that will affect the placement of a closure device and visualising the three-dimensional appearance of PFO once closure is being considered.[4] However, it comes after TTE or TCD in the evaluation hierarchy because it is a semi-invasive procedure with well-defined staff training criteria and potentially life-threatening complications,

such as oesophageal haemorrhage and perforation, and it is contraindicated in patients with severe bleeding risk. TTE is thus the most frequently used initial screening test because of its low cost, non-invasive nature and easy accessibility.

These observations lead to the conclusion that while the anatomical caveats of the heart remain unchanged from the times of Vesalius and Galen, it is our understanding of its biomechanical properties and of the variable diagnostic impact of these aforementioned anatomical issues that has changed over the course of time. In 1986, Wilmshurst and colleagues observed neurological decompression sickness in a recreational scuba diver after a 15 minute dive to 38 m, and attributed its cause to venous gas emboli passing through a previously undocumented atrial septal defect.[5] While large atrial septal defects such as the one demonstrated in Wilmshurst's case are rare, small defects such as PFOs are present in up to 30% of the population, and provide a similar route for bubbles to enter the arterial blood. Indeed, several investigators have demonstrated an association between PFO and certain types of DCS, predominantly cerebral, spinal cord, cutaneous and vestibulo-cochlear DCS.[6–11] In these studies, right-to-left shunts have been demonstrated in as many as 89% of symptomatic divers with vestibulo-cochlear DCS, and 60% of divers in cases of cerebral or spinal cord DCS, compared with 20–30% of control subjects.[6–11] A recent study has also elucidated the presence of bubbles in the skin microcirculation underlying cutis marmorata in DCS patients with large right to left shunts.[12]

The presence of a persistent large Eustachian valve diverts bubble contrast approaching from the superior vena caval territory away from the interatrial septum. This can be appreciated and actively sought for on an apical 4-chamber transthoracic echocardiogram view, where a contrast-free zone may be visible peri-septally on the right side of the interatrial septum. This can be appreciated from the imaging obtained during the workup of the present case (Figure 2).

This contrast-free zone peri-septally should be seen as an indicator of a potentially false negative TTE for shunt identification, or underestimated shunt magnitude. We would not advocate to immediately change to femoral vein access, given that there are a number of techniques and manoeuvres to alter the flow dynamics and the opacification by bubble contrast of the right atrium, such as changing the positioning of the patient and pressing on the abdomen to reduce IVC inflow (Valsalva and IVC compression aim to reduce right atrial venous return to shrink the heart, so a rapid ingress of blood, opacified with bubbles, can improve diagnostic quality). Even if moving the patient does not alter the peri-septal contrast-free zone and identify the shunt, a sharp inspiratory sniff can improve the opacification and frequently demonstrates the shunt. If this does not work, then a prolonged Valsalva will, after release, cause a sudden increase in venous return and increase in right atrial filling. If this is timed appropriately with bubble contrast injection the entire chamber becomes opacified, and as the left atrial pressure and volume takes a little longer to recover, the septum will swing towards the left side, and if there is an atrial level shunt, bubbles will cross the septum, and be visible on the transthoracic echocardiogram.

In situations where the diagnosis of an atrial level shunt is still in doubt due to the presence of a Eustachian valve interfering with the injected bubble contrast reaching the inter-atrial septum, injecting bubble contrast by repeating the ante-cubital injection of contrast should be attempted, before resorting to injection via the femoral vein, which should remain the very last resort technique. This will introduce contrast via the inferior vena cava and result in better contact with the interatrial septum (Figure 3). One must take into consideration that femoral vein injection is associated with the risk of producing arterio-venous fistulas and haematomas, but this can be minimised with ultrasound guidance, and thus we must re-iterate our advice to use this technique with utmost clinical judgement and acumen.

Conclusion

The anatomical location of the Eustachian valve presents identifiable challenges to the accuracy and the sensitivity of TTE with transcubital injection of bubble contrast for the diagnosis of PFO. We recommend repeating TTE with femoral injection of bubble contrast only as a very last resort in cases with negative, repeated transcubital studies despite the use of provocative manoeuvres such as inspiratory sniff and abdominal compression, and only when elevated clinical suspicion of PFO-associated paradoxical embolism persists clinically. We thus advocate for proper echocardiography techniques during antecubital injection of bubble contrast to counteract the effect of the Eustachian valve during the echocardiographic diagnostic process of PFOs in divers with a history of DCS.

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Spinal cord decompression sickness in an inside attendant after a standard hyperbaric oxygen treatment session

Jacek Kot[1,2], Ewa Lenkiewicz[2], Edward Lizak[2], Piotr Góralczyk[2], Urszula Chreptowicz[2]

[1] *National Centre for Hyperbaric Medicine, Institute of Maritime and Tropical Medicine in Gdynia, Medical University of Gdansk, Poland*
[2] *Department of Hyperbaric Medicine and Sea Rescue, University Centre for Maritime and Tropical Medicine in Gdynia, Poland*

Corresponding author: *Associate Professor Jacek Kot, National Centre for Hyperbaric Medicine , Institute of Maritime and Tropical Medicine, Medical University of Gdansk, Powstania Styczniowego 9B, 81-519 Gdynia, Poland*
jacek.kot@gumed.edu.pl

Key words
Decompression illness; Hyperbaric facilities; Occupational health; Working in compressed air

Abstract

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Medical personnel in hyperbaric treatment centres are at occupational risk for decompression sickness (DCS) while attending patients inside the multiplace hyperbaric chamber (MHC). A 51-year-old male hyperbaric physician, also an experienced diver, was working as an inside attendant during a standard hyperbaric oxygen therapy (HBOT) session (70 minutes at 253.3 kPa [2.5 atmospheres absolute, 15 metres' seawater equivalent]) in a large walk-in MHC. Within 10 minutes after the end of the session, symptoms of spinal DCS occurred. Recompression started within 90 minutes with an infusion of lignocaine and hydration. All neurological symptoms resolved within 10 minutes breathing 100% oxygen at 283.6 kPa (2.8 atmospheres absolute) and a standard US Navy Treatment Table 6 was completed. He returned to regular hyperbaric work after four weeks of avoiding hyperbaric exposures. Transoesophageal echocardiography with a bubble study was performed 18 months after the event without any sign of a persistent (patent) foramen ovale. Any hyperbaric exposure, even within no-decompression limits, is an essential occupational risk for decompression sickness in internal hyperbaric attendants, especially considering the additional risk factors typical for medical personnel (age, dehydration, tiredness, non-optimal physical capabilities and frequent problems with the lower back).

Introduction

Medical personnel at hyperbaric treatment centres are at occupational risk for decompression sickness (DCS) while attending patients inside the multiplace hyperbaric chamber (MHC). The risk depends on both environmental and physiological factors. The environmental factors define the amount of inert gas dissolved in all tissues, depending on ambient pressure, time of exposure, breathing mixture and the decompression profile after a session. Additionally, physiological factors including age, exercise capability, level of hydration and acclimatisation influence the risk of DCS.[1]

Case description

The patient consented to publication of the following case details.

The patient was a 51-year-old male hyperbaric physician with a medical history of hypertension (well-controlled with drugs) and overweight (body mass index 28 $kg \cdot m^{-2}$), who was an experienced diver (thousands of logged dives including technical ones) with a history of recurrent back pain induced by physical exercise since his youth. He was working as an inside attendant during a standard hyperbaric oxygen treatment (HBOT) session in a large walk-in MHC. The session consisted of a 6-minute linear compression, 70-minutes at the pressure of 253.3 kPa (2.5 atmospheres absolute, 15 metres' seawater equivalent), then a 6-minute linear decompression without any decompression stops according to the Polish regulations.[2] During the whole session, the inside attendant was breathing ambient compressed air. There was no substantial physical activity during the session, and the previous hyperbaric exposure was about three days before. Later on, he claimed that for several days he had been psychologically tired due to work overload, and was stressed, over-caffeinated and dehydrated.

About 10 min after the session, he reported a burning sensation from the back toward the left leg, down to the knee, the loss of feeling in that region, decreased muscle strength, and no Babinski sign or plantar reflex (already lost in his youth). Trans-thoracic echocardiography (TTE) was conducted about 5 minutes later, showing four heart

chambers without detection of any bubbles either in the supine position or after knee bends (done mostly on the right leg). An independent physician, anaesthesiologist and diving medicine/hyperbaric specialist confirmed objective neurological signs. A lignocaine infusion was started (1 $mg \cdot kg^{-1} \cdot hour^{-1}$) with oral rehydration (1.5 L water). The decision was made to commence recompression treatment as soon as possible starting with compression to 283.6 kPa (2.8 atmospheres absolute) with oxygen and then continuing either with US Navy Treatment Table 6 (USN TT6) or converting to Comex CX30 with heliox 50% oxygen/50% helium, as typically used in spinal cord DCS in our hyperbaric centre for diving accidents. Recompression effectively started 90 minutes after the onset of symptoms.

After 10 min of breathing oxygen at 2.8 ATA, the apparent resolution of neurological symptoms was reported by the patient, and the standard USN TT6 was completed without any extension. After the session, no neurological symptoms persisted, other than the permanently missing plantar reflex. The lignocaine infusion was stopped, and the patient was discharged from the centre. He returned to regular hyperbaric work after four weeks of avoiding hyperbaric exposure. Eighteen months after the incident, transoesophageal echocardiography (TOE) was conducted with bubble contrast injected just before the Valsalva manoeuvre with no sign of persistent (patent) foramen ovale (PFO).

Discussion

Venous gas emboli (VGE) have been observed in inside attendants in a number of cases, depending on exposure pressure and time.[3,4] However, the rate of decompression illness (DCI), defined either as decompression sickness (DCS) or arterial gas embolism (AGE), among IAs differs between centres. A 2018 review of 14 articles on occupational risks for inside attendants participating in 79,776 hyperbaric sessions reported nine DCI cases in two centres; an incidence of 0.01% or one case per 8,864 sessions.[5] In one of two papers where cases were observed, there were four DCS cases reported in 28,747 hyperbaric sessions, but none with a neurological background.[6] The other paper reported in total five cases in 8,072 hyperbaric sessions, including three cases of neurological DCS.[7] Unfortunately, there is no specific information about those DCS with neurological symptoms, other than in two cases it was related to the inner ear.

Severe neurological symptoms of DCS or eventual death of medical attendants after hyperbaric treatment sessions are rare events. Until now, there has been only one fatal case described with a direct relation between death and DCS. This fatality occurred in 1991, when a 52 year-old nurse died within 90 minutes of exiting a MHC and autopsy findings confirmed her cause of death as DCS.[8–12]

One case of severe neurological DCS occurred in 1999 when a 43-year-old hyperbaric nurse became permanently quadriplegic (eventually leading to death from overwhelming infection after several years) due to neurological complications from a premature exit from USN TT6 with omitted decompression and both pulmonary and spinal cord DCS as a result.[11–13] The other publication from 2002 reported that a medical attendant at a hospital hyperbaric centre suffered 'a serious episode of neurological decompression illness', without giving any detailed information.[3]

Another case of cerebral and spinal cord DCS involving an inside attendant, which happened in 2001, was related to rapid chamber decompression due to deterioration of the patient, a diver with ventricular fibrillation, after several hours under pressure with the maximum treatment pressure of 607.8 kPa (6.0 atmospheres absolute). The inside attendant involved, a 44-year-old nurse, breathed oxygen during a very rapid chamber decompression and some minutes later she was recompressed for omitted decompression. After the completion of recompression treatment, she exited from the chamber and eventually returned home, where she was found several hours later in acute distress. Serial hyperbaric oxygen therapy and supportive care were incompletely successful, and she remained cognitively impaired.[12,14]

Yet another case of neurologic DCS was described in 2012.[15] A 50-year-old male complained of weakness and paresthesias in the lower extremities which began after serving as an inside attendant during a standard wound-healing hyperbaric treatment (222.9 kPa, 2.2 atmospheres absolute, 90 minutes at pressure) in a MHC. Within 10 minutes after the conclusion of the session, the patient experienced irritability, confusion and was unable to walk. He was recompressed with a USN TT6 within 60 minutes. His symptoms improved with compression; the patient was then treated with 222.9 kPa (2.2 atmospheres absolute) HBOT sessions until he was asymptomatic. Transthoracic echocardiography with bubble contrast performed 18 months after the event demonstrated a large PFO.

In our case, the spinal cord DCS in an inside attendant occurred after a standard HBOT session with a maximum pressure of 253.3 kPa (2.5 atmospheres absolute) and a bottom time within no-decompression limits. He had no PFO, but some additional risk factors were clearly identified, including age, overweight, dehydration and tiredness.

There are at least several possible pathophysiological mechanisms that may contribute to spinal cord DCS, including gas emboli, venous infarction, autochthonous bubbles or a vacuum phenomenon.[16–17] The specific mechanism cannot be confirmed in our case. The open question is whether lower back problems in the past with some permanent residual signs (loss of planar reflex) can predispose to DCS. A relationship between spinal canal stenosis and the development of spinal cord DCS was described in recreational scuba diving.[18] In the described case, the lower back pain occasionally occurred after heavy

exercise with lifting and gradually subsided within hours or days. CT scans conducted several years before did not show any sign of spinal canal stenosis.

Recompression treatment and adjunctive therapy (mainly lignocaine, nonsteroidal anti-inflammatory drugs [NSAID]) in spinal cord DCS is still debatable. A Cochrane review concludes that both the use of heliox and the addition of NSAID may reduce the number of recompressions required but neither improve the odds of recovery.[19] The European Committee for Hyperbaric Medicine (ECHM) recommends HBOT/recompression treatment tables (USN TT6 or helium/oxygen (heliox) Comex Cx30 or equivalent) for the initial treatment of DCI (strong recommendation, low level of evidence), but at the same time suggests the use of lignocaine and heliox recompression tables for severe neurological DCI (weak recommendation, low level of evidence), as well as oral tenoxicam (or similar NSAID) for appropriately selected DCI cases (weak recommendation, moderate level of evidence).[20] The Undersea and Hyperbaric Medicine Society (UHMS) advocates using US Navy oxygen treatment tables (or the similar RN and Comex tables, with initial recompression to 283.6 kPa (2.82 atmospheres absolute, 18 metres' seawater, 60 feet seawater equivalent) claiming that treatments at pressures exceeding 283.6 kPa or using helium as a diluent gas has not been demonstrated to be superior, and that their 'speculative' use should be reserved for facilities with experience and suitable hardware.[21] On the other hand, the UHMS does not give clear recommendations for adjunctive pharmacological therapy for DCI but presents guidelines for clinical efficacy of using different drugs, including lignocaine and NSAID. In summary of those guidelines, usefulness/efficacy of both lignocaine and NSAID in neurological DCI is less well established by evidence/opinion (Class IIb) based either on the consensus opinion of experts only (for lignocaine) or data derived from a single randomised trial or nonrandomised studies (for NSAID). In our clinical practice, a decision on using heliox recompression tables, lignocaine and NSAID is left to the treating physician, but most patients with neurological DCI receive all of them. In this case, the decision was agreed between treating physician and the patient (also the hyperbaric specialist) to try an oxygen table first (USN TT6) before considering switching to heliox Cx30 table (available at any moment in the same chamber). Fast resolution of all symptoms within the first 10 minutes under pressure confirmed the choice and prompted cessation of pharmacological therapy after the session.

In our hyperbaric centre, the decompression schedule of medical staff after standard HBOT sessions is planned according to the Polish regulations for commercial diving operations.[2] For standard HBOT sessions at 253.3 kPa (2.5 atmospheres absolute), it is allowed to have a bottom time of 80 minutes for no-decompression exposures. According to standard operating procedures, the decompression utilises only compressed air breathing to ensure the freedom of attendants to take direct care of patients in case of need. However, the personnel are advised to breathe 100% oxygen for either 10 minutes before commencing decompression or during decompression and decompression stops, or both, according to the recommendations of the ECHM.[22] Unfortunately, during this particular session, oxygen was not used for breathing.

Conclusion

Any hyperbaric exposure, even within no-decompression limits, is an occupational risk for decompression sickness in hyperbaric attendants, especially considering the additional risk factors typical for medical personnel (age, dehydration, tiredness, non-optimal physical capabilities and frequent problems with the lower back).

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Hyperbaric oxygen treatment in recurrent development of complex regional pain syndrome: A case report

Michal Hájek[1,2], Dittmar Chmelař[2,3], Jakub Tlapák[4,5], František Novomeský[6], Veronika Rybárová[6], Miloslav Klugar[7]

[1] *Centre of Hyperbaric Medicine, Ostrava City Hospital, Ostrava, Czech Republic*
[2] *Department of Biomedical Sciences, Institute of Microbiology and Immunology, Faculty of Medicine, University of Ostrava, Ostrava, Czech Republic*
[3] *Czech Anaerobic Bacteria Reference Laboratory, Faculty of Medicine, University of Ostrava, Ostrava, Czech Republic*
[4] *The Institute of Aviation Medicine, Prague, Czech Republic*
[5] *Department of Military Surgery, Faculty of Military Health Sciences, University of Defence, Hradec Králové, Czech Republic*
[6] *Department of Forensic Medicine and Medicolegal Expertises, Jessenius Faculty of Medicine, Comenius University, University Hospital, Martin, Slovak Republic*
[7] *Czech National Centre for Evidence-Based Healthcare and Knowledge Translation (Cochrane Czech Republic, Czech EBHC; JBI Centre of Excellence, Masaryk University GRADE Centre), Institute of Biostatistics and Analyses, Faculty of Medicine, Masaryk University, Brno, Czech Republic*

Corresponding author: *Dr Michal Hájek, Center of Hyperbaric Medicine, Ostrava City Hospital, Nemocnicni 20, 728 80 Ostrava, Czech Republic*
michalhajek@email.cz

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Abstract

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A broad spectrum of conditions including neuropathic pain, complex regional pain syndrome (CRPS) and fibromyalgia, have been implicated as causes of chronic pain. There is a need for new and effective treatments that patients can tolerate without significant adverse effects. One potential intervention is hyperbaric oxygen treatment (HBOT). The case reported here is unique in describing repeated HBOT in a patient who developed recurrent post-traumatic CRPS of the lower as well as the upper limbs. In the first event, two months after distortion and abruption of the external right ankle, the patient suffered leg pain, oedema formation, mild hyperaemia, limited mobility of the ankle and CRPS Type 1. In the second event, the same patient suffered fracture-dislocation of the distal radius 1.5 years after the first injury. After the plaster cast was removed the patient developed pain, warmth, colour changes, oedema formation and limited wrist mobility with CRPS Type 1. Pharmacological treatment as well as HBOT were used with significant improvement of functional outcome in both cases. Some studies suggest that patients with a history of CRPS are more likely to develop secondary CRPS compared to the rates reported in the literature among the general population. Patients with a history of CRPS should be counselled that they may be at risk for developing secondary CRPS if they undergo surgery or sustain trauma to another extremity.

Introduction

Chronic pain is one of the most common complaints in clinical practice. A broad spectrum of conditions including neuropathic pain, complex regional pain syndrome, migraine and fibromyalgia, have been implicated as causes of a chronic pain condition.[1–4]

Complex regional pain syndrome (CRPS) is a chronic pain condition characterised by spontaneous and evoked regional pain, usually beginning in a distal extremity, that is disproportionate in magnitude or duration to the typical course of pain after similar tissue trauma.[5] Multiple peripheral and central mechanisms are involved, with the individual share of particular factors over time. Possible contributors include musculoskeletal, peripheral and central sensitisation, autonomic changes and sympatho-afferent coupling, alterations in receptor populations (e.g., upregulation of adrenoceptors and reduced cutaneous nerve fiber density), brain changes, genetic, psychological factors, inflammatory and immune alterations and central changes in autonomic drive, which seem to contribute to regional and systemic disturbances in sympathetic activity.[5–7]

Management of pain, especially when it becomes chronic, is a challenging task requiring a multidisciplinary approach. Currently, most pharmacological, nonpharmacological and interventional modalities achieve only temporary or

modest improvements in pain symptoms and often produce intolerable adverse effects that interfere with the quality of life and lead to poor compliance. There is a need for new and effective chronic pain treatments that patients can tolerate without significant adverse effects. One such novel treatment is hyperbaric oxygen treatment (HBOT). There is a growing body of evidence to suggest that HBOT is a noninvasive modality with lasting efficacy and minimal side effects that can be used to treat chronic pain conditions.[8–11]

The aim of this case report was to report the apparent effect of repeated HBOT in a patient with post-traumatic CRPS in the lower limb and subsequently the upper limb. The report was developed according to the CARE reporting guidelines.[12]

Case presentation

Written informed consent for publication of case details was obtained from the patient.

EVENT 1

This 65-year-old woman had a history of general osteoporosis, right knee arthrosis and lower limb varicosities. In July 2018, she suffered distortion and abruption of the external right ankle due to slipping and falling to the ground. She was treated by plaster fixation for four weeks. Due to the phlebothrombosis of the deep venous system of this extremity, novel oral anticoagulant therapy was applied for ten weeks. Two months after the injury, she suffered pain in the leg and calf below the knee. Oedema formation, mild hyperaemia, limited mobility of the ankle and an antalgic walking pattern were described. She was diagnosed with CRPS Type 1 (CRPS not associated with direct nerve injury). Pharmacological treatment included analgesics (non-steroidal anti-inflammatory drugs) and anxiolytic therapy, vitamin D and calcium substitution. Physical therapy was applied for eight weeks. A visual analogue pain scale was rated 6/10 with limb loading by walking. Four months after the injury, in November 2018, HBOT was started. Twenty HBOT sessions were given at pressures 202.6–243.1 kPa (2.0–2.4 atmospheres absolute). Significant improvement of functional outcome after the treatment was achieved, such as the disappearance of symptoms, alleviation of colour changes and oedema reduction. The limb was fully loaded, with no pain at rest and during walking. The VAS score was rated 0/10 after the end of HBOT in December 2018.

On 19 March 2019, she was referred to HBOT again for a gradual recurrence of problems, such as dysesthesia and pain of the right leg and calf. No oedema or colour changes were present. Ultrasound examination showed a patent deep venous system. Pharmacological treatment included anxiolytic therapy, vitamin D, calcium substitution, and natrium-risedronate (bisphosphonates) 35 mg once a week. Physical therapy was applied for six weeks before the start of HBOT. The patient underwent another sixteen sessions of HBOT 202.6–243.1 kPa finishing in April 2019. There were no complications and side effects during the HBOT. Improvement and pain reduction was reported by the patient. The VAS score was rated 3–4/10 after the end of HBOT compared to 5–6/10 rated at the beginning.

EVENT 2

In December 2019, the same patient suffered an injury of the left wrist after the tripping on the sidewalk and falling. A dislocated fracture of the distal radius (Smith‘s fracture) was shown on X-ray. Reposition under the local anaesthesia and plaster fixation for six weeks were performed. After the plaster fixation was removed the patient complained of pain, warmth, colour changes as well as oedema formation of the wrist. Limited mobility of the wrist (dorsal flexion up to thirty degrees, palmar flexion up to fifteen degrees) and fingers were described. An X-ray revealed good position of fragments and progressive healing changes. She was once again diagnosed with CRPS Type 1. Pharmacological treatment included analgesic (tramadol hydrochloride/paracetamol 37.5 mg/325 mg twice daily) and antidepressant therapy (dosulepin hydrochloride), as well as promethazine hydrochloride. HBOT was started in January 2020. Twenty-two sessions of HBOT 202.6–243.1 kPa were given, finishing in March 2020, due to the worsening of the epidemiological situation and coronavirus disease pandemic outbreak. Significant pain reduction and partial oedema reduction were achieved, but reduction of the wrist mobility persisted. Slightly better finger mobility was apparent. Before HBOT, the pain VAS score was rated 0/10 at rest, 6/10 during movement, and the function of the hand was rated 7–8/10 (higher is worse). After the end of HBOT, the pain VAS score was rated 0/10 at rest, 2–3/10 during movement and the functional VAS score was 4–5/10.

Discussion

This report describes the effects of repeated HBOT administration in a patient with recurrent post-traumatic CRPS on both the lower and upper limbs. At an interval of 18 months after the first injury, the same patient had a forearm injury to the upper limb with development of CRPS, which was successfully treated with HBOT.

It is known that patients with CRPS have a higher chance of recurrence with a subsequent injury. In a retrospective review the incidence of CRPS after subsequent surgery or injury in a previous unaffected extremity was determined and compared to an average incidence reported in the literature.[13] Ninety-three patients had a diagnosis of primary CRPS. Nineteen patients (20.4%) developed CRPS in one or more additional extremities compared to the incidence of 23.4 per 100,000 (0.0234%) in the literature. Twenty patients had a documented secondary injury or surgery in a second extremity. Fifteen patients (75%) developed secondary CRPS compared to a CRPS incidence rate of 6.4% following distal radius fracture.[13]

The aim of another study was to evaluate the risk factors for the development of complex regional pain syndrome (CRPS) after surgical treatment of traumatic hand injuries. CRPS was diagnosed in 68 patients (26.2 %). The mean postoperative pain score was greater in patients with CRPS than in those without CRPS. The patients with a pain score of ≥ 5 in the first three days after surgery and the patients with crush injury were at high risk for CRPS development after surgical treatment of traumatic hand injuries.[14]

HBOT may be effective in the treatment of CRPS. A double-blind, randomised, placebo-controlled study was designed to assess whether HBOT was superior to placebo in treating patients with post-traumatic CRPS of the wrist. Seventy-one patients were randomised into a treatment group ($n = 37$) that received fifteen daily 90-minute HBOT sessions at 243.1 kPa (2.4 atmospheres absolute) or a control group ($n = 34$) that received fifteen daily 90-minute sessions in the hyperbaric chamber (also at 243.1 kPa) breathing normal air. The CRPS patients who received HBOT were shown to have significantly lower (improved) VAS scores, wrist extension and less wrist oedema compared to the control group both after the final treatment.[15]

While there is some supportive evidence of a positive effect of HBOT on CRPS, this chronic pain condition does not appear on any of the lists of approved indications of major professional societies such as the list of indications of the 10th ECHM Consensus Conference 2016.[16] This treatment method is neglected in many recent review articles or systematic reviews (SR),[17–19] where it is either not mentioned at all or excluded from the analysis, most often because it is not considered a 'commonly used treatment method'.[20] The present case report serves as a 'reminder' to the hyperbaric, orthopedic or pain medicine communities, that this treatment option exists, albeit based on limited scientific evidence of the clinical efficacy.

Possible mechanisms of action are multiple in relation to the above-mentioned currently accepted pathophysiological causes of CRPS. A positive effect of HBOT in CRPS could be related to restoration of aerobic metabolism, correction of hypoxia, correction of acidosis, and modulation of nitric oxide (NO) activity and oxidative stress.[21] Previous animal studies have highlighted the analgesic effect caused by HBOT in models of nociceptive, inflammatory and neuropathic pain.[22,23] HBOT has been found to decrease mechanical hyperalgesia and inflammation in a rodent model. The antinociceptive effect was apparent immediately following HBOT and persisted up to 5 h post-treatment.[22] In patients with fibromyalgia syndrome (FMS) there is some evidence that HBOT can change brain metabolism and glial function to rectify the FMS-associated abnormal brain activity.[24] Work by one group suggests that HBOT can induce neuroplasticity that leads to repair of chronically impaired brain function and improved quality of life in post-stroke patients and patients with prolonged post concussion syndrome.[25–27] Data from models of Parkinson's disease show that HBOT may play a neuroprotective role because of its ability to reduce oxidative stress and neurodegeneration, and protect against neuronal apoptosis.[28] It was shown that HBOT induces significant anti-inflammatory effect in different conditions and pathologies[29,30] and may attenuate pain by reducing production of glial cell inflammatory mediators.[31,32]

Conclusions

HBOT is not a standard treatment for CRPS, but it is a promising intervention for both acute and chronic treatment of the disease. Because of symptoms that limit patients in their daily lives, early diagnosis and active treatment approaches immediately after the onset of CRPS are critical factors in improving a patient's prognosis. Further studies are needed to improve our understanding of the mechanisms underlying the effects of HBOT and clarify its role in the treatment of this troubling disorder.

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Decompression sickness after a highly conservative dive in a diver with known persistent foramen ovale: Case report

William Brampton[1], Martin DJ Sayer[2,3]

[1] *Hyperbaric Medicine Unit. Aberdeen Royal Infirmary, Aberdeen, Scotland*
[2] *Tritonia Scientific Ltd., Oban, Scotland*
[3] *West Scotland Centre for Diving and Hyperbaric Medicine, Oban, Scotland*

Corresponding author: *Dr William Brampton, Hyperbaric Medical Unit, Aberdeen Royal Infirmary, Foresterhill, Aberdeen, AB25 2ZN, Scotland, UK*
william.brampton@nhs.scot

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Abstract

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A diver returned to diving, 15 months after an episode of neuro-spinal decompression sickness (DCS) with relapse, after which she had been found to have a moderate to large provoked shunt across a persistent (patent) foramen ovale (PFO), which was not closed. She performed a single highly conservative dive in line with the recommendations contained in the 2015 position statement on PFO and diving published jointly by the South Pacific Underwater Medicine Society and the United Kingdom Sports Diving Medical Committee. An accidental Valsalva manoeuvre shortly after surfacing may have provoked initial symptoms which later progressed to DCS. Her symptoms and signs were milder but closely mirrored her previous episode of DCS and she required multiple hyperbaric oxygen treatments over several days, with residua on discharge. Although guidance in the joint statement was mostly followed, the outcome from this case indicates that there may be a subgroup of divers with an unclosed PFO, who have had a previous episode of serious DCS, who may not be safe to dive, even within conservative limits.

Introduction

Diving with a persistent (patent) foramen ovale (PFO) has been linked with many forms of decompression sickness (DCS).[1–3] The hypothesis is that the usual venous bubbles generated after diving can cross through the PFO to the arterial circulation. Some of these bubbles pass into tissues that are supersaturated with inert gas which then diffuses into them causing amplification and resulting in DCS.[4]

A joint position statement (JPS) from the South Pacific Underwater Medicine Society (SPUMS) and the United Kingdom Sports Diving Medical Committee (UKSDMC) provides advice on diving with a known PFO; this includes the option to continue diving but within conservative limits.[5] The example given is to dive well within no stop limits, restrict depths to less than 15 metres, perform only one dive per day, use nitrox with air planning tools, lengthen a safety stop or decompression time at shallow stops and avoid heavy exercise or unnecessary lifting or straining for at least three hours after diving. Follow-up studies have found conservative diving lowers the risk of recurrent DCS in divers, with or without a right-to-left shunt.[6,7]

We report the case of a diver with a PFO, who, 15 months after recovering from neurological DCS after a rapid ascent, returned to diving and stayed mostly within the JPS recommended limits yet developed significant DCS.

Case report

The diver provided written consent for her case to be reported. This account is constructed through direct involvement with her acute management (WB) combined with information provided by colleagues and the diver, together with case note review.

INCIDENT ONE

In October 2018, a 28-year-old female diver undertook a weekend of diving near Oban on the west coast of Scotland. Her previous diving experience was uneventful and was estimated at just over 30 dives, all of them cold water and with a maximum depth of 35 metres' sea water (msw). Following two shallow dives the previous day (both < 10 msw) she performed a wreck dive to a maximum depth of approximately 20 msw and duration of 40 min but at the end of the dive she and her dive buddy became entangled in

the line of a surface marker buoy during its deployment and both made a rapid ascent (estimated at 30 to 60 m·min^{-1}) to the surface. On surfacing at 12:55, her buddy had symptoms consistent with DCS; he was given oxygen (O_2) on the boat and was subsequently treated with a standard Royal Navy 62 hyperbaric oxygen table (RN62), the Oban hyperbaric unit's routine 283.6 kPa (2.8 atmospheres absolute, 18 msw-equivalent) treatment table.

The female diver also received O_2 on the dive boat and some oral fluids before being transferred to Oban hospital accident and emergency (A&E) department. Her initial assessment, by a physician from the hyperbaric medical team, was unremarkable; she reported a headache that was present before the dive, and some mild discomfort to the posterior neck plus mild tenderness along the line of the trapezius muscle that were attributed to mechanical injury caused by the rapid ascent. As is normal practice in Scotland for divers who have had an uncontrolled ascent but no DCS, she was not recompressed but continued to receive normobaric O_2 for 4 h in A&E, during which there was no change to the previous symptoms. As at her initial presentation, these symptoms were not judged attributable to DCS so she was discharged under supervision of friends with a review planned for the next morning. Coincidentally, she was reviewed again at 21:30, when the group collected the buddy following his treatment; she was asymptomatic.

At 01:10 she developed paraesthesia in her right arm in ulnar distribution that was spreading and worsening in severity and she re-presented. She was recompressed at 04:25 on a RN62 modified with extensions. The treatment was eventful with episodes of vomiting and diarrhoea; she surfaced at 11:20 with residual symptoms. When reassessed at 17:00 she had developed new hyperparaesthesia in buttocks, thighs and legs consistent with deteriorating spinal DCS so was recompressed at 21:30 with a second RN62 modified with extensions. She surfaced with residua and received two once daily Comex 12 msw (Cx12) treatments over the following two days. Subsequently, mild balance impairment and lower back discomfort persisted but it was assessed maximum benefit had been gained and she was discharged. She later reported that the residual symptoms resolved over a number of weeks. Standard discharge advice from the Oban Hyperbaric Unit, for all divers who have suffered neurological DCS, is not to dive again but with the caveat of being tested for a PFO if continuing to dive.

In February 2019 she underwent examination by bubble contrast transthoracic echocardiogram (TTE), performed at a non-specialist centre. She was diagnosed with a right-to-left shunt caused by a probable PFO based upon the appearance of more than 30 bubbles in the left ventricle (LV) within three 3 beats, after the release of a Valsalva manoeuvre. Beyond this, no specific comment was made about the size of the shunt, or if there was an unprovoked shunt. She consulted a cardiologist in June 2019, who reported a past medical history of mild migraine with aura and a family history of PFO. Considering the rapid ascent to have been a clear provoking factor explaining the DCS, without having to invoke embolism across a shunt, the cardiologist advised that PFO closure was not indicated. She then consulted a UKSDMC-approved medical referee, in September 2019, who cleared her to dive with care using DCIEM air tables, or computer, to 15 msw on air and on nitrox below that.

INCIDENT TWO

She returned to diving in January 2020, 15 months after the first incident. Her first dive back was a shore based cold-water dive in a sea loch on the west coast of Scotland. She dived to a maximum depth of 12 msw with a bottom time of 30 min. She breathed air from surface to depth and during a controlled ascent to 6 msw at which point she switched to 70% nitrox. She made planned 3 min stops at both 6 and 3 msw before surfacing at a controlled rate at 13:30, with a total dive time of 40 min. There were no unplanned or adverse events during the dive. Whilst de-kitting, she accidently performed a Valsalva manoeuvre when bending and straining to remove tight fins. This was followed by a sharp, sudden onset occipital headache, which passed off rapidly, but no other symptoms.

At 16:40, having driven home with only minor altitude changes to a maximum of 200 m above sea level, she developed an itchy right shoulder and upper arm, but no rash. This progressed over about 90 min to include altered sensation in her right lower arm and hand with aching elbows and fingers. A home trial of oxygen at 20:45 made no difference but she felt the symptoms worsened when discontinued. At 22:00 she contacted the Scottish Hyperbaric Helpline and was brought to the hyperbaric medical unit in Aberdeen. Here, her symptoms were confirmed together with her history of neurological DCS and subsequent diagnosis of PFO. On examination the only abnormal finding was an unsteady sharpened Romberg's test, immediately falling to right. The rest of the neurological examination, including unprovoked Romberg's test and gait, was normal.

A diagnosis of neurological DCS with possible cutaneous and joint components was made and she was treated with the Aberdeen unit's standard 283.6 kPa treatment table – an un-extended US Navy Treatment Table 6, commenced at 02:00. After surfacing at 06:55 she was asymptomatic from her DCS and her sharpened Romberg's was normal. She was admitted to hospital for observation. Later that day she relapsed; at 16:30 she reported bilateral heaviness of her legs and "*unusual sensation*" in both thighs. This was very similar to, but milder than, the relapse she experienced after her first HBO treatment in 2018. Her sharpened Romberg's test was also unsteady again but there was no other neurological abnormality on examination. These symptoms were mild and stable but, in view of the similarity to 2018, it was decided further HBO was indicated. She was suffering some

troublesome pulmonary O_2 toxicity symptoms so, in the absence of deterioration, further HBO treatment was delayed until the next morning, to give her a longer air break. She then received the first of three daily Cx12 treatments. The second and third Cx12 treatments were given because new left-hand paraesthesia developed after the first Cx12 and the lower limb and balance symptoms persisted, although they were improving. After these treatments, the mild left-hand paraesthesia and subjective poor balance persisted but it was assessed that maximum benefit had been obtained so she was discharged with advice to stop diving. The residual symptoms settled over the ensuing two weeks, without further treatment.

Most strikingly, the pattern and timings of symptoms during this incident virtually mirrored those of her previous DCS, although of milder severity in the second episode.

Discussion

The DCS that developed after the first incident is fully explainable by the diver's unplanned rapid ascent causing autochthonous bubble formation in tissues, without having to postulate shunt across a PFO. However, in our experience, it is atypical for this to present so late after four hours of prophylactic surface O_2. As part of the treating unit's standard discharge advice, she was advised to have a bubble contrast TTE, if continuing diving, and this revealed a likely PFO with shunt by provocation following a Valsalva manoeuvre. The TTE was not done in a specialist centre and it is unclear if the bubble count of > 30 bubbles in the LV, within three beats of release of the Valsalva, was from a single frame or an overall total. Also, the standard method is to count bubbles in the left atrium (LA) rather than LV. A single frame count in the LA of > 30 would be taken by most specialists to indicate a large shunt.[8,9] This indicates the diver had, at least, a moderate and, likely, a large provoked shunt but the study would have been better done in a specialist centre as recommended by the JPS. A transoesophageal echo scan (TOE) can measure the size of the defect but TTE is the investigation of choice recommended by the JPS[5] and, in the UK, TOE is only likely to be used if a decision to close a PFO was being considered.

For the second episode of DCS, we postulate this was shunt related, but tissue inert gas load was low, so it is very unlikely to be caused by the mechanism of bubble amplification within supersaturated tissues, as is normally hypothesised.[4] Her dive followed the conservative diving approach by keeping to a maximum depth that was shallower than recommended as a conservative diving profile,[5,6,10,11] with a bottom time that was well within no-decompression limits (her bottom time was 120 min less than the no-decompression limit for 12 msw (150 min) following the DCIEM air decompression tables[12]), and with intentionally performed safety stops, using 70% nitrox, that were not required. This dive would have theoretically generated some gas supersaturation in her tissues, but it would have been low and short lived, as indicated by Repetitive Group 'B' on the DCIEM tables, had the dive been conducted without the safety stops. Shortly after surfacing, however, she did breach the JPS guidelines with an accidental Valsalva whilst de-kitting. We hypothesise that the occipital headache associated with this was indicative of a shower of venous inert gas bubbles passing through the PFO to the arterial side causing transient meningeal irritation and that, simultaneously, additional bubbles impacted other tissues, initiating the pathophysiological processes leading to DCS. Symptoms began some three hours after surfacing, which is longer than expected after usual shunt-related DCS,[1,13] but we submit that a different, and apparently slower, mechanism was in play with an arterial shower of bubbles alone being sufficient to provoke DCS in those areas damaged by the first episode. The similarity of the second DCS to her more severe previous one, suggests the presence of residual sub-clinical damage with vulnerability to further insult.

A previous study demonstrated that 14 of 19 divers with a 'grade 3' PFO (defined as a Valsalva provoked shower of bubbles too numerous to count in middle cerebral artery) generated detectable venous bubbles following a chamber dive to 30 msw, and six of these had arterial bubbles detected. This compared with divers in whom the PFO has been successfully closed where, although 11 of the 15 had venous bubbles detected, none had arterial bubbles.[14] In a deeper simulated dive to 50 msw in the same study, seven out of eight divers with a PFO had detectable venous bubbles, all of whom also had arterial bubbles but, although all five divers with a closed PFO generated venous bubbles, none had arterial bubbles detected. The dives in that study were deeper than the second dive in our case where the liberation of venous bubbles after surfacing would be expected to be low because of the conservative dive profile. However, we postulate from Honěk et al.,[14] that, if venous bubbling occurs after any dive, there is likely a significant chance of arterialisation across a PFO, particularly under provocation. The timing of the accidental Valsalva in this present case presumably coincided with venous bubbling, which then shunted to the arterial circulation.

Another cohort study compared divers with unclosed right to left shunt, who had been advised to dive conservatively, against those who had a closure procedure and found a higher risk of DCS in the former group.[7] Recent correspondence from Honěk's group, describing results from their DIVE-PFO registry, reports continuing incidences of 'unprovoked' DCS in divers with unclosed PFO but not in those who have had closure.[15] Both studies have limitations, they had a low number of end points, relied upon self-reporting by divers and do not describe the dive profiles associated with each DCS. However, they do demonstrate continued diving with a PFO carries an increased risk of DCS compared to diving after PFO closure.

In the case we report, other pathologies could have been considered in the differential diagnosis, such as cervical disc herniation or spinal cord pathology, but the diagnosis of DCS on each occasion was felt secure at the time, so these were not investigated. That DCS was the diagnosis would be strongly supported by the clear precipitating cause in the first incident and, on both occasions, by the proximity to diving, the pattern of evolution, response to hyperbaric oxygen, subsequent resolution of residual symptoms and absence of symptomatology before, between or since these incidents. It is possible that the diver had existing cervical cord pathology that predisposed to DCS. Spinal canal narrowing is more common in divers who have previously had DCS than those who have not.[16] Appropriate investigations and onward referral should be considered in divers who have suffered spinal cord DCS.

The JPS provides an important package of guidance[5] and this case illustrates how ambiguity can be introduced if it is not followed as a whole. In particular, PFO testing should be undertaken by centres well practiced in the technique who can provide definitive assessment of the significance of the shunt. The diver may have been better advised if this higher quality information had been available.

The JPS is based upon the available evidence but this, inevitably, only reaches level IIa at best.[5] The recommendations for divers with unclosed PFOs returning to diving following DCS are based on level IV evidence, expert consensus, and are founded upon the hypothesis of bubble amplification in supersaturated tissues. This may well apply to the majority of cases but even a single case that indicates it is not universally applicable is important. In the present case, it is the diver's second incident that casts doubt. It appears that, despite a very conservative dive, arterial bubbles embolised into tissues with a low inert gas load and this alone was sufficient to cause DCS, probably because of previous damage from an earlier, severe episode and possibly predisposed to by undiagnosed cervical cord pathology. There may be a subgroup of divers with a similar history who are not necessarily safe to dive, even within very conservative limits, with a PFO. In any case where the PFO is not closed, and the diver chooses to continue diving, this decision should be informed by high quality information about the shunt with expert interpretation. In addition, the necessity to avoid Valsalva manoeuvres following diving should be stringently reinforced.

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Hyperbaric oxygen treatment for late low colorectal anastomosis ischaemia: Case report

Marcello Sanzi[1], Alberto Aiolfi[1], Jacopo Nicolò Marin[1], Abd El Hakim Darawsh[2], Davide Bona[1]

[1] *Department of Biomedical Science for Health, Division of General Surgery, University of Milan, Istitituto Clinico Sant'Ambrogio, Milan, Italy*
[2] *Istituto Lombardo di Medicina Iperbarica, Milan, Italy*

Corresponding author: *Dr Marcello Sanzi, Istituto Clinico Sant'Ambrogio, Via Luigi Giuseppe Faravelli, 16, 20149 Milan, Italy*
marcello.sanzi@gmail.com

Key words
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Abstract
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Introduction: This report describes the use of hyperbaric oxygen treatment (HBOT) to treat a case of colorectal anastomosis ischaemia following colorectal surgery.
Case report: A 47-year-old man developed post-operative colorectal anastomosis ischaemia with leak after laparoscopic low anterior resection for T3N0 adenocarcinoma of the rectum. The leak with concomitant ischaemia presented 17 days after surgery. HBOT was administrated in 11 sessions over three weeks and the patient followed endoscopically and radiologically for two months. At two months the anastomosis showed both endoscopic and radiological healing; therefore the ileostomy was closed. Anal function was satisfactory with no incontinence or evidence of sepsis.
Conclusions: Intra-operative or late leak with concomitant ischaemia of a colorectal anastomosis is a challenging event in colorectal surgery. HBOT may be beneficial in promoting healing in selected patients. Further studies are needed to evaluate conservative treatments and the role of HBOT.

Introduction

Anastomotic leakage (AL) is a severe complication of colorectal surgery, with reported incidences ranging from 3% to 12%.[1] AL seems associated with additional life-threatening complications and mortality, and could have an adverse impact on disease-free survival, and local recurrence rates.[1,2] Independent risk factors for anastomotic leak include male sex, age, diabetes, preoperative radiotherapy for rectal cancer, and certain characteristics of rectal cancers including tumour size and distance from the anal verge. Modifiable risk factors include alcohol consumption, smoking, obesity, and immunosuppressant therapy, such as steroids. A diverting stoma at the time of primary surgery does not appear to reduce the frequency of AL but may reduce morbidity, mortality and the need for additional surgery if an anastomotic leak does occur.[1,3] Management options can be conservative with use of broad-spectrum antibiotics, radiological (e.g., drainage of pelvic collections), vacuum therapy, or reoperation.[4,5] As experience in minimally invasive surgical techniques such as laparoscopy or transanal surgery is spreading, these less invasive approaches for surgical management of AL are advocated.[6] We report a case of a 47-year-old man who experienced anastomotic leakage with segmental ischaemia after laparoscopic low anterior resection with ileostomy, that was treated with a conservative approach and hyperbaric oxygen treatment (HBOT).

Case report

The patient consented to the publication of this case report.

A 47-year-old male presented in October 2019 with tenesmus. Colonoscopy showed a substenotic neoplasm at 7 cm from the anal verge. Histopathology confirmed rectal adenocarcinoma. A computed tomography (CT) scan, magnetic resonance imaging (MRI) scan, and total-body positron emission tomography (PET) scan were performed, showing the presence of locally advanced rectal neoplasia and suspected loco-regional adenopathy. After multidisciplinary evaluation, a neoadjuvant treatment was adopted, consisting of simultaneous integrated boost mode radiotherapy with a total dose of 55 Gy (T, N+) and 45 Gy (regional lymph nodes), together with chemotherapy (Capecitabine). In December 2019 repeat CT and MRI scans revealed significant dimensional reduction. At the end of January 2020 the patient underwent laparoscopic anterior rectal resection with formation of a transanal ultra-low anastomosis and protective ileostomy. Definitive histological

Figure 1
Endoscopy showing segmental perianastomotic ischaemia at day 17 after surgery

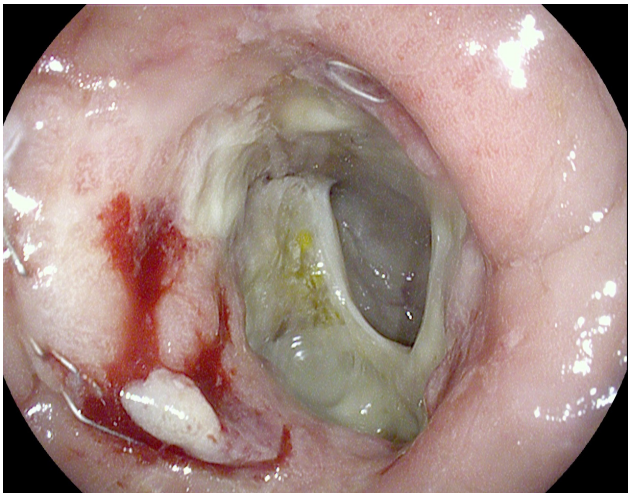
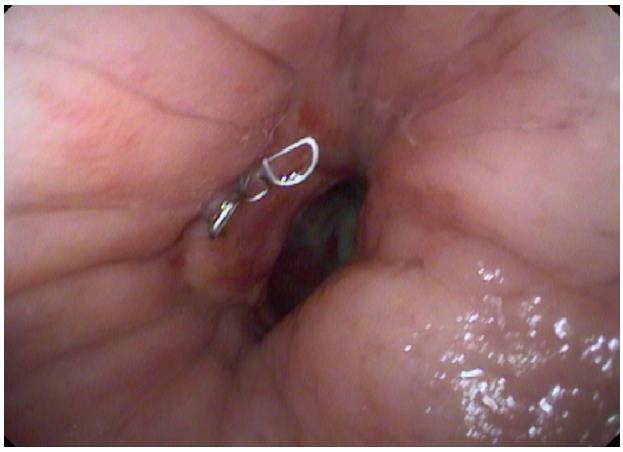


Figure 2
Endoscopy showing resolution of the segmental ischaemia and normal perianastomotic mucosa two months after the first HBOT session



exam revealed a moderately differentiated adenocarcinoma (T3, N0, Mx). The patient was discharged after a week without early postoperative complications.

Seventeen days after surgery the patient presented with perineal pain. A CT scan was performed with evidence of vascularisation deficiency 3.5 cm upstream of the anastomosis. Colonoscopy showed peri-anastomotic ischaemia with a 3 mm anastomotic leak (Figure 1). The patient was afebrile and was managed with empiric antibiotic therapy. After multidisciplinary discussion it was decided to manage the segmental ischaemia conservatively with HBOT as the patient had no contraindication to this treatment. He was discharged and an outpatient clinic follow-up with monthly CT scan and endoscopy arranged. Beginning 35 days after surgery, the patient underwent 11 sessions of HBOT administrated daily from Monday to Friday. The protocol for each session consisted of two 39 minute oxygen breathing periods at administration at 253.3 kPa (2.5 atmospheres absolute). Originally it was intended to continue for four to six weeks, but the patient had to interrupt it for regional restrictions related to COVID-19 infection spreading in the local area during March 2020. The patient did not receive any other treatment during the observational period. No complication related to administration of hyperbaric oxygen was observed. He was almost asymptomatic, experiencing occasional mild perianal discomfort that responded to analgesic therapy. Blood tests (red and white blood cell count) performed two weekly were normal during follow up. CT scan and endoscopic images showed progressive resolution of the anastomotic leakage and segmental ischaemia (Figure 2). Ileostomy closure was then performed and the patient was discharged home seven days later after physiological reactivation of bowel function. At follow-up day 30 after ileostomy closure the patient was asymptomatic with no anal leakage and in control of gas and solid evacuation, with a Wexner incontinence score of 0.[7]

Discussion

Rectal surgery remains a demanding procedure. Patients who have an anastomotic problems present a great challenge.[2] This report presents a single experience with the use of HBOT as an adjunct in nonoperative management of colorectal anastomotic complications. The case represents a singular success for this approach, as in our historical experience, these patients almost always require a definitive colostomy. Oxygen plays a central role in inflammation and wound healing, and HBOT has demonstrated its efficacy in the treatment of complex wound-healing problems in other settings. Oxygen is necessary for oxidative function of neutrophils, activation of leukocytes, fibroblast production, angiogenesis, and re-epithelialisation, which are of great importance in wound healing.[8] Evidence is still poor in the use of HBOT in surgical complications. The literature provides evidence of possible benefits of HBOT in animal models of colorectal anastomosis dehiscence with significant differences in oxidative stress markers in tissue specimen of the perianastomotic region after HBOT administration.[9,10] A small number of cases indicate benefit in tracheal and upper gastrointestinal surgery.[11] Few authors have explored potential benefit in humans with complications of lower gastrointestinal tract surgery,[12] although several relevant cases have been described,[13] and the present case adds to that experience. We believe more evidence is needed in order to encourage surgeons to use HBOT in selected patients.

Conclusions

Very low colorectal anastomosis leakage with segmental ischaemia accounts for significant morbidity and mortality. Conservative approaches to anastomotic leakage are

desirable in order to avoid further major surgery. There is level IV evidence of use of HBOT in this setting. HBOT may be a valuable option in the treatment of anastomotic leak and segmental ischaemia in selected patients. The present case affirms the possible success of a wait-and-see approach in the management of this complication using HBOT. More related evidence is needed.

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Pneumomediastinum and the use of hyperbaric oxygen treatment

Stephanie M Price[1,2], Will D Price[1,3], Mickaila J Johnston[1,2,4]

[1] *Naval Hospital Guam, Agana Heights, Guam*
[2] *USS FRANK CABLE, Santa Rita, Guam*
[3] *USS EMORY S LAND, Santa Rita, Guam*
[4] *Naval Medical Readiness Training Unit, Silverdale, WA, USA*

Corresponding author: *Dr Stephanie M Price, LT, MC, USN – USS FRANK CABLE, Unit 100105 Box 211, FPO, AP 96657, USA*
wetzelst10@gmail.com

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Pulmonary barotrauma; Pulmonary overinflation syndrome; Diving barotrauma

Abstract

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Pulmonary barotrauma may occur in diving and can result in a spectrum of injuries referred to as pulmonary over-inflation syndrome (POIS). Pneumomediastinum is a part of the POIS spectrum and only rarely results in respiratory symptoms. We present a case of a civilian diver who developed pneumomediastinum with respiratory symptoms which did not respond to normobaric 100% oxygen. After investigation for pneumothorax, he underwent hyperbaric oxygen treatment which resulted in significant alleviation of his symptoms. This is a novel case example of this treatment algorithm.

Introduction

Pulmonary over-inflation syndrome (POIS) is the spectrum of injuries that can result when pulmonary barotrauma (PBT) occurs. This barotrauma is the result of expanding gas within the lungs which occurs typically during ascent in a water column. The mechanism is explained by Boyle's law which states that pressure and volume are inversely related.[1] Thus, as the pressure decreases up a water a column, the volume of gas in a diver's lungs increases. This gas should be released through normal exhalation during ascent. Pathology arises when anything inhibiting or affecting the rate of gas release from the lungs occurs during the dive.[1] This report presents an example of PBT in a diver that resulted in symptomatic pneumomediastinum which responded to hyperbaric oxygen treatment (HBOT).

Case presentation

The patient gave permission for publication of this case report.

An otherwise healthy 32-year-old male made a planned recreational dive to 24 metres' seawater (msw) breathing air on open circuit scuba with a bottom time of 12 minutes and total dive time 50 minutes. At the end of the dive, he began to experience chest tightness within 3 msw of reaching the surface. On the surface he had obvious facial swelling, voice changes, chest pain and throat tightness with difficulty breathing.

He was wearing a dive watch that he reported sounded no safety alarms throughout his dive. Attempts to adjust his buoyancy compensating device (BCD) within the first two minutes of his dive resulted in an undesired ascent in the water column from 10 to 4 msw. His dive partner was with him during the entire dive but did not make this ascent in the water column. The dive partner remained asymptomatic.

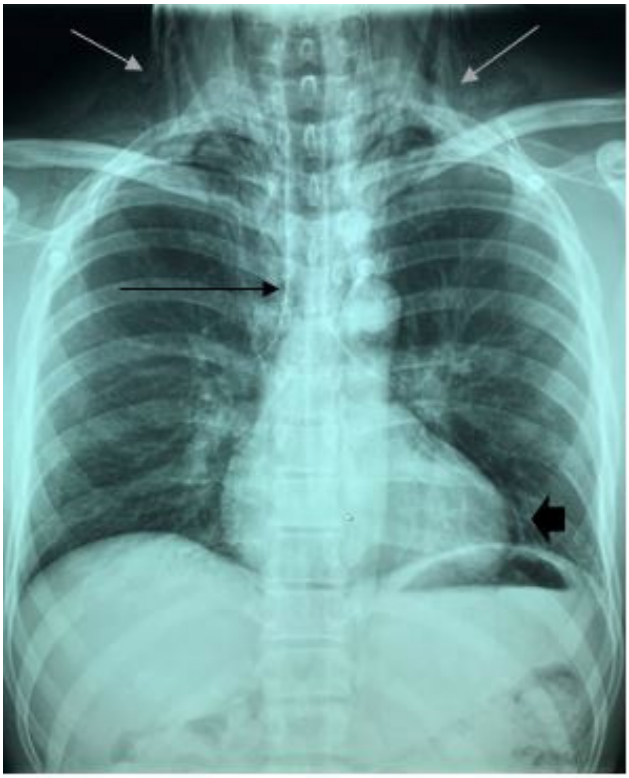
He was taken to the local emergency room where he was in obvious respiratory discomfort, leaning forward with hands on knees, with a respiratory rate of 26–30. His oxygen saturation (96–98%), heart rate (70–80·min^{-1}) and blood pressure (120–130/70-80) were all within normal limits. A 12-lead electrocardiogram showed normal sinus rhythm. He was started on normobaric 100% oxygen. On examination, there were notable voice changes and palpable crepitus along the cheeks and anterior neck and upper chest bilaterally. He had significant central chest discomfort throughout his respiratory cycle which was more notable during inspiration. Breath sounds were clear and were present bilaterally. A full neurological exam including mental status, motor, sensation, deep tendon reflexes and cerebellar signs demonstrated no deficits. His oxygen saturation was 100% during oxygen breathing. After 60 minutes of oxygen treatment, his respiratory rate had not decreased, he continued to demonstrate respiratory distress and there was only minimal improvement of his chest pain and dyspnoea. Chest radiographs showed pneumomediastinum, subcutaneous emphysema tracking up the neck and into the face and possible pneumopericardium. There was no pneumothorax

(Figure 1). Given the normal electrocardiogram, no cardiac blood tests were drawn.

Given the minimal alleviation of symptoms with supplemental oxygen and no contraindication to hyperbaric oxygen, the decision was made to treat him in the hyperbaric chamber located nearby. The chamber utilised was a standard Navy double-lock recompression chamber. Treatment was instigated within four hours of the initial injury. The US Navy Diving Manual recommends shallow recompression in the setting of severe pneumomediastinum in the absence of a pneumothorax. The table consists of breathing 100% oxygen at the shallowest depth of relief (usually 5–10 feet of seawater [fsw]) for one hour or longer if needed.[8] The chamber was initially pressurised to 131.7 kPa (1.3 atmospheres absolute, 3 msw, 10 fsw equivalent), and the patient was placed on 100% O_2 via a built in breathing system (BIBS) mask. Symptoms of chest and throat tightness, as well as voice changes, resolved 10 minutes into HBOT, and his respiratory rate normalised to 14–16·min^{-1}. There were no issues on compression or decompression in the chamber. He remained in the chamber at a pressure of 131.7 kPa for 60 total minutes. He had no recurrence of symptoms after surfacing. The subcutaneous emphysema resolved clinically approximately 72 h after the initial injury. Four weeks later he underwent thoracic computed tomography (CT) scanning without contrast, which was negative for any pulmonary abnormalities or residual injury.

Figure 1

Chest radiograph demonstrating pneumomediastinum with distinct outlining of the trachea (thin black arrow). Partial 'halo sign' (thick black arrow) along the left border of the cardiac silhouette. Extensive subcutaneous emphysema tracking bilaterally up the neck (thin gray arrows)



Discussion

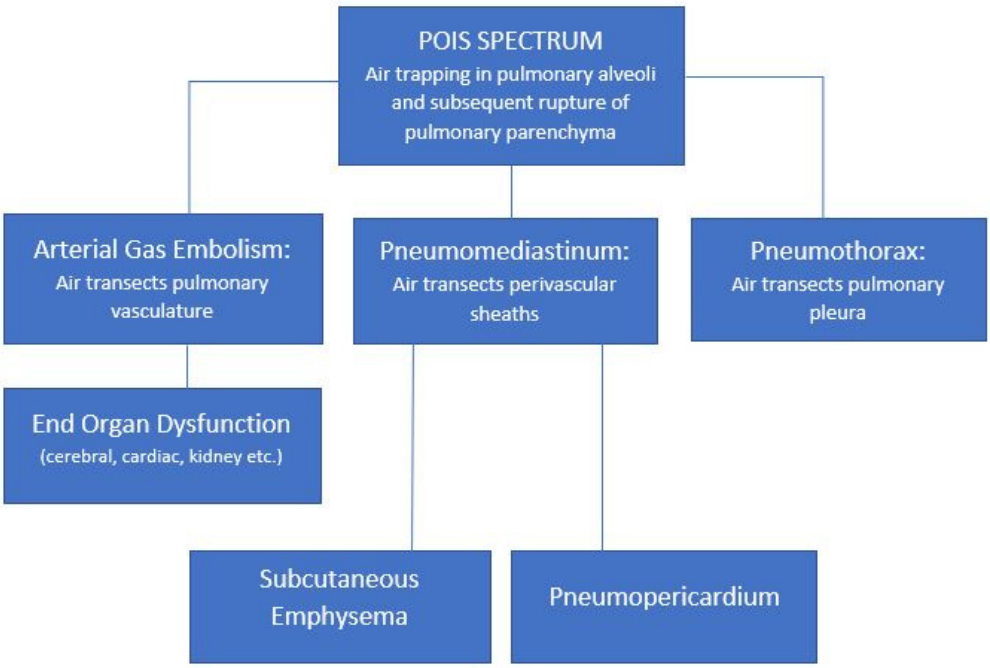
PBT is the second most common type of barotrauma after ear/sinus barotrauma.[2] POIS refers to the spectrum of injuries that result specifically from PBT which range from life threatening arterial gas embolism (AGE) to subcutaneous emphysema. As air expands within the lung parenchyma and reaches the necessary pressure difference that air can transect into the pulmonary vasculature, the perivascular sheaths and/or the pleura, resulting in one or a combination of POIS injuries (Figure 2).[1] Isolated pneumomediastinum results from air migration along the perivascular sheaths and pulmonary interstitium to the hilum to enter the mediastinal space.[1]

Risk factors for developing POIS are related directly to a diver's ability to appropriately exhale the expanding gas in the lungs. Rapid ascent, decreased forced expiratory volume (FEV), anatomic weakness in the pulmonary parenchyma and pulmonary obstruction are several risk factors commonly discussed in the literature.[1,3,4] This patient had no history of pulmonary disease such as asthma or recurrent bronchitis, or indications that he would have limitations to his respiratory flow-volume curve. However, no pulmonary function tests were conducted. The most severe consequence of POIS would be AGE, which he did not have. There is no clear explanation why some people develop AGE during a POIS injury and others do not.[1,5]

Most pneumomediastinum events are asymptomatic or mildly symptomatic.[6] Standard treatment for symptomatic pneumomediastinum is normobaric 100% oxygen.[6] Treatment with oxygen should alleviate most symptoms. Currently no definitive guidelines exist for what constitutes 'severe' pneumomediastinum. This patient was demonstrating signs of respiratory distress and elevated RR. Treatment with supplemental oxygen provided minimal relief of these symptoms which was the main factor in determining to undertake HBOT.

The chest X-ray was suspect for a potential pneumopericardium. This is generally rare and often benign however it can become life-threatening causing tamponade and cardiopulmonary failure.[3] Radiographic signs may include a decrease in the cardiac silhouette, or 'small heart sign', along with the heart being partially or completely surrounded by air, the 'halo sign'.[3] More commonly, the 'halo sign' finding is not attributable to pneumopericardium but instead further evidence of pneumomediastinum, with the anterior portion of the pleural reflection off the left border of the heart.[1] Although our patient had central chest discomfort, given his normal blood pressure, heart rate and 12-lead electrocardiogram we did not think there was clinical evidence of cardiac compromise. Had there been any

Figure 2
POIS spectrum diagram. These injuries can occur in conjunction or isolation of one another.



evidence to the contrary, cardiac blood investigations would have been a reasonable first step in work up.

The patient did have clinically obvious subcutaneous emphysema, which caused obvious symptoms with his facial swelling and voice changes. Subcutaneous emphysema is a part of the POIS spectrum and most typically ranges from asymptomatic to mildly symptomatic, presenting most commonly with soft tissue swelling/crepitus and voice changes.[7] However, symptoms can be severe and result in airway and/or vascular compromise.[7,8] The challenge then becomes how to successfully manage this type of airway, potentially under positive pressure ventilation, without further exacerbating the instigating pulmonary injury. For this specific patient, had his respiratory status deteriorated and there was concern for worsening tracheal obstruction, intubation may have become necessary. Carefully managing tidal volumes and positive end-expiratory pressure is critical in a patient like this.[8] Also considering other invasive means of trapped gas release, such as subcutaneous drain placement on low suction in areas of gas accumulation, may become necessary in order to avoid worsening the obstruction.[7] In this case, his respiratory discomfort could have been secondary to the subcutaneous emphysema he experienced or, more likely, a combination of the pneumomediastinum and the subcutaneous emphysema. This is further supported by the fact that after treatment in the chamber he clinically improved while there was still obvious clinical evidence of subcutaneous emphysema in the soft tissues of his face and neck.

Only one other reported case of an isolated POIS injury without AGE was found in which symptoms were manifested and treated with HBO_2. That case is from the 1950s; a male performing submarine escape training from 30 msw depth who developed symptomatic POIS injury without AGE or radiographic evidence of pneumothorax on a plain chest film. He was compressed to the depth of near complete symptomatic relief at approximately 608 kPa (50 msw equivalent) and subsequently underwent a US Navy Treatment Table 3.[9] As described above, the present case did not require such extensive recompression in order to achieve relief of symptoms. The patient was taken directly to 131.4 kPa (3 msw equivalent) on 100% oxygen with near complete resolution of symptoms within 10 minutes and was kept at depth for one hour. The US Navy Diving Manual recommends 'shallow' recompression in the setting of severe pneumomediastinum in the absence of a pneumothorax. The table consists of breathing 100% oxygen at 116.5–131.4 kPa (1.5–3 msw equivalent) for one hour or longer if needed.[10] Had this patient not experienced relief of symptoms, treatment could have progressed to deeper depth, while weighing the risk-to-benefit ratio of prolonged recompression to symptom relief.

Pneumothorax is the least common manifestation of PBT, however, the clinical consequences of compressing a patient with a pneumothorax could be severe given the risk of developing a tension pneumothorax during ascent.[1,3,11] Pretreatment evaluation of a PBT patient for pneumothorax is recommended when time and patient stability permit.

There was no evidence of pneumothorax on posterior-anterior and lateral chest radiographs. However, as a recent case report demonstrates, a negative CXR does not mean there is no pneumothorax.[12] There is always the potential a subclinical pneumothorax could be present during treatment and treating providers should be prepared to perform chest tube thoracostomy if this complication were to develop.[3,13–15] Clinical ultrasound and CT scans are more sensitive when compared to radiographs; however, they are not perfect modalities. In addition to being more sensitive for detecting a pneumothorax, a CT can evaluate for the presence of pulmonary bullae or bleb disease with more accuracy compared to plain radiograph.[13–17] These structural abnormalities are known risk factors for POIS injuries.[18] In retrospect, despite the negative CXR and reassuring exam, given the expedience and increased sensitivity, a point of care ultrasound at bedside would have been the ideal imaging to obtain for this patient to ensure he did not have a small pneumothorax that was missed on the radiograph prior to compressing in the chamber.

While the aetiology of this diver's injury is ultimately unknown, it is likely that during his initial uncontrolled ascent from 10 to 4 msw he may have unintentionally and unknowingly held his breath while trying to adjust his BCD. Prior studies which examined breath holding during ascent, found that out-of-air situations or panic due to unfamiliarity with equipment are the most common causes of PBT.[1,2] Given the type of injury and ultimate uncertainty regarding the aetiology, a CT was obtained post-injury to assess for the presence of any pulmonary abnormalities. Despite the negative imaging, and that this patient is an experienced diver with no previous episodes of injury, it was explained to him that given this POIS event his risk of recurrent injury may be higher if he chose to dive again, including the potential for more severe consequences including AGE.[19] For this specific patient the increased risks should be carefully considered before engaging in diving in the future.

Conclusions

This case provides a clinical example of HBOT in the setting of symptomatic pneumomediastinum with clear benefit to the patient. There is a paucity of documented cases of treating symptomatic POIS in the absence of AGE with HBOT. A suitable treatment table is offered as an option in the US Navy Dive Manual for more severe cases of pneumomediastinum in the absence of a pneumothorax. Questions of resource cost, availability and symptom severity should be considered when determining whether HBOT is a good option for a patient with symptomatic pneumomediastinum.

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Disclaimer

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Letters to the Editor

Commentary on "*Fatal air embolism in a breath-hold diver*" and the implied dangers of technical freediving

We read the recent publication titled "*Fatal air embolism in a breath-hold diver*" with great interest.[1] In this case report, the authors describe a breath-hold (BH) diver who breathed from a compressed gas cylinder at 10 metres' seawater (msw) and then ascended without exhaling. Upon surfacing, he became unconscious and was noted to have bloody sputum. Resuscitation at the scene was unsuccessful, and the young man died. A subsequent computed tomography scan demonstrated extensive pulmonary barotrauma (PBt) and cerebral arterial gas embolism (CAGE).[1]

Appropriately, the authors cite that this type of injury is rare in true BH divers. Additionally, there are only a few case reports of PBt and/or CAGE secondary to BH divers who have breathed from a scuba cylinder.[2,3]

However, a growing segment of BH divers, who are referred to as technical freedivers, use breathing from compressed gas cylinders at depth to facilitate deeper and longer dives.[4] As one would surmise, instructors teaching this technique train students to exhale gas prior to the surface to avoid PBt. A safety diver is typically employed to remind a diver upon ascent that they need to exhale prior to surfacing.[4]

Kirk Krack, a professional BH diver and pioneer of technical freediving, breathes from compressed gas cylinders as deep as 20 msw. With this technique and others, such as the use of diver propulsion vehicles to aid in descent, technical freedivers can reach and enjoy underwater settings once relegated to scuba divers only. In 2016, Krack and others visited Truk Lagoon and used technical freediving approaches to dive the multitude of sunken WWII wrecks.[4]

As classes teaching technical freediving expand, the number of participants employing the techniques, such as breathing from compressed cylinders at depth, will increase. Undersea medicine physicians should certainly be aware of these practices and educate those participating about the potentially deadly consequences of not exhaling prior to reaching shallow water or the surface.

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Derek Covington, Chris Giordano
Address for correspondence: *Department of Anesthesiology, University of Florida College of Medicine, Gainesville FL, USA*
covington.d@gmail.com

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Acute central nervous system oxygen toxicity at normobaric pressure

That acute central nervous system oxygen toxicity may occur at normobaric pressure[1] was suggested to me by a clinical experience nearly half a century ago. A healthy, burly male in his 30s was riding home on his motorcycle late one evening when he drove into a wire that had been stretched across the road, hitting him in the neck. Despite major haemorrhage and ruptures of his larynx and oesophagus, an emergency room physician successfully intubated him.

The following morning, in the operating room (OR) he was transferred from breathing 100% oxygen (O_2) spontaneously via his endotracheal tube and an Ambu bag to 100% O_2 from an old Boyle's anaesthetic machine via a circle circuit. Over a few minutes, he became increasingly tachypnoeic and restless and then deeply cyanosed before it was realised that nitrous oxide (N_2O), not O_2, had been turned on unintentionally. He was immediately turned onto high-flow 100% O_2 and his colour pinked up within a few breaths. Shortly thereafter (perhaps 10 seconds), he had a grand mal convulsion, which was controlled by an intravenous bolus of sodium thiopentone. No subsequent complications arose from this episode of which he had no recollection and he had no further convulsions during his long hospital stay.

In the early 1970s, continuous O_2 and carbon dioxide (CO_2) monitoring and modern anaesthesia machines were things of the future. On review, the anaesthetist, working in that OR for the first time, discovered that the N_2O rotameter was mounted on the left of the rotameter block on that Boyle's machine, whereas on all other machines in his previous clinical experience, including in all the other ORs in that hospital, the O_2 rotameter had been situated on the left of the block. He had turned the rotameter knob by touch behind him whilst attending to the patient.

Whilst the causation of his convulsion remains conjecture, the two most likely mechanisms are acute hypoxic hypoxia and acute oxygen toxicity. Hypoxic hypoxia occurs in a variety of environmental (breath-hold diving, altitude) and traumatic (drowning,[2] choking, strangulation) situations and in neonatal hypoxia. Seizures are well documented following hypoxic hypoxia.[3] Multifocal myoclonic jerks have been reported after loss of consciousness from breath-hold diving.[4]

Given the chronological sequence described above, the other possible explanation is that this was an oxygen-induced convulsion since it did not occur until the patient was fully re-oxygenated. Hypoxia is known to impair cerebral autoregulation,[5] though the interactions of O_2 and CO_2 are complex. The writer suggests that the cerebral circulation would have been vasodilated from the severe acute hypoxia when it was perfused by blood with a high partial pressure of O_2 which was then imparted to the cerebral tissues before autoregulation could be restored. Since CO_2 monitoring was not available on anaesthetic machines in the early 1970s, it is unknown whether or not hypercapnia contributed to this episode, as appears to have been so with the diver in the case report.[1]

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F Michael Davis
Address for correspondence: *Department of Anaesthesiology, University of Auckland, Auckland, New Zealand*
simo01@xtra.co.nz

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Oxygen; Toxicity; Central nervous system; Pressure; Case reports

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EUBS Notices and news

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

EUBS President's report

Ole Hyldegaard

EUBS Webinar on Diving – HBOT and COVID-19

On the 10 March 2021 we will be launching our webinar on HBOT, diving and COVID-19. The pandemic has had significant impact on the way we perform our daily practice of hyperbaric oxygen treatment, how divers work in the commercial sector, saturation diving and especially procedures for the safe enjoyment of recreational diving as well as fitness to dive after COVID-19 infection evaluations. To date we have had more than 400 registered participants for the EUBS webinar on HBOT – Diving and COVID-19, many of whom are not EUBS members. The interest has been overwhelming and illustrates the need amongst colleagues in our society on how to work in a hyperbaric setting in a future where COVID-19 will impact for years to come. I hope all participants have enjoyed the webinar and look forward to meeting with our distinguished speakers and audience.

EUBS Annual Scientific meeting 2021 – situation still unresolved with respect to COVID-19

The Annual Scientific conference meeting is currently scheduled for 08–11 September 2021, at the same location, the NH Prague City Hotel. Hopefully the situation will allow a physical meeting, but at the time of writing this, the decision will be made during March/April and we will confirm this via our website and accept the fact that the situation may not stabilize and force us to seek an alternative way. Rest assured, that we at the EUBS ExCom and our meeting organizers and colleagues in Prague will do our utmost to make the meeting happen as a physical one, but acknowledge the uncertainties and limitations we face with COVID-19 travel restrictions. In cooperation with the local organizers we will inform you on the final developments through our website.

Ole Hyldegaard
EUBS President

EUBS Notices and news

Annual Scientific Meeting: EUBS2020 in 2021

The unpredictable events associated with the spread of the COVID-19 pandemic in recent months have already prompted us to change and postpone the date of this scientific event, from 2020 to September 2021. The Annual 46th EUBS Conference will be held for the first time in its nearly 50-year history with an interval of two years. At least that's what we're hoping for at the moment, filled with optimism and belief in improving the situation. We believe that with all epidemiological measures and vaccination of the general population, the virus can be brought under control.

The conference meeting is currently scheduled for 08–11 September 2021, at the same location, the NH Prague City Hotel. However, in early March there will be important and decisive discussions between the members of the organising committee and the Executive Committee of the EUBS, which will decide whether, and what form the conference will take. Our hope is that the epidemiological situation in the world will allow personal participation of as many participants as possible, as we feel that in our professional community there is a major benefit from meeting in person. Both during lectures and during discussions in- and outside the meeting room and, above all, during social events, the scientific program gains much higher value, as has been observed during dozens of previous conferences. However, at this moment, we cannot rule out that a 'physical' meeting might have to be adapted to either a mixed (hybrid) or even purely online meeting.

The situation is evolving very rapidly, not only on a continental scale but even more at national and regional levels – and fluctuations are frequent, both in positive and negative direction. So, deciding the right option is not an easy task, and it is evident that it will not be clear until the last minute whether the choice was the right one.

In the meantime, please find the latest information on the website https://eubs2020.com/. As for now, registration is scheduled to start on 01 April 2021, along with the submission of abstracts.

EUBS Executive Committee

Every year, a new Executive Committee member needs to be elected – elections start well before our next General Assembly (during the EUBS Annual Scientific Meeting). Candidates will be presented by the Executive Committee by June 2021, and the voting will be, as usual, by internet ballot, starting on 30 June. If you want to contribute and help our Society, please come forward and send your short CV to our secretary: secretary@eubs.org.

This year, we will need a new Member-at-Large and a new Vice-President. If you do not feel up to presenting yourself, why not nominate someone else? Suggestions are welcome at the same email address.

EUBS Affiliate Society agreements

For 2021, an agreement has been renewed with the following Scientific Societies in order to promote membership and contact among the hyperbaric and diving scientists and practitioners in Europe and (why not) worldwide. Members of these Societies benefit from a 10% reduction on the EUBS membership fees, when providing proof of their membership of the 'other' society. Simply indicate the Affiliate Society on the EUBS Membership Application or Renewal Form.

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

For 2021, the Austrian Society for Underwater and Hyperbaric Medicine (https://www.oeguhm.at/) (ASUHM) has joined our Affiliate Society list.

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

In addition, we are discussing new agreements and invite other national societies to contact us to further expand these affiliate agreements.

Obviously, members of SPUMS already automatically benefit from most of our EUBS membership benefits, such as the DHM Journal, a reduced registration fee for the EUBS Annuals Scientific Meetings and access to the GTÜEM Database of non-indexed scientific literature.

EUBS website

Please visit the EUBS Website for the latest news and updates. The 'EUBS History' section (under the Menu item 'The Society') is still missing some information missing in the list of EUBS Meetings, Presidents and Members at Large – please dig into your memories and help us complete this list!

By popular demand, EUBS Members can also download the complete Abstract Book of previous EUBS Meetings from the Members Area.

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

OXYNET Database updated

Since 2004, a public online database of European Hyperbaric Chambers and Centres has been available, started and initially maintained by the OXYNET Working Group of the COST B14 project of the European Commission, later by the European Committee for Hyperbaric Medicine (ECHM). The original database (although not maintained) is still available on http://www.oxynet.org/.

However, over the past few years, the list and contact information of the OXYNET database have been updated thanks to the efforts of EUBS ExCom members, and hopefully, by the time you read this, be available online.

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

The

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.

Notices and news

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

SPUMS President's message

Neil Banham

I hope that all members, their families and loved ones have had a safe new year!

2021 continues for us, as 2020 finished in Australasia, small isolated COVID-19 outbreaks between periods of near normalcy, while our colleagues in USA and Europe continue to be hugely impacted by this virulent and deadly disease. The UK recently had similar case numbers and deaths per day to the total numbers in Australia from the start of the pandemic just over a year ago, and the USA is much worse. Living in such a terrible situation is unimaginable to us and hopefully with the increasing rate of vaccination, these dreadful numbers will rapidly decline. On behalf of SPUMS I extend our best wishes to our friends, family and colleagues elsewhere.

Despite COVID-19, SPUMS continues to maintain strong membership, over half of last year's members have renewed already. For those who have not yet done so, I encourage you to renew your membership so you may continue to benefit from being a part of our society, and enjoy access to the latest issues of our journal, the pre-eminent publication in the field of diving and hyperbaric medicine.

The outbreak of a case of COVID-19 from hotel quarantine in Perth at the end of January resulted in a lockdown and interstate travel restrictions which led to the postponement of the ANZHMG Introductory Course in Diving and Hyperbaric Medicine which was scheduled to be held in Fremantle from 15–26 February. This will hopefully run mid-year, depending on the COVID-19 situation at the time. Proposed dates are 24 May to 04 June 2021.

The good news is that our SPUMS Secretary, Doug Falconer has confirmed dates for a SPUMS Annual Scientific Meeting for 2021. This will be held at HMAS Penguin over three days in May with the possibility of those unable to travel to Sydney being able to join via Zoom. Unfortunately, unlike previous meetings, diving will not play a central role in this meeting.

Theme: 50 years of Diving Medicine: Remembering the past and preparing for the future
Date: 21–23 May 2021 (Fri/Sat/Sun)
Keynote Speaker: Dr Richard Harris

I encourage you to strongly consider attending our ASM and to submit an abstract early if you are considering presenting either in person or virtually. Please submit all abstracts to secretary@spums.org.au.

On another positive note, work is continuing by Nicky Telles to have all issues and individual articles of *Diving and Hyperbaric Medicine* (DHM) posted on the DHM website. This will be from the first issue of DHM published in March 2006, to the latest issue and will be completed by the end of April this year. Our thanks again to the Australasian Diving Safety Foundation who have generously funded this project. Prior to 2006, our journal was named *SPUMS Journal*, we have plans to have our existing digital copies of issues back to 2001 posted on the SPUMS website and eventually to have all issues back to the first in 1971 available, these will have to be scanned from hard copy.

Our journal continues to go from strength to strength with a continued rate of high quality submissions being received. This has increased the workload for our Editor, Simon Mitchell and Editorial Assistant Nicky Telles, thank you both for your efforts!

In closing, I would like to welcome Dr David Cooper on board as the new SPUMS Education Officer. David has succeeded Dr David Wilkinson OAM in this role. *'Wilko'* has been a trusted servant to our Society over many years and as such we are greatly indebted to him. We wish him all the best in his well deserved retirement.

Hopefully by the time you read this, the COVID-19 situation in Australasia and the rest of the world will be much improved and we can look forward to the day when life will really return to normal. Stay safe!

Neil Banham
SPUMS President

SPUMS Facebook page

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

2021 SPUMS 50th Annual Scientific Meeting

We are going (mostly) online for the SPUMS 2021 ASM.

With travel restrictions changing daily, the ASM in our 50th year will be delivered via Zoom as well as face-to-face, to accommodate the current travel rules.

Logo created by Chris Chivers

50 years of Diving Medicine:

Remembering the past and preparing for the future

Date: 21–23 May (Fri/Sat/Sun) 2021
Keynote Speaker: Dr Richard Harris
Location: HMAS Penguin (Sydney) or via Zoom (COVID pending)
Abstract submissions: Please submit all abstracts to secretary@spums.org.au (form to follow)

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 https://www.anzca.edu.au/education-training/anzca-diploma-of-advanced-diving-and-hyperbaric-me.

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

Written examination	11 August 2021
Viva examination	08 September 2021

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.

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 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 October 2020, the SPUMS Academic Board consists of:

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

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

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 the autumn of 2021. Dates are to be confirmed.

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

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

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

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

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

German Diving and Hyperbaric Medical Society's (GTÜM) website is currently unavailable owing to a new website being built. They have advised that a notification will sent when their database will be available again, They apologise for any inconvenience this may cause.

Scott Haldane Foundation

As an institute dedicated to education in diving medicine, the Scott Haldane Foundation has organised more than 295 courses all over the world, over the past 28 years.

SHF is targeting more and more on an international audience with courses worldwide. Due to the COVID-19 Pandemic some courses are re-scheduled. Fortunately we were able to find new dates for all postponed courses. Below the upcoming SHF-courses in 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).

2021

19–20 March	Medical Examiner of Divers part 1(level 1), Amsterdam, NL
25–27 March	Medical Examiner of Divers part 2 (level 1), Amsterdam Univ. Med. Centre, NL
10–07 April	Medical Examiner of Divers part 2 (level 1), Bonaire, Dutch Caribbean
11–12 June	In-depth course Decompression, Hoeven, NL Recompression and HBOT (Level 2d)
September	28th In-depth course diving and mental health (2d), tbd
01–02 October	Medical Examiner of Divers part 1 (level 1), Zeist, NL
07–09 October	Medical Examiner of Divers part 2 (level 1), Amsterdam Univ. Med. Centre, NL
Spring 2021	Internship different types of diving (2d) Royal Dutch Navy-Den Helder NL
On request	Internship HBOt (level 2d certification) NL/Belgium

The course calendar will be supplemented regularly. For the latest information see: https://www.scotthaldane.nl/en/. Please also check the COVID-19 news update on this website for the latest schedule changes.

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

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

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

NEW ZEALAND – NZUA
0800-4DES-111 (in New Zealand toll free)
+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.
