Diving and Hyperbaric Medicine

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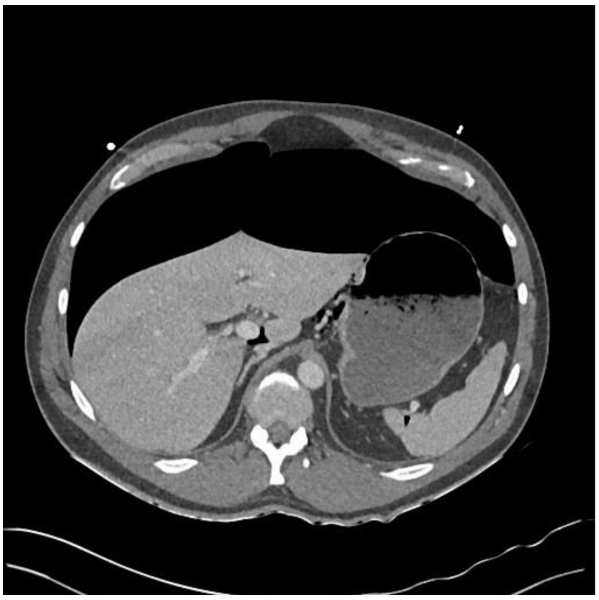

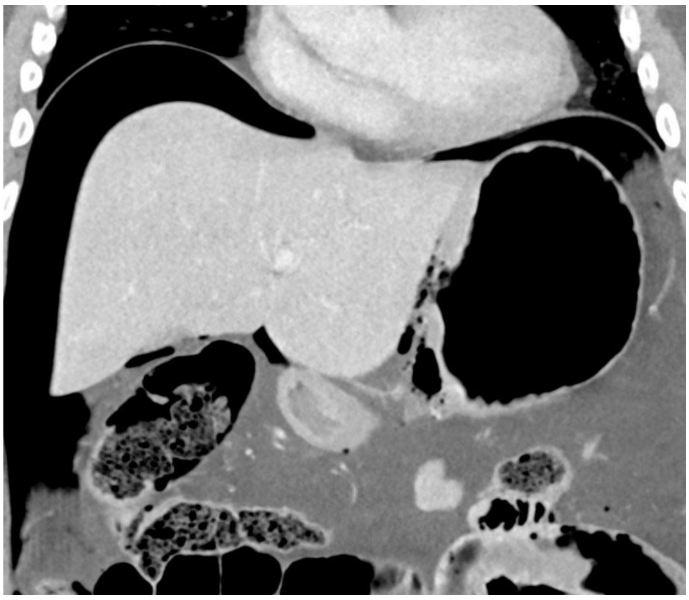

Gastric barotrauma in scuba diving

Economics of HBOT in wound healing

Number of HBOT treatments for hearing loss

Lung squeeze incidents in freediving

Risk factors for recreational diving accidents

Does meclizine increase risk of oxygen toxicity

Trends in competitive freediving accidents

Diver treatments at Townsville, Australia

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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

Welcome to the final issue of DHM for 2024; another large and eclectic one. In this issue we publish an economic analysis of hyperbaric oxygen treatment (HBOT) for limb salvage in patients with diabetic foot ulcers by Robin Brouwer and colleagues from Amsterdam UMC. The study is based on data from the recent DAMO$_2$CLES study. It must be remembered that such analyses are immutably affected by the clinical ecosystem in which they are conducted (in this case the Netherlands) which affects key parameters like patient selection and costs of treatment. With that limitation in mind (as clearly acknowledged by the authors), across the broad spectrum of wound severity there were no demonstrated advantages with HBOT, but when the analysis was confined to patients with Wagner III-IV lesions (which aligns with UHMS recommendations for use of HBO) there was a clear trend towards better effectiveness and cost-effectiveness.

Elsewhere on the hyperbaric medicine side, Brenda Laupland and colleagues provide observational evidence that while HBOT seems effective in this patient in sudden hearing loss, there is probably no advantage for more than 10 HBO treatments treating these patients. The remainder of this issue is given to diving-related papers.

Elaine Yu and colleagues explain the rationale for the term 'freediving induced pulmonary syndrome' and report survey data (acknowledging its inherent limitations) which define the incidence of 'lung squeeze' events. These are becoming more widely reported with the popularization of competitive freediving. In a related paper, Jeremie Allinger and colleagues provide a fascinating 'big data' evaluation of a competitive freediving database to define the incidence of loss of consciousness and pulmonary barotrauma events; unquestionably the most definitive work in this regard to date.

Kurt Tournoy and colleagues surveyed Belgian divers and (once again with acknowledgement of the potential limitations of survey data) correlated health conditions among respondents to the risk of diving related hospitalisation. Cardiac medication and ear-nose and throat disease were the strongest associations with diving accidents.

Guy Weiner and colleagues point out the potential utility of the combined antihistamine / anticholinergic agent meclizine in preventing sea sickness, and conducted experiments in mice to demonstrate no increase in the risk of cerebral oxygen toxicity in meclizine-treated mice.

Denise Blake and colleagues report on the divers evacuated to the Townsville Hyperbaric Unit over approximately 15 years. As reported by authors in other jurisdictions, for those divers recompressed, delays to recompression were long. In divers with decompression sickness, more severe symptoms were the only factor predictive of a poorer outcome.

In an account we have designated as a 'technical report' Felix Wood and colleagues describe the diving activity and diving medical capability / infrastructure at the Rothera British Antarctic Survey station. This is an interesting and useful account of precautions and contingencies for diving in austere remote environments.

Although long recognised as an issue in clinical HBOT, hyperoxic myopia has been less conspicuous among divers. This is changing, especially during multiday expeditions conducted by technical divers. With reference to a specific case, Sofia Sokolowski and colleagues review the literature on 'myopization' related to diving.

There are two societal position statements or guidelines in this issue. One pertaining to paediatric and adolescent diving (from SPUMS) and the other to immersion pulmonary oedema (IPO) (from SPUMS and the UK Diving Medical Committee). The latter has a particular emphasis on return to diving (or not) after an episode of IPO.

Finally, there are three case series / reports pertaining to gastric barotrauma, shunt related decompression sickness in a hyperbaric attendant, and the potentially controversial treatment of inner ear barotrauma (IEBt) with HBOT. In the latter series, five cases of IEBt appeared to benefit from HBOT with no adverse effects.

I recently had the privilege of attending two fabulous diving education events: Diving Talks in Portugal and Baltic Tech in Poland, and offer my congratulations to Arlindo Serrao and Tomasz Stachura (respectively) for their organizational efforts. I strongly recommend future iterations of these events to DHM readers, along with the Omani Navy International Diving and Hyperbaric Medicine Conference in Muscat in February 2025 (see advertisement on page 371). My best wishes to all readers for the Christmas / New Year season.

Simon Mitchell
Editor, Diving and Hyperbaric Medicine Journal

Cover photo: Axial and coronal computed tomography views of a diver who suffered severe pneumoperitoneum following gastric barotrauma (from Ayad et al. in this issue).

Original articles

Economic analysis of hyperbaric oxygen therapy for the treatment of ischaemic diabetic foot ulcers

Robin J Brouwer[1,2*], Nick S van Reijen[3*], Marcel G Dijkgraaf[4], Rigo Hoencamp[2,5,6], Mark JW Koelemay[3], Robert A van Hulst[1], Dirk T Ubbink[3]

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Keywords
Peripheral arterial occlusive disease; Cost-effectiveness; Wound healing

Abstract
(Brouwer RJ, van Reijen NS, Dijkgraaf MG, Hoencamp R, Koelemay MJW, van Hulst RA, Ubbink DT. Economic analysis of hyperbaric oxygen therapy for the treatment of ischaemic diabetic foot ulcers. Diving and Hyperbaric Medicine. 2024 20 December;54(4):265–274. doi: 10.28920/dhm54.4.265-274. PMID: 39675733.)
Introduction: The aim was to determine the cost-effectiveness and cost-utility of additional hyperbaric oxygen therapy (HBOT) compared to standard care (SC) for ischaemic diabetic foot ulcers (DFUs) regarding limb salvage and health status.
Methods: An economic analysis was conducted, comprising cost-effectiveness and cost-utility analyses, with a 12-month time horizon, using data from the DAMO$_2$CLES multicentre randomised clinical trial. Cost-effectiveness was defined as cost per limb saved and cost-utility as cost per quality-adjusted life year (QALY). The difference in cost effectiveness between HBOT+SC and SC alone was determined via an incremental cost-effectiveness ratio (ICER).
Results: One-hundred and twenty patients were included, with 60 allocated to HBOT+SC and 60 to SC. No significant cost difference was found in the intention-to-treat analysis: €3,791 (bias corrected and accelerated [BCA] 95% CI, €3,556 –€-11,138). Cost per limb saved showed an ICER of €37,912 (BCA 95% CI €-112,188–€1,063,561) for HBOT+SC vs. SC. There was no significant difference in mean QALYs: 0.54 for HBOT+SC vs. 0.56 for SC alone (-0.02; BCA 95% CI -0.11–0.08). This resulted in a cost-utility of minus €227,035 (BCA 95% CI €-361,569,550–€-52,588) per QALY. Subgroup analysis for Wagner stages III/IV showed an ICER of €19,005 (BCA 95%CI, €-18,487–€264,334) while HBOT did not show any benefit for Wagner stage II.
Conclusions: HBOT as an adjunct to SC showed no significant differences in costs and effectiveness for patients with DFUs regarding limb salvage and health status. However, for patients with Wagner stage III/IV ischaemic DFUs there was a trend towards better effectiveness and cost-effectiveness.

Introduction

Diabetes mellitus is a major healthcare issue, with a worldwide prevalence of 422 million patients.[1] Diabetic foot ulcers (DFUs) are a serious complication of diabetes,[1] and are often associated with peripheral arterial occlusive disease.[2] Two out of three amputations are related to DFUs, with a yearly amputation rate of 2.5% for diabetic patients.[3,4] Treatment of DFUs is complex and consists of offloading of the ulcer, restoration of skin perfusion, treatment of infection, metabolic control, local wound care, education, and prevention of recurrence.[5]

Hyperbaric oxygen therapy (HBOT) has been approved for 15 different indications, including the adjunctive treatment for DFUs;[6,7] this involves breathing 100% oxygen at an elevated atmospheric pressure in a hyperbaric chamber to promote tissue oxygenation.[8] Hyperbaric oxygen may promote wound healing through stimulation of neovascularisation, stem cells and growth factors, inhibition

of the inflammatory response, and a bacteriostatic effect on anaerobic bacteria.[9] It is considered a low-risk, yet cumbersome therapy. Relevant adverse effects are middle ear barotrauma (up to 2%), myopia, and sinus barotrauma.[10] An untreated pneumothorax is an absolute contra-indication for HBOT and there are many relative contra-indications including claustrophobia, chronic obstructive pulmonary disease, heart failure, metastasised malignancy, pregnancy or chemotherapy.[11]

It is important to make a distinction between ischaemic and non-ischaemic DFUs as HBOT appears more effective in the former group. A recent meta-analysis found that adjunctive HBOT significantly reduced the risk of major amputation as compared to standard treatment in patients with ischaemic DFUs (Risk difference 15% (95% CI, 6–25%).[12] In contrast, this benefit of HBOT could not be found in a systematic review that only included patients with non-ischaemic DFUs.[13]

Evidence regarding the cost-effectiveness of HBOT for ischaemic DFUs is scarce, since previous cost-effectiveness analyses did not distinguish between ischaemic and non-ischaemic DFUs.[14,15] A cost-effectiveness analysis from 2003 on a small sample of 18 patients for ischaemic DFUs estimated a potential cost saving of £2,960 for each patient treated with HBOT.[16] The lack of solid evidence on the costs and effectiveness of HBOT may be one of the reasons why the treatment is still not fully endorsed and implemented for (ischaemic) DFUs.

Hence, the aim of the current study was to determine the cost-effectiveness and cost-utility of additional HBOT compared to standard care for ischaemic DFUs regarding limb salvage and health status, based on data from the DAMO$_2$CLES trial, the largest study so far on HBOT for ischaemic DFU patients.

Methods

This economic analysis is reported according to the Consolidated Health Economic Reporting Standards (CHEERS).[17] The full checklist is enclosed in the Online appendix *. Data for this analysis were derived from the DAMO$_2$CLES trial.[18] In brief, the DAMO$_2$CLES trial was a multicentre, randomised, parallel-group, superiority trial, conducted in 24 hospitals in the Netherlands and one in Belgium. The study was approved by the medical ethics review board of the Amsterdam UMC, location AMC and by the local site investigators. The protocol (NTR3944) and primary results have been reported previously.[18,19]

PATIENTS

Participants were eligible for inclusion if they met all of the following criteria: type 1 or 2 diabetes; an ulcer of the lower extremities categorised as Wagner grades II-IV, present for at least four weeks; and limb ischaemia, defined as an absolute ankle systolic blood pressure < 70 mmHg, an absolute toe systolic blood pressure < 50 mmHg, or a forefoot transcutaneous oxygen pressure ($TcPO_2$) < 40 mmHg. The indication for revascularisation was assessed before randomisation and according to local practice.

Patients were excluded if they met any of the following criteria: previous ipsilateral major amputation (i.e., above the ankle); absolute contraindication for HBOT; or inability to complete questionnaires in Dutch.

TREATMENT

All patients enrolled in this trial received standard care (SC), which included open or endovascular revascularisation if feasible, and optimal conservative treatment (antibiotics, anticoagulants, glycaemic control), as well as local wound treatment, according to the guideline issued by the International Working Group on the Diabetic Foot,[20] and local practice. Patients allocated to SC plus HBOT were referred to a HBOT facility. Hyperbaric treatment included sessions of 90 minutes in a multi-place chamber, pressurised at 243 or 253 kPa (2.4 or 2.5 atmospheres absolute [atm abs]) during which patients were breathing 100% F_iO_2, except for three blocks of five minutes during which ambient air was administered to reduce the risk of oxygen toxicity. Hyperbaric treatment was scheduled for five days a week until a maximum of 40 sessions or until complete wound healing was achieved.

DATA COLLECTION AND OUTCOME MEASURES

General considerations

The economic evaluation was undertaken as a cost-effectiveness analysis and a cost-utility analysis. When conducting a cost effectiveness analysis, the so-called incremental cost-effectiveness ratio (ICER) is calculated, which (here) is the difference in *cost* between HBOT+SC and SC only, divided by the difference in *effectiveness* of HBOT+SC versus SC only. When limb salvage, based on major (above the ankle) amputation rates, is chosen as measure of effectiveness, the ICER shows the amount of money needed per additional limb saved. Usually, this ICER is calculated by repeating various scenarios ('bootstrapping') to get a more reliable estimate. Obviously, the more money is spent, the more limbs may be saved. Society as a whole should interpret the magnitude of this ICER to decide which amount they are willing to spend to save an extra limb, which might vary depending on the country and culture.

Similarly, a cost utility analysis was performed with the costs per quality-adjusted life-year (QALY) gained as outcome. An incremental cost utility ratio (ICUR) is calculated by

*The Online appendix can be found on the DHM Journal website: https://www.dhmjournal.com/index.php/journals?id=343.

dividing the difference in cost between HBOT+SC and SC only by the difference in QALYs between both treatment groups. Again, society should judge what amount they are willing to pay for each patient in order to gain one additional QALY.

Cost effectiveness analysis was performed from a limited societal perspective. Time horizon was set at 12 months. With this time horizon no discounting of costs and effects was performed. Both an intention-to-treat and two per-protocol analyses were performed. In per protocol analysis A, we compared patients who had a complete HBOT treatment course, meaning that treatment was continued until complete closure of the wound or for at least 30 completed HBOT sessions, with those who did not complete this HBOT regimen and those who received SC. For per protocol analysis B, we compared all patients who underwent at least one HBOT treatment with those who did not receive any HBOT treatment.

Resource analysis

Resource use was derived from the prospectively collected $DAMO_2CLES$ data for hospital stay, surgical and endovascular procedures, HBOT sessions, rehabilitation after major amputation, and wound care. Diagnostic procedures, such as duplex ultrasonography, magnetic resonance imaging and angiography, were not taken into account, as these were performed in all patients before inclusion in the study.

The number of rehabilitation treatments and costs of care after major amputation were not available for all patients, so an estimate was made based on unit cost prices from the national guideline for costing in healthcare.

Cost analysis

Costs were expressed in Euros and unit costs were taken from the Dutch cost manual[21] and, if not available, from the Amsterdam University Medical Center's hospital ledger. Costs derived from different calendar years were price-indexed for the year 2019, based on the price index numbers from Statistics Netherlands to present the most recent available costs. Standard national reimbursement tariffs for HBOT were used. The tariffs used for the unit costs of HBOT treatments, healthcare costs and out-of-pocket expenses derived from the various sources can be found in the appendix. If unit costs were available from more than one source, the Dutch cost manual tariff was used for reasons of generalisability.

Direct medical costs related to HBOT and other necessary treatments were assessed and compared between the SC and additional HBOT groups. Direct non-medical, patient-related costs included out-of-pocket costs of wound care products and travel expenses for HBOT sessions and outpatient visits. These were recorded by self-reported questionnaires at three and 12 months. The costs were calculated per kilometre and, if not available, the average travel distance of included patients (24.4 km) was used. Considering that nearly all patients had retired, indirect non-medical costs were considered negligible and therefore not taken into account.

Effectiveness of treatment

The occurrence of a major amputation was registered during follow-up. The EQ-5D-3L questionnaire was completed at baseline and after three, six and 12 months of follow-up to generate health status scoring profiles over time, which were transposed into health utilities using population-based tariffs of time trade-off ratings of health states.[22] Based on the health utility scores over time, QALYs were calculated as the area under the curve following interpolation of scores at successive measurements during the 12 months of follow-up.

DATA COLLECTION AND STATISTICAL ANALYSIS

Usage of resources was reported as totals per resource and as means per patient for HBOT treatment, healthcare, and out-of-pocket costs. Differences in estimates of the mean costs for these major cost components were analysed using an independent-samples t-test with their bias-corrected and accelerated 95% confidence intervals (BCA 95% CI) after bootstrapping, drawing 10,000 samples of the same size as the original samples, and with replacement. Bootstrapping was stratified by treatment group. Subgroup analysis was performed for Wagner stage II and stage III or IV wounds. Although not described in the original protocol, this subgroup analysis was added since international HBOT-guidelines advocate that HBOT should only be used for patients with Wagner stage III wounds or higher.[7]

The ICER and ICUR results were visualised by cost-effectiveness and cost-utility planes showing scatter plots of differences in costs on the Y-axis against differences in effect on the X-axis. These plots show the mean ICERs and ICURs, each with their dispersions over the four quadrants of costs vs. effectiveness and QALYs, respectively. All statistical analyses were performed using SPSS version 28 (IBM SPSS, Armonk, NY, USA) and R-studio version 3.6.1.[23]

HANDLING OF MISSING DATA

Planned EQ-5D-3L measurements were missing in the HBOT group for 23–40% of follow-up moments and in the standard care group in 20–30% of occasions after assigning '0'-values to foregone assessments following a patient's death. No apparent attrition bias emerged in patterns of missing data over time. Assuming missing data to be completely at random and considering the amount of missing health utility data, we imputed eleven data sets including group allocation, gender, age, having had a major amputation during follow-up (at months three, six and 12), and available health utility scores as predictors. The imputed

health utility scores were constrained to the theoretical range for Dutch health utilities (-0.329 to 1). The mean of the health utilities per patient per time point were used to derive QALY estimates. QALYs were estimated by linear interpolation between successive points in time.

SUBGROUP ANALYSIS

Wagner stage II and Wagner stages III/IV patients were analysed as separate subgroups because this classification may lead to effect modification.

Results

From June 2013 until December 2015, 120 patients were included in the $DAMO_2CLES$ trial, of whom 60 were allocated to HBOT+SC and 60 to SC alone. Baseline patient characteristics are shown in Table 1 and were similar in both study arms, except for age and haemoglobin level. Of the 60 patients allocated to HBOT, 49 patients actually started the treatment, and 39 completed all treatments. Of the 60 patients allocated to SC, four received HBOT at their own request.

COST OF TREATMENT

The volumes and costs of treatment per study can be found in Table 2. Table 3 shows the outcomes after bootstrapping. Mean cost for HBOT+SC was €26,228 (BCA 95% CI, 21,229–31,644) vs. €22,437 (BCA 95% CI, 18,141–27,407) for SC only. Mean difference between treatment groups was €3,791, which is not statistically significant (BCA 95% CI, -3,251–11,138).

COST-EFFECTIVENESS

The amputation rate in the HBOT+SC group (12%) was not significantly lower than in the SC group (22%), risk difference (RD) 10% (95% CI, -4–23).[18] This resulted in a mean ICER of €37,912 per limb saved (Figure 1, BCA 95% CI, -112,188–1,063,561). Meaning it would cost €37,912 to preserve one limb. Neither per protocol analysis showed different or statistically significant results. (Tables 4 and 5)

COST-UTILITY

Table 3 shows the mean QALYs during follow-up resulting from the EQ-5D-3L scores. Mean QALY for HBOT+SC was 0.54 (BCA 95% CI, 0.48–0.60) and for standard care 0.56 (BCA 95% CI, 0.49–0.63), which is a non-significant difference of minus 0.02 (BCA 95% CI, -0.11–0.08). Mean ICUR was minus €227,035 per QALY (Figure 2, BCA 95% CI, -361,569,550– -52,588). This mean negative result suggests that in general, HBOT+SC was less effective and more expensive than SC alone. This is also illustrated in Figure 2, showing fewer patients in the right half, and especially in the lower right quadrant, of the scatter plot. The per protocol B analysis showed a similar result, while no significant differences were found in the PP A analysis. (Tables 4 and 5)

SUBGROUP ANALYSES

In Wagner III/IV patients the amputation rates were 9% in the HBOT+SC group and 32% in the SC group (RD 23%; 95%CI, 3–43) which is a statistically significant difference. In Wagner II patients the mean ICER was minus 577,390 (Figure 3, BCA 95% CI, -16,922,632– -468,585), meaning that HBOT+SC was generally less effective and more expensive regarding limb salvage. The ICER in the Wagner III/IV group was €19,005 (Figure 4, BCA 95% CI, -18,487–264,334) per limb saved, showing a trend that HBOT+SC was more effective, but also more expensive regarding limb salvage.

Mean number of QALYs during follow-up in the Wagner II group was 0.58 in the HBOT+SC group compared to 0.54 in the SC group (RD 0.04, BCA 95% CI, -0.09–0.17). In the Wagner III/IV group the mean QALY during follow-up was 0.51 in the HBOT+SC group compared to 0.59 in the SC group (RD -0.08, BCA 95% CI, -0.22–0.07). An ICUR of €70,985 (Figure 5, BCA 95% CI, -90,987–17,809,244) was found for patients with Wagner II, meaning HBOT+SC was generally more effective and more expensive regarding quality of life. For Wagner III/IV the ICUR was minus €55,556 (Figure 6, BCA 95% CI, -3,911,072–104,704), meaning HBOT+SC was generally less effective and more expensive regarding quality of life.

Discussion

This cost-effectiveness analysis of the $DAMO_2CLES$-trial, the largest study on HBOT for DFU patients at present shows no significant differences in cost-effectiveness and cost-utility for adding HBOT to standard care for patients with ischaemic DFUs. However, Wagner III/IV patients might benefit from additional HBOT in terms of limb salvage, at the cost of €19,005 per limb saved. Although this was a non-significant estimate, the extra costs of HBOT may be acceptable for limb salvage from a societal point of view in Western countries.[24] Our study also shows no benefit to treat Wagner II ischaemic DFUs with HBOT and therefore current guidelines should not recommend HBOT for such wounds.[25]

Although no difference was found in health status between the two treatment groups, the cost-utility analyses suggest that HBOT generally was more expensive while yielding less benefit in terms of QALYs, both overall and for Wagner III/IV patients in particular. Thus, only a minority of patients would benefit from additional HBOT. A possible explanation could be that quality of life in patients with a DFU may also improve after a major amputation, irrespective of additional HBOT treatment.[26]

Table 1

Baseline characteristics; BMI, body mass index; HBOT – hyperbaric oxygen therapy; SC – standard care; SD – standard deviation; TIA – transient ischaemic attack; *including angioplasty, myocardial infarction, or previous coronary intervention; **not requiring dialysis

Parameter	HBOT+SC (n = 60)	SC (n = 60)
Mean age, years, mean (SD)	67.6 (10.0)	70.6 (11.2)
Sex, male *n* (%)	51 (85)	46 (77)
BMI, $kg \cdot m^{-2}$, mean (SD)	28.3 (6.0)	27.1 (4.8)
Haemoglobin level, $mmol \cdot L^{-1}$, mean (SD)	7.8 (1.2)	7.4 (1.1)
Wound dimension and duration		
Wound diameter, cm, mean (SD)	3.2 (2.7)	3.5 (2.9)
Wound diameter < 3 cm, *n* (%)	34 (57)	33 (55)
Wound diameter > 3 cm, *n* (%)	26 (43)	27 (45)
Wound duration, months, mean (SD)	5.6 (6.4)	6.0 (6.8)
Wound classification, *n* (%)		
Wagner grade II	27 (45)	35 (58)
Wagner grade III	20 (33)	16 (27)
Wagner grade IV	13 (22)	9 (15)
Index wound location, *n* (%)		
Toe	30 (50)	31 (52)
Foot (below ankle)	23 (38)	19 (32)
Forefoot after amputation	6 (10)	9 (15)
Above ankle	1 (2)	1 (2)
Diabetes type 2	54 (90)	52 (87)
Duration of diabetes in years, mean (SD)	16.6 (11.2)	18.8 (15.1)
Peripheral arterial circulation parameters, mean (SD) mmHg		
Mean absolute ankle systolic blood pressure	110 (43)	102 (61)
Mean absolute toe systolic blood pressure	45 (30)	41 (35)
Mean foot dorsum transcutaneous oxygen pressure	23 (15)	23 (17)
Amenable for revascularization at inclusion, *n* (%)		
Total	25 (42)	24 (40)
Endovascular	22 (88)	19 (79)
Bypass	3 (12)	4 (17)
Endarterectomy + endovascular revascularization	0 (0)	1 (4)
Previous procedures index limb, *n* (%)		
Peripheral arterial revascularization	38 (63)	33 (55)
Minor amputation	20 (33)	23 (20)
Mobility, *n* (%)		
Walking	27 (45)	21 (35)
Moderately disabled	23 (38)	34 (57)
Wheelchair dependent	9 (15)	5 (8)
Bedridden	1 (2)	0 (0)
Smoking status, *n* (%)		
Non-smoker	13 (22)	14 (23)
Former	34 (57)	33 (55)
Current	13 (22)	13 (22)
Comorbidity, *n* (%)		
Hypertension	39 (65)	45 (75)
Cardiovascular heart disease*	20 (33)	28 (47)
Previous TIA or stroke	8 (13)	6 (10)
Distal neuropathy	32 (53)	41 (68)
Nephropathy**	8 (13)	12 (20)
Retinopathy	17 (28)	24 (40)
Medication *n* (%)		
Insulin	41 (68)	41 (68)
Oral antidiabetic medication	43 (72)	45 (75)
Statins	44 (73)	47 (78)
Antibiotics	22 (37)	24 (40)
Antihypertensive medication	44 (73)	41 (68)
Anticoagulants	45 (75)	45 (75)

Table 2

Volumes and cost per treatment allocation group; CI – confidence interval; DFU – diabetic foot ulcer; HBOT – hyperbaric oxygen therapy; ICU – intensive care unit; km – kilometre; PVD – peripheral vascular disease; SC – standard care; * Calculated as distance or cost (€0.19·km^{-1}) x 2 (return journey) x number of sessions; **Due to patients who crossed over to HBOT

Parameter	HBOT+SC		SC	
	Volume	Costs	Volume	Costs
HBOT treatment				
Hyperbaric oxygen treatment sessions	1,621	€293,401	149**	€26,969
Distance (km) and cost*	1,223.9	€13,624	153.6	€2,094
Subtotal HBOT treatment cost		**€307,025**		**€29,063**
Mean cost per patient (95% CI)	**€5,117 (4,312–5,917)**		**€484 (94–986)**	
Mean difference HBOT+SC - SC (95% CI)	**€4,633 (3,704–5.520)**			
In-patient hospital care (excluding HBOT treatment)				
(Re)admissions without surgery				
Max. 5 days admission for PVD	13	€25,025	8	€15,400
6–28 days admission for PVD	25	€156,625	12	€75,180
More than 28 days admission for PVD	2	€4,800	4	€9,600
Max. 5 days admission for DFU (incl. day care)	6	€36,870	7	€43,050
6–28 days admission for DFU	113	€46,330	363	€148,830
ICU stay per day (incl. diagnostics and medication)	22	€44,792	6	€12,216
Surgery or endovascular treatments				
For PVD with hospital stay	7	€70,665	11	€111,045
For DFU with hospital stay	6	€47,070	3	€23,535
Percutaneous angioplasty	19	€56,620	20	€59,600
Minor amputation with hospital stay for DFU	25	€180,250	29	€209,090
Major amputation with hospital stay for DFU	7	€87,010	13	€161,590
Surgical treatment during outpatient visit	1	€550	0	€0
Outpatient hospital or out-of-hospital care				
Outpatient visits	329	€44,415	168	€22,680
Rehabilitation clinic per day after major amputation (standard six-week period)	7	€136,170	13	€253,890
Wound care at home during follow-up period per day	11,700	€117,000	11,774	€117,740
Subtotal healthcare cost		**€1,054,732**		**€1,263,446**
Mean cost per patient (95% CI)	**€16,958 (12,857–21,156)**		**€20,269 (16,155–24,604)**	
Mean difference HBOT+SC - SC (95% CI)	**-€3,311 (-9,767–3,130)**			
Out-of-pocket expenses				
Out of pocket expenses (pharmacy/wound care)	***	€2,049	***	€3,047
Transportation				
Transportation to outpatient hospital visits	***	€2,243	***	€1,091
Subtotal		**€4,292**		**€4,138**
Mean cost per patient (95% CI)	**€71.53 (33.09–117.86)**		**€68.97 (30.51–124.78)**	
Mean difference HBOT+SC – SC (95% CI)	**€2.57 (-68.24–82.44)**			
Overall cost		**€1,328,782**		**€1,249,326**
Mean total cost per patient (95% CI)	€22,146 (17,851–26,364)		€20,822 (16,620–25,232)	
Mean difference HBOT+SC – SC (95% CI)	**€1,324 (-5,175–8,013)**			

A previous cost-effectiveness analysis on a small sample of 18 patients for ischaemic DFUs estimated a potential cost saving of £2,960 for each patient treated with HBOT.[16] This study, however, did not provide a confidence interval or information whether this outcome was statistically significant. In addition, only costs for wound dressings and HBOT were part of this analysis. Two other studies both performed cost-effectiveness analyses on hypothetical cohorts based on data of earlier studies.[14,15] Both concluded that HBOT is cost-effective. However, these results were based on studies that did not have the same time horizon of 12 months as was used for the cost-effectiveness analyses. Moreover, the hypothetical cohorts were based on older studies with lower methodological quality. Also, these studies did not distinguish between ischaemic and non-ischaemic DFUs, while later studies showed these conditions should be discerned.[27] A strong feature of the current study is that we included only patients with ischaemic DFUs and were able to retrieve the costs on an individual basis rather than based upon statistical modelling.

STUDY LIMITATIONS

An important factor to consider is that the cost-effectiveness results are solely based on data from Dutch hospitals. The costs of treatment (including HBOT) might differ considerably from other countries, based on national guidelines and health insurances. Also, the optimum

Table 3

Outcomes of the intention-to-treat analysis; BCA – bias-corrected and accelerated bootstrap; HBOT – hyperbaric oxygen therapy; ICER – incremental cost-utility ratio; QALY – quality-adjusted life years; SC – standard care

Parameter	HBOT+SC *n* = 60	SC *n* = 60
Mean QALY (BCA 95% CI)	0.54 (0.48–0.60)	0.56 (0.49–0.63)
Wagner II	0.58 (0.47–0.67)	0.54 (0.44–0.62)
Wagner III / IV	0.51 (0.42–0.60)	0.59 (0.47–0.70)
Mean difference per QALY (BCA 95% CI)	-0.02 (-0.11–0.08)	
Wagner II	0.04 (-0.09–0.17)	
Wagner III / IV	-0.08 (-0.22–0.07)	
Mean cost (BCA 95% CI)	€26,228 (21,229–32,644)	€22,437 (18,141–27,407)
Wagner II	€25,423 (18,058–35,224)	€22,369 (16,182–29,904)
Wagner III / IV	€26,886 (20,466–36,418)	€22,532 (16,980–28,215)
Mean difference in cost (BCA 95% CI)	€ 3,791 (-3,251–11,138)	
Wagner II	€ 3,055 (-7,463–14,380)	
Wagner III / IV	€ 4,354 (-4,417–14,492)	
Mean cost per QALY (BCA 95%CI)	€-227,035 (-361,569,550– -52,588)	
Wagner II	€70,985 (-90,987–17,809,244)	
Wagner III / IV	€-55,556 (-3,911,072–104,704)	
Amputations	12%	22%
Wagner II	15%	14%
Wagner III / IV	9%	32%
Mean cost per limb saved (BCA 95%CI)	€37,912 (-112,188–1,063,561)	
Wagner II	€-577,390 (-16,922,632– -468,585)	
Wagner III / IV	€19,005 (-18,487–264,334)	

Figure 1

Cost-effectiveness plane cost per limb saved

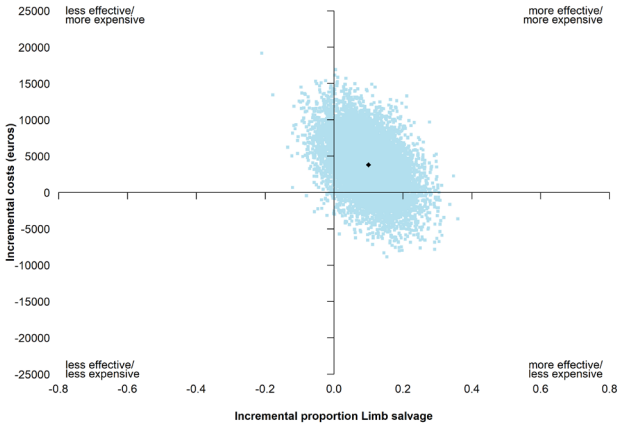
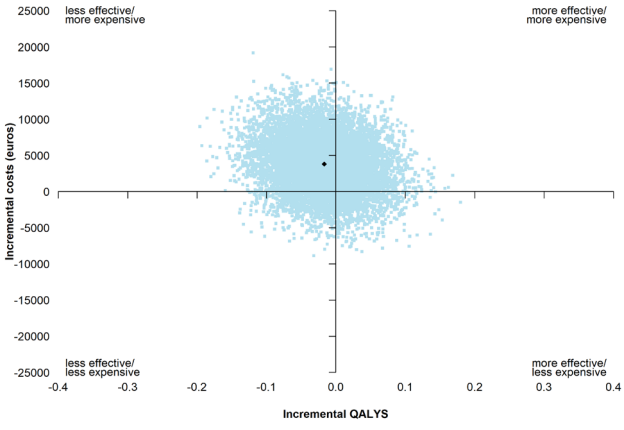


Figure 2

Cost-effectiveness plane cost per QALY



number of HBOT treatments to reach an effect is still not known. The current consensus from the commonly used guidelines suggests at least 30 HBOT sessions.[7] Currently, the DIONYSIUS study is being performed to assess these outcomes and the minimal number of HBOT treatments that is needed to achieve these outcomes.[28]

Furthermore, if HBOT is widely implemented, the costs per treatment might become lower and the accessibility of centres might improve. On the other hand, the burden for the patients increases with a larger number of treatments, taking up to two hours daily for five days a week and adding up to considerable traveling times, which could decrease adherence to treatment. This notion should stimulate healthcare professionals to apply shared decision-making when deciding about HBOT as a treatment option.

The DAMO$_2$CLES trial was powered to detect a difference in wound healing and limb salvage, and to account for health status and quality of life. Therefore, our (subgroup) analyses

Table 4

Outcomes of the per-protocol analysis A; BCA – bias-corrected and accelerated bootstrap; HBOT – hyperbaric oxygen therapy; ICER – incremental cost-utility ratio; QALY – quality-adjusted life years; SC – standard care

Parameter	HBOT+SC *n* = 39	SC *n* = 81
Mean QALY (BCA 95% CI)	0.60 (0.52–0.68)	0.53 (0.47–0.58)
Wagner II	0.59 (0.43–0.71)	0.54 (0.47–0.61)
Wagner III / IV	0.61 (0.52–0.71)	0.51 (0.41–0.60)
Mean difference per QALY (BCA 95% CI)	0.08 (-0.03–0.17)	
Wagner II	0.05 (-0.12–0.19)	
Wagner III / IV	0.11 (-0.02–0.24)	
Mean cost (BCA 95% CI)	€25,681 (20,967–32,476)	€23,682 (19,441–28,862)
Wagner II	€28,493 (20,279–40,144)	€21,737 (16,107–28,526)
Wagner III / IV	€23,272 (18,450–31,600)	€25,995 (20,060–34,787)
Mean difference in cost (BCA 95% CI)	€1,999 (-5,004–9,725)	
Wagner II	€6,755 (-3,609–19,400)	
Wagner III / IV	€-2,723 (-12,040–6678)	
Mean cost per QALY (BCA 95% CI)	€25,573 (-139,582–940,894)	
Wagner II	€132,124 (-28,845–131,559,363)	
Wagner III / IV	€-25,560 (-441,174–192,918)	
Amputations	5%	22%
Wagner II	6%	18%
Wagner III / IV	5%	27%
Mean cost per limb saved (BCA 95% CI)	€11,694 (-24,710–131,986)	
Wagner II	€53,501 (-50,697–1,378,383)	
Wagner III / IV	€-12,232 (-62,126–64,353)	

Table 5

Outcomes of the per-protocol analysis B; BCA – bias-corrected and accelerated bootstrap; HBOT – hyperbaric oxygen therapy; ICER – incremental cost-utility ratio; QALY – quality-adjusted life years; SC – standard care

Parameter	HBOT+SC *n* = 49	SC *n* = 71
Mean QALY (BCA 95% CI)	0.55 (0.47–0.62)	0.55 (0.49–0.61)
Wagner II	0.59 (0.45–0.69)	0.54 (0.46–0.62)
Wagner III / IV	0.51 (0.41–0.61)	0.57 (0.47–0.66)
Mean difference per QALY (BCA 95% CI)	-0.01 (-0.11–0.09)	
Wagner II	0.05 (-0.10–0.18)	
Wagner III / IV	-0.06 (-0.20–0.08)	
Mean cost (BCA 95% CI)	€27,948 (22,482–35,189)	€21,837 (17,800–26,505)
Wagner II	€30,289 (21,791–40,827)	€20,324 (14,890–27,100)
Wagner III / IV	€26,193 (19,737–36,959)	€23,905 (18,197–30,758)
Mean difference in cost (BCA 95%CI)	€6,111 (-1,135–14,367)	
Wagner II	€9,965 (-920–21,634)	
Wagner III / IV	€2,288 (-6,985–13,732)	
Mean cost per QALY (BCA 95% CI)	€-931,638 (-198,110,372– -502,704)	
Wagner II	€187,165 (-55,715–41,807,221)	
Wagner III / IV	€-37,476 (-2,380,683–222,074)	
Amputations	12%	20%
Wagner II	14%	15%
Wagner III / IV	11%	27%
Mean cost per limb saved (BCA 95% CI)	€81,771 (-146,080–4,581,121)	
Wagner II	€2,859,970 (-5,087,884–19,585,325)	
Wagner III / IV	€14,339 (-75,274–612,803)	

Figure 3
Cost-effectiveness plane cost per limb saved for the Wagner II subgroup

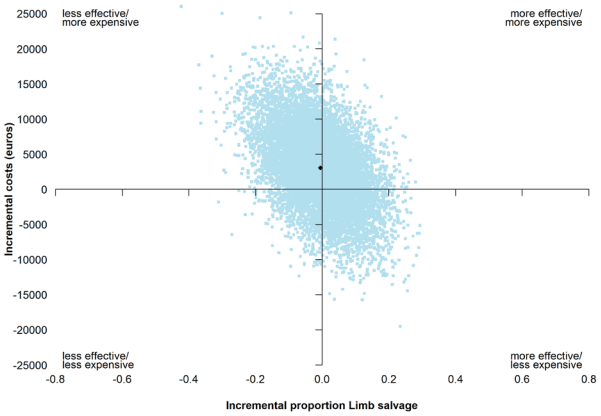


Figure 4
Cost-effectiveness plane cost per limb saved for the Wagner III/IV subgroup

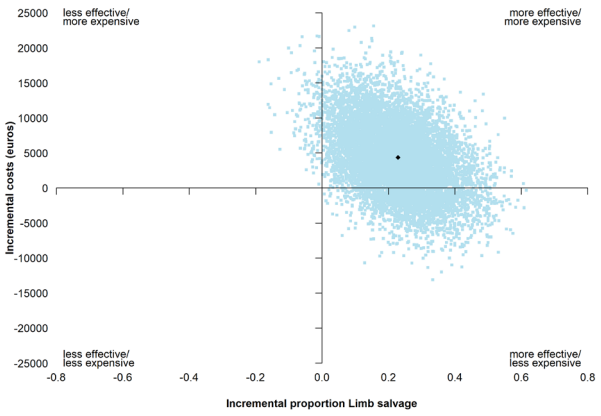


Figure 5
Cost-effectiveness plane cost per QALY for the Wagner II subgroup

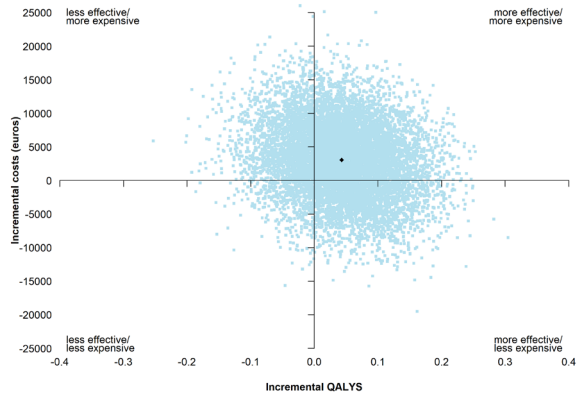
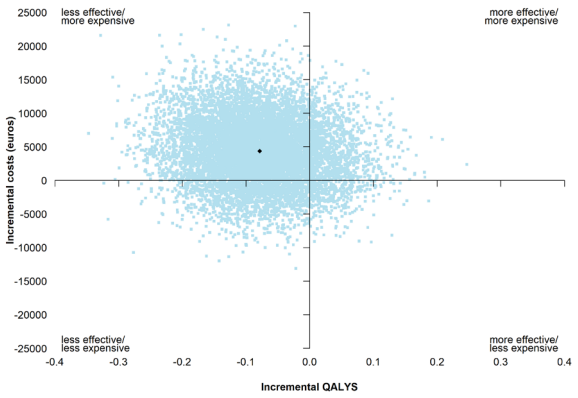


Figure 6
Cost-effectiveness plane cost per QALY for the Wagner III/IV subgroup



may be underpowered and mask the effects of additional HBOT treatment. However, the trend found towards a higher limb salvage rate in the Wagner III/IV subgroup in the post-hoc analysis is clinically relevant and advocates further research with sufficient power to obtain more evidence.

Another factor was that the compliance with HBOT was lower than expected based on earlier studies which adds to the possible underestimation of its effect in the current study. There was a considerable amount of missing data regarding the EQ5D which was accounted for by imputation of data. This might, however, may have skewed the results in either direction.

Conclusions

The current study showed no clear cost-effectiveness or cost-utility of additional HBOT compared to standard wound care to prevent amputation or improve health status of patients with ischaemic DFUs. However, patients with Wagner stage III or IV ulcers might benefit from adjunctive HBOT, which was not associated with higher costs than standard care.

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Hyperbaric oxygen therapy for idiopathic sudden sensorineural hearing loss: a cohort study of 10 versus more than 10 treatments

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Keywords
Dose; Hyperbaric research; Number of treatments; Outcomes

Abstract
(Laupland BR, Laupland KB, Thistlethwaite K. Hyperbaric oxygen therapy for idiopathic sudden sensorineural hearing loss: a cohort study of 10 versus more than 10 treatments. Diving and Hyperbaric Medicine. 2024 20 December;54(4):275–280. doi: 10.28920/dhm54.4.275-280. PMID: 39675734.)

Introduction: Current treatment of idiopathic sudden sensorineural hearing loss (ISSNHL) includes a combination of corticosteroids and hyperbaric oxygen therapy (HBOT) without established dose. The objective of this study was to investigate whether > 10 HBOT treatments offers improved outcome over 10 treatments.

Methods: A retrospective chart review was performed of patients treated with HBOT for ISSNHL between 2013 and 2022 at the Royal Brisbane and Women's Hospital. Pure tone average results from 500, 1,000, 2,000, 4,000 hertz (PTA4) were obtained pre-treatment, after treatment 10, and six weeks post-treatment.

Results: There were 479 patients treated for ISSNHL: 144 having audiograms six weeks post-treatment, 140 of whom also had an audiogram after treatment 10. At six weeks post treatment 22% (32/144) had normal hearing (PTA4 < 25 dB), and 69% (99/144) had a PTA4 gain ≥ 10 dB. At the treatment 10 audiogram, 83/140 (59%) were improved. From these, 5/21 (24%) with 10 treatments and 14/57 (25%) with > 10 treatments had a further PTA4 gain of ≥ 10 dB occurring after treatment 10. For those 57/140 (41%) not improved at treatment 10, 7/26 (27%) with 10 treatments and 12/31 (39%) with > 10 treatments were improved at six weeks post-treatment with 5/7 (71%) and 8/12 (67%) of the 10 and > 10 groups respectively having ≥ 10 dB gain in PTA4 occurring after treatment 10. Overall, there was no significant difference in mean (SD) hearing gain from treatment 10 to six weeks post treatment between the 10 treatments and > 10 treatments groups: 4.73 (8.90) versus 5.93 (11.25) dB, $P = 0.53$.

Conclusions: In conjunction with steroids, 10 treatments of hyperbaric oxygen therapy appear to offer equivalent benefit to > 10 treatments. Similar improvements in PTA4 and hearing recovery occur after 10 HBOT treatments independent of ongoing HBOT. A prospective trial comparing 10 versus > 10 treatments for ISSNHL with outcome measured beyond treatment completion is warranted.

Introduction

Idiopathic sudden sensorineural hearing loss (ISSNHL) is defined as an unexplained hearing loss of at least 30 dB in three consecutive frequencies on the audiogram which manifests within three days.[1–3] This occurs in between two and 30 people per 100,000 population depending on geographical area, and its incidence appears to be increasing.[4–8] Although the natural history of this form of hearing loss is not well defined, several studies have reported that 30–65% of cases will improve spontaneously, with the majority improving in the first two weeks following the loss.[9–11]

Given the consequences of hearing loss and lack of a unifying causative factor, many treatment options have been trialled including corticosteroids, vasodilators, vitamins, anticoagulants, and hyperbaric oxygen therapy (HBOT). The 2019 American Academy of Otolaryngology-Head and Neck Surgery clinical practice guidelines for sudden hearing loss suggest that a combination of HBOT and steroids started within 14 days provides the best opportunity for recovery,[3] with rates of improvement being around 60–70% with this regimen.[12–16]

Despite the recommendation for use of HBOT, no optimal duration of this therapy has been defined. In a survey of European hyperbaric centres in 2016, the number of treatment sessions varied from five to 40.[17] The Undersea and Hyperbaric Medical Society (UHMS) guidelines currently recommend 10 to 20 daily HBOT sessions for the treatment of ISSNHL[18] and many units utilise audiograms

at 10 treatments to determine the utility of further sessions and to aid with resource allocation.[19]

Given the cost, risk for complications, and inconvenience to patients, it is important to know whether more than 10 HBOT treatment sessions provide additional clinical benefit. To our knowledge, no studies have directly investigated this question. The objective of this study was therefore to investigate whether completion of more than 10 HBOT treatments among patients with ISSNHL is associated with improved outcomes.

Methods

The project was reviewed and approved by the Townsville Hospital and Health Service ethics review board and found to be exempt from full ethics review as it is considered negligible risk research. (EX/2022/QTHS/88690 [Aug ver 2]).

The Royal Brisbane and Women's Hospital Hyperbaric Medicine Unit currently treats ISSNHL with 10 to 20 sessions of 80 minutes breathing oxygen at 243 kPa (2.4 atmospheres absolute pressure) with a five-minute mid-session air-break (40 minutes – five minutes – 40 minutes). Our unit routinely accepts patients diagnosed with ISSNHL for treatment within 14 days of onset of hearing loss, and for salvage therapy up to 30 days from the loss. Occasional patients are accepted outside these timeframes or for less severe loss if there are extenuating circumstances such as new hearing loss in the only functioning ear or occupational dependence on hearing.

All patients are concurrently followed by an ear, nose and throat specialist and are treated with oral or intratympanic steroid unless there is a contraindication. An audiogram is routinely performed prior to the start of HBOT and after treatment 10. Following this, an individualised decision is made to continue or end HBOT based on improvement in the pure tone average scale (PTA4), speech discrimination, and patient factors.

This study used a retrospective cohort design and initially included all patients treated in our unit from January 2013 – December 2022. Charts were obtained from the clinic database using the code for ISSNHL. Only patients who had an audiogram performed six weeks following the end of HBOT were included in the final study. Patients were also excluded if they had not completed at least eight treatments. Number of treatments were defined as '10 treatments' for eight to 12 HBOT sessions (some patients had one or two sessions past 10 while awaiting audiology), and '> 10 treatments' for 13 or more sessions. Improvement in hearing was defined as ≥ 10 dB gain in the four frequency PTA4 between audiograms. Normal hearing was defined as PTA4 of < 25 dB.

Data were analysed using Stata 17.1 (StataCorp LLC, College Station, USA). Analysis was primarily descriptive. Prior to analysis, continuous variables were assessed for their underlying distribution using histograms. Normally or near normally distributed variables were described using means and standard deviations (SD) and were compared using *t*-tests. Skewed variables were described using medians with interquartile ranges (IQR) and compared using the Wilcoxon signed-rank test. Differences between patient measures over time were compared using paired *t*-tests. Categorical data were compared using Fisher's exact test. *P*-values less than 0.05 were considered significant.

Results

During the study period, 479 patients were treated for ISSNHL of which 144 patients fulfilled study inclusion criteria. Four patients did not have audiograms after treatment 10 and were excluded from the flow chart aspect of the analysis.

The mean (SD) age was 53 (16.0) years and 68 (47%) were male. The median (IQR) time from hearing loss to start of treatment was 10 (5–16) days. The majority (65%) of those treated had severe (> 60 dB) loss. The mean (SD) PTA4 pre-treatment was 71.54 (26.37) dB and the six-week post-treatment PTA4 was 50.36 (28.03) dB. At six weeks post-treatment, 22% (32/144) of patients had normal hearing and 69% (99/144) of patients had improved hearing.

Comparison characteristics of the '10 treatments' versus

Table 1
Comparison of patient characteristics in '10' and '> 10' treatment groups; IQR – interquartile range; SD – standard deviation

Parameter	10 treatments (*n* = 48)	> 10 treatments (*n* = 96)	*P*
Male *n* (%)	24 (50%)	44 (46%)	0.7
Age (years) Mean (SD)	50 (15.33)	54 (16.22)	0.13
Days from hearing loss to treatment initiation Median (IQR)	8.5 (4–20)	10 (5–15)	0.80
Baseline PTA4 (dB) Mean (SD)	68.7 (26.6)	73.0 (26.3)	0.29
Final PTA4 (dB) Mean (SD)	50.8 (34.8)	50.1 (24.2)	0.89
Final hearing gain (dB) Mean (SD)	17.9 (22.6)	22.8 (19.2)	0.17
Gain ≥ 10 dB to final *n* (%)	29 (60%)	70 (73%)	0.13
Normal final hearing *n* (%)	15 (31%)	17 (18%)	0.09

Table 2
Hearing improvement by degree of loss; PTA4 – four frequency pure tone average hearing loss (dB); SD – standard deviation

Degree of loss	Initial PTA4 (dB)	*n* (%)	Gain to six weeks post-treatment (dB) Mean (SD)	*P*
Mild	≤ 40	22 (15)	9.62 (9.42)	< 0.001
Moderate	41–60	28 (20)	16.28 (17.02)	
Severe	61–80	39 (27)	19.79 (19.58)	
Profound	> 80	55 (38)	29.28 (22.80)	

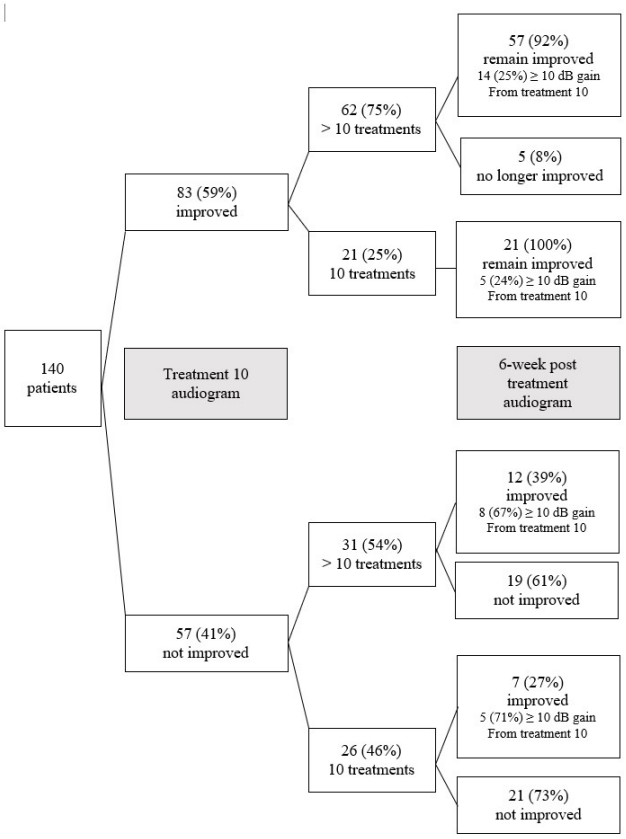
'> 10 treatments' groups can be found in Table 1. The mean (SD) number of treatments in the '10 treatments' group was 10.02 (0.81) and the '> 10 treatments' group was 17.54 (3.77).

Hearing gain arranged by degree of loss can be seen in Table 2 which shows increasing gains in PTA4 for patients with more severe hearing loss.

In examining improvement of hearing by number of treatments, there were 140 patients with audiograms after treatment 10. Of these, 93/140 (66%) patients were considered to have '> 10 treatments' and 47/140 (34%) '10 treatments'. Comparing these two groups at treatment 10, significantly more patients in the '> 10 treatments' group had improved hearing than in the '10 treatments' group: 62/93 (67%) versus 21/47 (45%), $P = 0.018$. However, when comparing these groups from treatment 10 to six weeks post-treatment, similar percentages of patients were improved for both groups: 24/93 (26%) of '> 10 treatments' versus 10/47 (21%) of '10 treatments' $P = 0.68$. In addition, there was no significant difference in the mean (SD) PTA4 gain after treatment 10 for either group: 5.93 (11.25) dB for '> 10 treatments' versus 4.73 (8.90) dB for '10 treatments', $P = 0.53$.

Figure 1 shows a flow chart analysis which initially divides the 140 patients into 'improved' or 'not improved' according to their treatment 10 audiogram. These two groups are then subdivided into '10 treatments' and '> 10 treatments' and further examined for improvement at six weeks. Of those 83/140 (59%) considered improved at treatment 10, 57/62 (92%) of the '> 10 treatments' and 21/21 (100%) of the '10 treatments' remained improved at six weeks with 14/57 (25%) and 5/21 (24%) of each group respectively having a ≥ 10 dB PTA4 gain between treatment 10 and six week follow up. Of the 57/140 (41%) patients not improved at treatment 10, 12/31 (39%) of the '> 10 treatments' and 7/26 (27%) of the '10 treatments' were considered improved at six weeks, with 8/12 (67%) and 5/7 (71%) of these respectively having a ≥ 10 dB gain in PTA4 occurring after the treatment 10 audiogram.

Figure 1
Flow chart showing improved (PTA4 gain ≥ 10 dB) hearing in 140 patients divided into '10 treatment' and '> 10 treatment' groups based on improvement at treatment 10 and examined for further improvement at six weeks



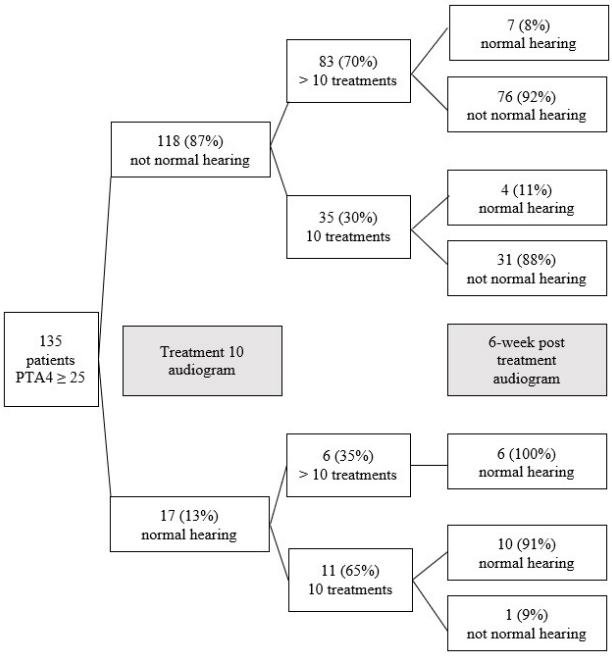
In examining normalisation of hearing, there were 135 patients with audiograms post 10 treatments who did not have normal (PTA4 < 25 dB) hearing on initial audiogram. Of these 46/135 (34%) had '10 treatments' and 89/135 (66%) had '> 10 treatments'. There was a significant difference in normal hearing between groups at the treatment 10 audiogram: 11/46 (24%) for '10 treatments' versus 6/89 (7%) for '> 10 treatments', $P = 0.006$.

Figure 2 is a flow chart analysis for normalisation of hearing. It divides patients into 'normal' and 'not normal' hearing following the treatment 10 audiogram. These groups are then sub-divided into '10 treatments' and '> 10 treatments' and further evaluated for normal hearing at six weeks post-treatment. Of the group of 118/135 (87%) with non-normal hearing at treatment 10, there was no significant difference in normalisation of hearing at the six week post treatment audiogram between those having '10 treatments' versus '> 10 treatments': 4/35 (11%) versus 7/83 (8%), $P = 0.73$.

Discussion

In our study, 99/144 (69%) patients with ISSNHL improved and 32/144 (22%) had normalised hearing when treated with

Figure 2
Flow chart of 135 patients with abnormal initial hearing divided into '10 treatments' and '> 10 treatments' groups depending on normal hearing (PTA4 < 25) at treatment 10 and examined for normal hearing at six weeks



a combination of hyperbaric oxygen therapy and steroid treatment. Those with severe to profound loss showed the most gains. We found no significant difference at six weeks post treatment between the '10 treatments' and '> 10 treatments' groups in either improved hearing, normal hearing, or overall hearing gain. Uniquely, we show similar ongoing gains in both hearing improvement and recovery of normal hearing from treatment 10 onward independent of further HBOT sessions.

Given the high spontaneous recovery rates of untreated ISSNHL, the utility of treating ISSNHL with any modality has been questioned. However, these spontaneous recovery rates are quite inconsistent between studies and likely the result of different inclusion criteria and differing definitions of ISSNHL and recovery. Along with these confounders, the ubiquitous use of corticosteroids as treatment has made placebo control groups rare in recent studies which may have more homogeneous definitions.

Two recent meta-analysis have tried to better define the natural history of ISSNHL with results that are difficult to interpret. Chashu et al. in 2023 performed a meta-analysis for spontaneous rates of recovery in studies of hearing loss treatments that included a placebo group.[20] They found an overall recovery rate of 60.3% CI 33.9–79.9%, with large heterogeneity between included studies ($I^2 = 86\%$). However, when they limited their analysis to those studies with a standard definition of ISSNHL (a loss of ≥ 30 dB in three consecutive frequencies occurring in < 3 days), they found lower, slightly narrower improvement rates of 33–54%. Ying et al. in a 2024 meta-analysis, found a mean hearing gain of up to 24 dB (95% CI, 2.6–45.4, $P = 0.03$) in untreated patients at 2–3 months post-loss with a heterogeneity of $I^2 = 88.4\%$.[21] The authors acknowledge the large variations across included studies limit their conclusions. Our study uses Chashu's standard definition of ISSNHL with an improvement rate of 69% suggesting there is benefit to our treatment.

Supporting the overall use of HBOT for ISSNHL are the international consensus (ICON) on treatment of sudden hearing loss 2018's methodological recommendations and two meta-analyses comparing the addition of HBOT to standard medical treatment including steroids.[22] The ICON group recommend that any new treatment for hearing loss should provide better results than steroids, and that a hearing gain in PTA of ≥ 10 dB be considered an improvement. Joshua et al. in their 2022 meta-analysis found that mean PTA4 gain, final PTA4 and hearing recovery were all significantly improved in the HBOT group.[23] The mean difference in absolute hearing gain between groups was 10.3 dB (95% CI, 6.5–14.1) in favour of the HBOT group with a heterogeneity of $I^2 = 0\%$ lending additional weight to this result. Another meta-analysis in 2018 had more heterogeneity, but also significantly favoured HBOT + medical treatment over medical treatment alone for complete hearing recovery, any hearing recovery and absolute hearing gain.[24] These studies would suggest that HBOT does provide benefit for ISSNHL.

In comparing numbers of HBOT treatments for ISSNHL there is no clear consensus in the literature. Korpinar et al. in 2011 retrospectively analysed 80 patients undergoing twice daily HBOT.[25] Patients received between five and 31 treatments (mean 18.2) over an average of 10.4 days. They concluded that higher numbers of HBOT sessions improved hearing gains. Sherlock et al. in 2016, as a part of a retrospective review of 76 patients who received both steroids and daily HBOT treatments, analysed patients who had ≤ 10 treatments versus > 10 (mean = 14) and found no significant change in hearing gain between the two groups.[12] Another retrospective examination of 178 patients who had undergone between four and 34 sessions of HBOT (mean 16.8) twice daily found in their univariate analysis that the recovery group (gain > 15 dB) had fewer treatments than the no recovery group (14.9 versus 17.8).[7] However in their multivariate analysis, they demonstrated that the number of HBOT sessions was not a factor in hearing recovery and concluded that 20 sessions is enough to show therapeutic effect. Finally, Chin et al. in 2022 retrospectively studied 102 patients who had undergone 1–5 sessions of HBOT and compared them to 46 patients who had undergone 6–10 sessions.[26] They found that 6–10 sessions did not provide further improvement over the shorter treatment group.

Of note, none of these studies utilised a similar point in time for their final audiometric outcome or examined audiograms beyond the completion of therapy. Korpinar et al., Sherlock et al. and Wu et al. made decisions to terminate HBOT based on audiological follow-up with audiograms which occurred at different time points depending on the number of HBOT sessions given.[7,12,20] Chin et al. specifically compared audiometry after 1–5 sessions with after 6–10.[21] If the gain seen in our study after 10 treatments regardless of further treatment is reproducible, it would suggest bias in these studies towards longer treatments.

The ongoing hearing gain following completion of treatment evidenced in our results is also evident in several other studies. Rauch et al. in a prospective, randomised comparison of oral versus intra-tympanic steroids, demonstrated ongoing improvement in audiograms that was significant out to two months and that stabilised at six months after the start of treatment.[27] Cho et al. prospectively looked at patients treated with oral and intratympanic steroids with and without the addition of 10 sessions of HBOT.[28] They too demonstrated ongoing improvement in both groups, continuing beyond the 10 days of treatment and stabilising two to three months post-treatment. Yildrim et al. retrospectively found similar results in patients treated with 20 daily HBOT sessions.[29] Like these, our study shows similar trends of improvement out to two months after initiation of treatment. Uniquely, we suggest that this improvement is not impacted by further HBOT sessions.

Continued improvement 2–3 months from the initiation of treatment, and similar gains seen after 10 treatments regardless of ongoing hyperbaric sessions implies that one must be cautious in interpreting results from studies comparing different numbers of HBOT treatments without final audiograms done at a similar timepoint from the start of treatment. If the final audiometric outcome after a shorter treatment course were measured and compared to one after a longer treatment course, the results may favour longer treatment times as being more efficacious. Future studies comparing efficacy of treatment durations should assess outcome of treatment at similar time frames, preferably at least 12 weeks from initiation of treatment.

Although our study is suggestive of 10 treatments being equivalent to > 10, it is limited in that it is retrospective, not randomised, and relatively small in numbers. As our study was not controlled, and patients underwent different timings of adjunctive treatments from their otolaryngologist, it is possible that some of the ongoing gain seen post HBOT may have been due to treatment with intratympanic steroids occurring after the completion of HBOT. It is also possible that our inclusion criteria of a six-week post-treatment audiogram selected for patients who had had improvement in their hearing and were interested in outcome which may skew our result toward improvement even for those who had fewer treatments.

Conclusions

In conjunction with steroids, 10 HBOT treatments appear to offer equivalent benefit to more than 10 treatments. Similar improvements in PTA4 gain and hearing recovery occurs after 10 HBOT treatments regardless of whether HBOT is continued. A prospective study of 10 versus 20 treatments is warranted. All studies comparing numbers of HBOT treatments should consider an outcome beyond the completion of HBOT.

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Occurrence and resolution of freediving-induced pulmonary syndrome in breath-hold divers: an online survey of lung squeeze incidents

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Keywords
Barotrauma; Pulmonary barotrauma; Pulmonary edema; Pulmonary oedema; Survey

Abstract
(Yu E, Dong GZ, Patron T, Coombs M, Lindholm P, Tillmans F. Occurrence and resolution of freediving-induced pulmonary syndrome in breath-hold divers: an online survey of lung squeeze incidents. Diving and Hyperbaric Medicine. 2024 20 December;54(4):281–286. doi: 10.28920/dhm54.4.281-286. PMID: 39675735.)
Introduction: Breath-hold divers occasionally surface with signs of fluid accumulation and/or bleeding in air-filled spaces. This constellation of symptoms, recently termed 'freediving induced pulmonary syndrome', is thought to come from immersion pulmonary oedema and/or barotrauma of descent and is colloquially termed a 'squeeze'. There is limited understanding of the causes, diagnosis, management, and return to diving recommendations after a squeeze.
Methods: We developed an online survey that queried breath-hold divers on the circumstances and management of individual squeeze events.
Results: A total of 132 (94 M, 38 F) breath-hold divers filled out the survey. Most were recreational or competitive freedivers with mean age of 37 years old and nine years of experience. Of those, 129 (98%) held a certification in freediving from an accredited training agency. A total of 103 individuals reported 140 squeeze events from 2008–2023. The average depth at which a squeeze occurred was 43 m. The top contributors to lung squeezes were described as movement at depth, contractions, and inadequate warm-up. The most common symptoms of a squeeze were cough, sputum production, and fatigue. Divers were instructed to wait an average of two months before returning to diving after a squeeze. On average, divers were able to achieve the same depth of their squeeze event three months after the incident.
Conclusions: Inadequate warm-up, contractions, and abnormal movement at depth are the most reported causes for a squeeze. Most divers do not seek medical treatment after a lung squeeze event and can return to the same depth within three months.

Introduction

Breath-hold divers occasionally surface with signs of fluid accumulation and/or bleeding in air-filled spaces. This constellation of symptoms is thought to come from barotrauma of descent and is colloquially termed a 'squeeze'.[1,2] A mask squeeze results in subconjunctival haemorrhage[3] while a middle ear squeeze may result in tympanic membrane rupture.[1] Not all squeezes result in obvious bleeding. A sinus squeeze may cause epistaxis or be limited to sinus discomfort while a laryngeal squeeze may cause haemoptysis or be limited to voice changes.[1] A squeeze in the lung may result in more subtle symptoms and frank haemoptysis may be absent.

The pathophysiology of lung squeeze is poorly understood and thought to be a combination of factors including pulmonary vascular engorgement,[4] diaphragmatic contractions,[5] equalisation, and movement at depth. The constellation of symptoms is similar to immersion pulmonary oedema experienced by compressed air divers and surface swimmers.[6] Alveolar haemorrhage and interstitial oedema can both lead to impaired ventilation, resulting in respiratory discomfort, difficulty, or distress. It was recently suggested to encompass these symptoms under 'freediving induced pulmonary syndrome' (FIPS) as an umbrella term.[7] Auscultation, pulse oximetry,[8] and point-of-care ultrasound devices are the most commonly used tools to diagnose lung squeeze in a field setting.[2] In-hospital radiographs,[9] computed tomography,[10] and bronchoscopy can help aid in the diagnosis.[11] Squeezes are often self-limited, and therefore divers may not present for medical evaluation.

Since many competitive events have limitations on participation with recent dive injuries, mild symptoms may be underreported. As such, there are no universal clinical guidelines for returning to diving after a squeeze.

At present, this time frame is dictated by coaches, fellow divers, competition judges, medics, or health practitioners who may not be well-versed in freediving pathophysiology. This survey sought to explore the incidence of lung squeezes, medical management of squeeze symptoms, and return to diving after a squeeze.

Methods

Ethical approval was granted by the Institutional Review Board of Divers Alert Network (DAN) under IRB 033-23; data collection was open for eight weeks from 28 August to 25 October 2023.

An online survey was developed using REDCap and distributed to breath-hold divers through DAN's social media outlets (Facebook, Instagram, and Twitter). The study included divers 18 years and older. Participants were presented with a participant information page and this required them to indicate consent before progressing to the survey. Each diver's demographic information, training and experience in different breath-hold diving disciplines was collected, as well as symptoms of individual lung squeeze incidents, and medical care received if applicable. Divers were also asked to share their thoughts on what contributed to their lung squeeze incident.

Data were analysed using GraphPad Prism(R) 10. Descriptive statistics were reported for demographic information and dive experience calculating average with standard deviation or median with interquartile range.

All data were downloaded from REDCap to a database on a secure server at Divers Alert Network and identifying information (voluntarily provided contact information) was removed before datasets were analysed.

Results

RESPONDENTS

There were 164 submissions received, of which 27 were incomplete. Of the 137 full submissions, five datasets were identified as duplicates, leaving 132 datasets for analysis that were de-identified. Of the participants, 94 (71.2%) identified as male. Their ages ranged from 20–74, with a mean (standard deviation [SD]) age of 37 (SD 9) years and a median (interquartile range [IQR]) of 35 (IQR 31–42) years. Their experience ranged from under one to 45 years, with a mean of nine (SD 8) years and a median of 6.5 (IQR 3–11) years. They participated in the following breath-hold diving activities: recreational freediving (122), competitive freediving (89), spearfishing (36), underwater hockey (4), aquathlon (3), and underwater target shooting (2). All reported various training frequencies, ranging from daily (32, 24%) to weekly (60, 45%) to monthly (8, 6%) to seasonally (32, 24%).

Of the respondents, 129 (98%) were certified by one or more organisations. Certifying organisations included the Association Internationale pour le Développement de l'Apnée (AIDA) (68), Molchanovs (47), Scuba Schools International (SSI) (27), Confederation Mondiale des Activites Subaquatiques (CMAS) (21), Professional Association of Diving Instructors (PADI) (13), Apnea Academy (eight), and the following with five or less survey participants certified: Performance Freediving International, Apnea Total, Freediving Instructors International, National Association of Underwater Instructors, Rebreather Association of International Divers, Fédération Française d'Études et de Sports Sous-Marins, Apnea College, Apnea International, Professional Scuba Schools, and Pure Apnea. Levels of certification distinguished between pool (47), 10 m depth (18), 20 m depth (20), 30 m depth (38), 40+ m depth (61), instructor (82), instructor trainer (9), competitor (46), and safety diver (28). Of the respondents, 94 (71%) also reported being certified in self-contained underwater breathing apparatus (scuba).

Of the pertinent cardiopulmonary medical problems, five participants disclosed hypertension, 13 allergies, nine asthma, one unspecified congenital heart disease, and two reported a known patent foramen ovale. Regarding surgical procedures, one reported previous heart surgery, one chest surgery, and 22 had previous oral or otolaryngological surgery excluding dental work. A total of 127 (96.2%) of respondents reported having experienced a squeeze. The number of squeezes ranged from 1–200, with an average of eight (SD 20) and a median of 3 (IQR 1–5) squeezes.

SQUEEZE EVENTS

In total, 103 respondents filled out information regarding one or more squeeze events, totaling 140 events reported between 2008–2023 with 55% of events within the 12-months prior to completing the survey. The age at the time of the squeeze incidents ranged from 16–65, with an average of 35 (SD 8) years. The water temperature ranged from 3–37°C, with an average of 23.7°C (SD 6.3) and a median of 25°C (IQR 20–28). Wetsuit thickness ranged from 1–7 mm. The type of breath-hold diving at the time of the incident included training (98), recreational freediving (24), competition (10), safety (2), and fishing (2) (Figure 1). The discipline at the time of the incident included free immersion (51), constant weight (25), constant weight bifins (35), constant weight no fins (11), variable weight (3), and dynamic apnoea (1); there were 14 incidents in which a discipline was not specified (Figure 2).

Excluding the one incident that occurred during dynamic apnoea (swimming just below the surface), the depths of the dives resulting in a squeeze ranged from 10–113 m, with an average target and reached depth of 43 (SD 22) m and median of 38 (IQR 25–57) m (Figure 3). 24 (17%) of dives did not reach the target depth, while 8 (6%) exceeded the target depth. Of all the divers, 23 (16%) were pushing their

Figure 1
Diving type / activity performed during lung squeeze incidents

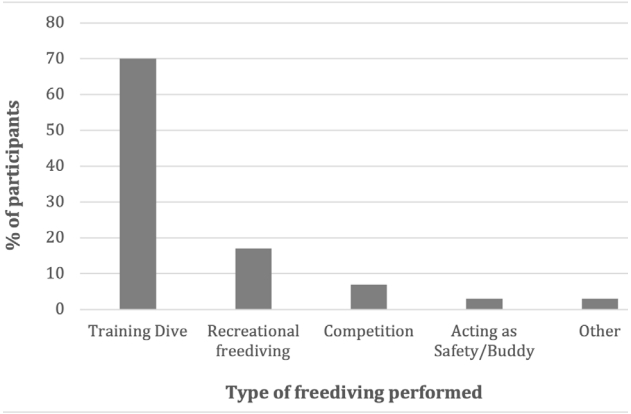


Figure 2
Diving discipline performed during lung squeeze incidents; CWTB –constant weight with bifins; CWT – constant weight with monofin; CNF – constant weight no fins; DYN – dynamic apnoea with monofin; FIM – free immersion; VWT – variable weight; 10% unspecified

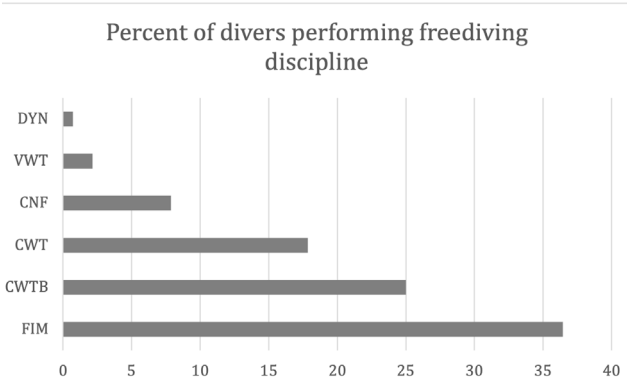


Figure 3
Achieved depth (metres) of dives resulting in a squeeze incident

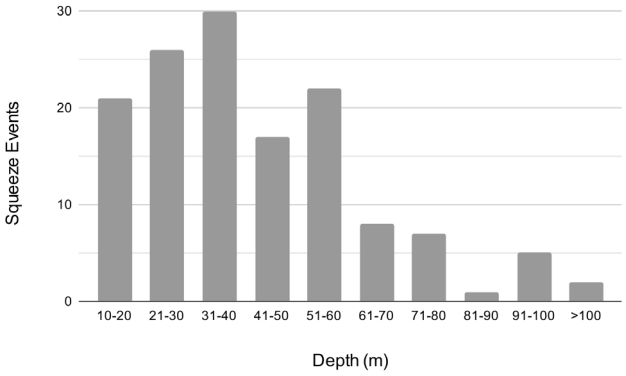


Figure 4
Achieved depth (metres) during squeeze event (grey dots) vs previous personal best depth (black solid line)

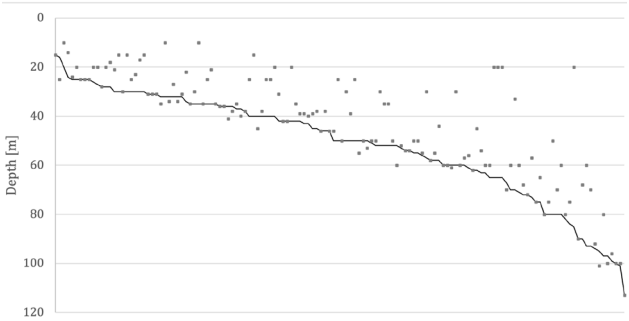
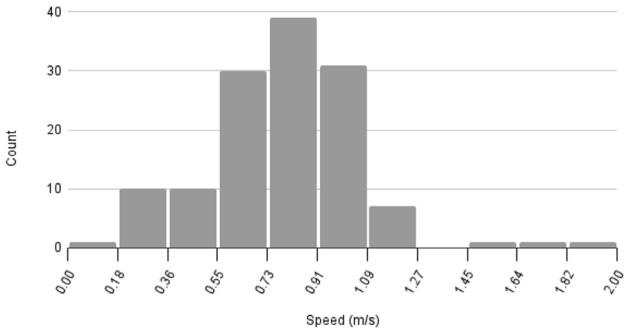


Figure 5
Calculated diving speed of squeeze incidents



personal best depth while 30 (22%) were trying to match a previous personal best depth. Of all the dives, 50 (36%) of dives matched or exceeded a previous personal best (Figure 4). The dive time ranged from 30 seconds to 3 minutes 50 seconds, with an mean of 1 minute 50 seconds. The mean speed of the divers who squeezed was 0.79 metres per second (Figure 5).

Equalisation methods used during the dives included mouthfill (75), reverse packing (14), Frenzel (88), and Valsalva (5). Thirty-five (25%) of divers reported equalisation problems during the incident dive. Of all the divers, 104 (74%) reported diaphragmatic contractions during the incident dive. The mental state of divers during the incident dive was categorised as anxious/stressed/uncomfortable in 31 (22%), doubtful/not confident in 24 (17%), neutral in 41 (29%), or positive in 38 (27%). There was a wide variety of theories of why the squeeze incident occurred (Table 1).

The symptoms experienced during squeeze incidents included cardiopulmonary, otolaryngological, and neurologic complaints (Table 2). Only 4 (3%) squeezes were associated with a blackout. Of all the divers who squeezed, 112 (80%) did not receive any treatment while 25 (18%) received oxygen and 3 (2%) received in-water recompression. Of all

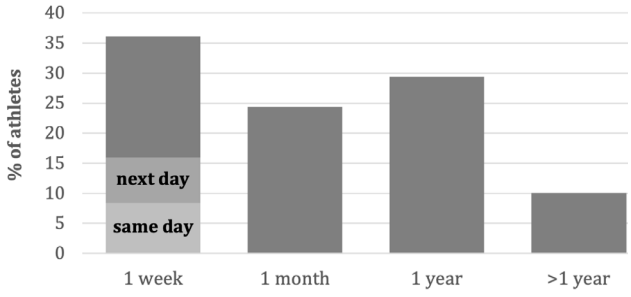
Table 1
Possible contributors to squeeze stratified into three categories

Health and wellness	Preparation and training	Incident during dive
Sleep deprivation (20) Upper respiratory infection (16) Hydration status or hunger (15) Recent squeeze (3) Menstrual cycle (1)	Inadequate warm-up or depth adaptation (29) Diving for too long (16) Pushing personal limit (16) Trouble relaxing (13) Cold water (6) Residual volume dive (1)	Movement at depth (55) Contractions (47) Equalization issue (11) Dive speed (4) Emergency underwater (3) Gear issue (1)

Table 2
Squeeze symptoms stratified within three organ system categories

Cardiopulmonary	Otolaryngological	Neurological
Cough (84) - Hemoptysis (18) Chest tightness (52) Dyspnoea (50) - At rest (29) - With minimal exertion (31) - With heavy exertion (13) Chest pain (6) "*Lung freshness*" (1) "*Felt wet*" (1)	Sputum production (75) - Bloody (57) - Frothy (18) - Thick (5) - Yellow/green (3) - Clear/white (2) Congestion (19) Voice change (11) Throat pain/irritation (7) "*Raspy and gurgly*" (1)	Fatigue (59) Lightheadedness (11) Dizziness (5) Syncope (4) Confusion (3)

Figure 6
Time to return to the same depth after a lung squeeze incident; a third of the athletes returned to their previous depth within a week with a significant number on the same or the following day



the divers who squeezed, 36 (26%) sought further medical attention and 14 (10%) were admitted to hospital. Diagnostic testing included laboratory blood tests (5), radiographs (11), computed tomography (CT) (9), ultrasound (2), magnetic resonance imaging (1), and bronchoscopy (1).

Of the divers who received medical evaluation, four were instructed to get further testing before returning to dive, three were instructed to get repeat X-rays, two were instructed to get repeat CT scans, and one was instructed to get a pulmonary function test. Of the divers who received return-to-dive guidance, 12 were instructed to wait before returning to dive with a range of three days to one year, with a median of one month. The actual time those divers waited ranged from one week to six months, with a median of two months. For all divers regardless of whether they sought medical care, the time until return to the same depth ranged from the same day to four years, with a median of 10 days (Figure 6).

Discussion

This survey captured data from a largely professional group of divers who had experience in freediving instruction and competition for several years. Almost all were certified by one or more freediving organisations, indicating a wide range of freediving training experiences. Almost all respondents had squeezed at least once, indicating a high prevalence of squeeze, even though there were very few divers with underlying medical problems or previous surgeries. We were able to collect data on 140 individual squeeze events over 15 years. It is not surprising that most squeezes occurred during training, as that is a time when divers are pushing their limits or finessing technique within a discipline.

The discipline that required the most movement against resistance (free immersion, which allows use of the arms to pull on the vertical shot line during descent and ascent) resulted in the most squeezes. This is confluent with the respondents' theories that movement at depth was a top

contributor to their squeeze event. These dives may have led to the most squeeze events because the divers were pulling on the rope with more force, thereby straining the thorax more than they would in an arm stroke against the resistance of water.

Most squeeze events occurred on dives shallower than 60 m, with many shallower than the diver's previous personal best depth. This indicates that squeeze can happen even when divers aren't pushing their limits in depth and are likely more affected by other factors during a dive. Many divers cited inadequate warm-up and diaphragmatic contractions from a build-up of carbon dioxide (CO_2) as major contributors to their squeeze event, which agrees with previous reports citing contractions as a major contributor to squeeze.[5] Feeling cold was cited as a rare cause of squeeze, therefore a diver's wetsuit thickness should be appropriate for the water temperature. Carbon dioxide tolerance can be trained over time,[12] but it is unclear how consistent one would need to be with training to build and maintain this adaptation leading up to a dive. It is worth noting that less than a quarter of respondents reported training daily, with a quarter training only seasonally. As this survey did not ask what part of a training season or cycle the squeeze event(s) occurred, it is unclear if these squeezes occurred early in a training season.

In freediving, the standard speed of travel is usually 1 $m \cdot s^{-1}$, though optimal speeds vary between disciplines.[13] For many of the squeeze events reported, the speed of travel was slower than 1 $m \cdot s^{-1}$ which could have resulted in higher oxygen consumption, a more rapid build-up of CO_2, and diaphragm contractions that led to the squeeze. Contractions were cited as the second-highest contributor to squeeze events in this survey. Mental state was cited as a less common cause of squeeze, and > 50% of the squeeze events reported occurred during a positive or neutral mental state, indicating it is a smaller contributor to squeeze. Similarly, 25% of squeeze incidents were attributed to an equalisation issue although most divers utilised mouthfill and Frenzel techniques, indicating equalisation is a lesser contributor to squeeze.

Lastly, it is interesting to note that very few respondents sought medical treatment or evaluation after a squeeze. This follows the current presumption that squeeze events are largely under-reported and under-recognised. Many of the squeeze symptoms could easily be confused for other more common ailments, such as a respiratory infection, or mistaken for other dive injuries, such as decompression illness. That may indicate why a few of the respondents reported receiving in-water recompression as treatment for their squeeze.

A Diver's Alert Network (DAN) workshop on swimming-induced pulmonary oedema and barotrauma of descent in breath-hold diving suggested a general terminology for freedivers who surface with respiratory symptoms: freediving-induced pulmonary syndrome (FIPS).[7] There is still missing information on the exact pathophysiological mechanisms and resolution of the pulmonary pathology that is colloquially called a lung squeeze.

The wide range of diagnostic tests and return-to-diving recommendations speak to the lack of medical guidance on this condition. Most divers who sought medical attention seemed to adhere to a two-month break after a squeeze. However, many more divers who did not seek medical care were able to return to diving at the same depth within a week. The ideal time out of the water after a squeeze remains to be determined.

LIMITATIONS

The authors acknowledge that as with any retrospective survey, there are limitations to consider regarding this data collection. The survey title included the phrase 'lung squeeze', which is colloquially used in the freediving community for barotrauma of descent, it is likely that only freedivers who had experienced a squeeze before took the survey. A prevalence of squeeze injuries in the freediving community can therefore not be established with the existing dataset. Some events that were described occurred months and years before taking the survey; it is common for memories of traumatic events to become slightly modified over time or perceived timelines to be altered.

Conclusions

The findings of this survey suggest that a person who is professionally involved in the sport is very likely to experience a lung squeeze at least once in their career. The severity of lung squeezes varies in respect of signs and symptoms and victims seem to be reluctant to report squeezes or seek medical care after a lung squeeze incident, leaving these events largely under-reported. It is advisable to carefully review the current course content of freediving training agencies and educate freedivers about post-squeeze medical follow-up and return-to-diving recommendations.

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Modelling the risk factors for accidents in recreational divers: results from a cross-sectional evaluation in Belgium

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Keywords
Diving incidents; Diving medicine; Diving research; ENT; Epidemiology; Fitness to dive; Health surveys

Abstract
(Tournoy KG, Vandebotermet M, Neuville P, Germonpré P. Modelling the risk factors for accidents in recreational divers: results from a cross-sectional evaluation in Belgium. Diving and Hyperbaric Medicine. 2024 20 December;54(4):287–295. doi: 10.28920/dhm54.4.287-295. PMID: 39675736.)
Introduction: Characterisation of the recreational diving community could help to identify scuba divers at risk for accidents.
Methods: We performed a cross-sectional evaluation in a federation for recreational scuba divers in Belgium (Duiken. Vlaanderen). Using binary logistic regression, factors predictive for accidents leading to hospitalisation were identified.
Results: Of the 710 members, 210 (29.6%) participated in the survey, representing 140,133 dives. Age was > 50 years in 55% and the median (interquartile range [IQR]) number of dives was 380 (IQR 140–935). Cardiac (9.5%), orthopaedic (11.0%), ear-nose-throat (ENT, 10.5%) and allergic diseases (30.5%) were the top four morbidities. Twenty percent reported taking cardiovascular medication. Decompression accidents, barotrauma of the ear and musculoskeletal injuries were reported in 11.0, 11.9 and 11.0%. Fifty-five divers (26.2%) reported incidents not necessitating a medical intervention. For 36 divers (17.1%), medical interventions were necessary. Among these, 13 divers (6.2%) were hospitalised at least once and 12 (5.7%) of these needed hyperbaric oxygen therapy (HBOT). The absolute risk for hospitalisation or HBOT was 0.01% per dive. Age, advanced diving qualification, more dives annually, cardiac or ENT pathology and cardiac medication were significantly associated with an increased likelihood of hospitalisation resulting from diving accidents. In a multivariate risk model, ENT comorbidity (odds ratio [OR] 9.3; $P = 0.006$) and cardiac medication (OR 5.6; $P = 0.05$) predicted hospitalisation due to a diving accident.
Conclusions: One in six recreational scuba divers required a medical intervention at least once during their career, while 6.2% were hospitalised or received HBOT. Ear nose and throat comorbidity and cardiac medication were strong predictors for accidents. These should be given sufficient weight in dive medical examination.

Introduction

Balancing with its appeal to explore underwater environments, scuba diving poses inherent risks, ranging from minor discomforts to life-threatening accidents.[1,2] Minor diving incidents to major accidents can result from various factors, including equipment failure and diver-related issues such as health status and experience level. In addition, environmental conditions such as colder waters, strong currents or poor visibility impose additional challenges to the diver.[3] Few studies exist that document diving-related injuries and individual risk factors within particular divers populations.[4–8] Understanding the characteristics of the diving community and the factors contributing to these risks is crucial for enhancing diver safety and guiding medical practitioners in their assessments and interventions.

Data on the risks of the recreational diving community in Belgium which is exposed to a specific blend of dive types, is lacking. Dives in the tidal North Sea and the Eastern Scheldt Estuary complement popular (fresh water) quarry explorations. Additionally, many engage in more classical 'holiday dives', often in tropical waters abroad. The literature is devoid of multivariate risk models weighing the relative importance of risk features in divers with or without a history of accidents. Fitness-to-dive assessments in Belgium are done primarily by general physicians, who most often lack formal diving medicine qualifications. Although guidelines and questionnaires to assess the fitness-to-dive do exist there is no information available on their adherence.[9] In addition, these guidelines do not necessarily account for particular risk factors that may be important for certain diver populations.

The aim of the current study was to identify predictors of serious diving-related outcomes, such as hospitalisation and the need for hyperbaric oxygen treatment (HBOT), by assessing divers' profiles, comorbidities, and accident reports in Flanders, Belgium. We wanted to measure the past and current adherence to questionnaire-based medical examinations when fitness to dive is evaluated. By aligning medical examination protocols with newly identified risks, we hope to support both divers and healthcare providers in promoting a safer diving environment.

Methods

An online questionnaire (Forms, Office 365 – Microsoft Corporation, Redmond, WA, USA) was presented to all members of the scuba dive clubs affiliated with 'Duiken. Vlaanderen', one of the diving federations in Belgium. Members had to provide informed consent before access was be given to the separate and anonymised questionnaire. The study was supervised by the independent juridical and ethical committee of the federation 'Duiken.Vlaanderen'. The invitation was sent three times to the divers between January and March 2024.

The questionnaire was developed by the medical committee of the federation to meet the objectives of this research project. The questionnaire comprised 86 questions. There were four parts to be completed: (i) general biometrics and diving history, (ii) general medical information with inquiries for comorbidities and medication, (iii) diving incidents and accidents and (iv) data on the fitness-to-dive medical examination. Participants were instructed to document their situation from the start of their diving career until the cut-off date of 1 January 2024. A 'diving incident' was defined as an unexpected or unusual event that occurs during a dive resulting in harm or injury to the diver, but without the necessity of a formal medical intervention and solved by the diver or his buddy. A 'diving accident' specifically refers to a situation resulting in harm ranging in severity from minor injuries to more serious situations such as decompression sickness or barotrauma, but always requiring professional medical intervention. The full questionnaire (available in Dutch due to the specific diving population) is available upon simple request to the corresponding author.

The anonymised data matrix was transferred as an Excel file to SPSS 28.0 (IBM Corp. Released 2021. IBM SPSS Statistics for Windows, Version 28.0. Armonk, NY: IBM Corp) for statistical analysis. Data are presented as medians (range and interquartile range [IQR]) for continuous variables and as numbers (frequencies) for categorical variables. To compare the frequencies and medians of selected variables in subgroups, Fisher's exact and Mann-Whitney U tests were used. To identify risk factors for diving accidents and hospitalisations due to diving accidents, a binary logistic regression model was constructed using those variables identified as significant in the univariate analysis. The model included a constant value, and the variables were included using the enter method. The Nagelkerke R square values were calculated to adjust the Cox and Snell measures providing a more interpretable metric of model fit (data not shown). Odds ratios (OR) with 95% confidence intervals (95% CI) were calculated. A significance level of $P < 0.05$ was adopted for all analyses.

Results

POPULATION CHARACTERISTICS

The recreational scuba federation 'Duiken.Vlaanderen' member count was 710 in January 2024 – date of the survey. Two-hundred fourteen members gave consent to participate to the survey (30.1%), but four (1.9%) had no diving experience other than in a pool. The clinical characteristics of the 210 divers are shown in Table 1. Age was over 50 in 55.2%, and 83.8% were males. A history of tobacco smoke exposure (either active, passive or former smoker, with the latter defined as having quit at least one year ago) was present in 41.9%. Among the respondents, 35.7% were diving instructors with a significant proportion engaging in more technical profiles: 53.8% were qualified for decompression or deep dives and 76.7% were trained for Nitrox diving. The median years of diving experience was 18 (IQR 8–28), the number of dives was 380 (IQR 140–935), with 23 (IQR 15–44) dives annually. A total of 140,133 logged scuba dives were reported in this survey.

COMORBIDITY AND MEDICATION USE

There were 80 divers (38.1%) reporting no morbidities. In Table 2, the self-reported morbidities of the other divers are listed, many having multiple comorbidities. Nine and a half percent of the respondents indicated they had been hospitalised or that they are receiving medical care or follow-up for cardiac diseases (mainly hypertension and cardiac arrhythmias). This was 10.5% for ear-nose-throat (ENT) problems and 30.5% for allergies. Musculoskeletal and rheumatic m respectively. Pulmonary diseases were reported in 3.3% (mainly asthma). There were no divers with a history of primary pneumothorax, but two reported a secondary pneumothorax. Of note, 98.1% of divers reported to have been vaccinated at least twice for COVID-19. Table 3 summarises the active medication profiles. The most frequent medications taken were cardiovascular in 20.0%, anti-allergic in 15.2% or pulmonary in 4.8%. Four divers (1.9%) were on anticancer drugs.

DIVING INCIDENTS AND ACCIDENTS

Fifty-five divers (26.2%) reported a total of 116 incidents related to scuba diving, not necessitating the intervention of a medical doctor. This translates into an absolute risk of 0.08% per dive (Table 4). One or more diving accidents necessitating medical interventions were reported by 36 (17.1%). There were 13 divers (6.2%) who reported at least one hospital admission because of a diving accident.

Table 1
Population characteristics of the study cohort; AOW – advanced open water; IQR – interquartile range; * vocational or higher education

Characteristic	*n* = 210
Age category, *n* (%)	
< 20 yr	5 (2.3)
21–30 yr	13 (6.2)
31–40 yr	25 (11.9)
41–50 yr	51 (24.3)
51–60 yr	72 (34.3)
61–70 yr	38 (18.1)
71–80 yr	6 (2.9)
Male, *n* (%)	176 (83.8)
Body mass index, kg·m^{-2} median (range; IQR)	27 (15–42; 24–30)
Education, *n* (%)	
Primary	2 (1.0)
Secondary	67 (31.9)
Postsecondary*	141 (67.1)
Smoking, *n* (%)	
Never	122 (58.1)
Passive	11 (5.2)
Active	17 (8.1)
Ex-smoker	60 (28.6)
Diver qualification, *n* (%)	
Candidate	4 (1.9)
D1 (Open water)	14 (6.7)
D2 (AOW)	41 (19.5)
D3 (Master diver)	50 (23.8)
D4 (Rescue diver)	26 (12.4)
Instructor	75 (35.7)
Years of diving median (range; IQR)	18 (1–54; 8–28)
Outdoor dives median (range; IQR)	380 (1–4,386; 140–935)
Dives per year median (range; IQR)	23 (1–186; 15–44)
Specialties, *n* (%)	
Dry suit	108 (54.4)
Deep / decompression	113 (53.8)
Nitrox diving	161 (76.7)

Table 3
Medications used by subjects in the study cohort; *includes blood pressure medication, anticoagulants and antiplatelet agents including low dose aspirin, lipid lowering drugs, anti-arrythmia medication; **inhalers

Medications in 210 subjects	*n* (%)
Cardiac medication*	42 (20.0)
Anti-allergy medication	32 (15.2)
Pulmonary medication**	10 (4.8)
Diabetes medication	6 (2.9)
Neuropsychiatric medication	5 (2.4)
Cancer therapy	4 (1.9)

Table 2
Comorbidity burden among subjects in the study cohort; COPD – chronic obstructive pulmonary disease; * with or without stent or prior coronary bypass grafting

Comorbidities in 210 subjects	*n* (%)
Cardiac diseases	20 (9.5)
High blood pressure	10 (4.8)
Cardiac arrythmia	8 (3.8)
Pacemaker or defibrillator	0 (0)
Myocardial infarction*	0 (0)
Valvular disease	2 (1.0)
Other cardiac diseases	4 (1.9)
Pulmonary diseases	7 (3.3)
Asthma	5 (2.4)
COPD	0 (0)
Thoracic surgery	1 (0.5)
Other pulmonary diseases	1 (0.5)
ENT diseases	22 (10.5)
Kidney diseases	2 (1.0)
Gastro-intestinal disease	6 (2.9)
Diabetes	6 (2.9)
Rheumatic diseases	17 (8.1)
Neurologic diseases	4 (1.9)
Psychiatric diseases	4 (1.9)
Oncologic diseases	10 (4.8)
Orthopaedic diseases	23 (11.0)
Lower back problems	9 (4.3)
Back surgery	8 (3.8)
Arthrosis	9 (4.3)
Prosthesis	7 (3.3)
Allergies	64 (30.5)
Claustrophobia	5 (2.4)
COVID	
Vaccinated (at least 2 vaccines)	206 (98.1)
At least one positive COVID test	140 (66.7)
Hospitalised due to COVID	1 (0.5)
Other pathologies	31 (14.8)

Twelve of these were treated with HBOT. There were 24 divers (11.4%) who sought emergency medical help, but without subsequent HBOT or hospitalisation (one of these divers also reported hospitalisation on another occasion). Decompression sickness, middle ear barotrauma and orthopaedic events were reported in 11.0, 11.9 and 11.0% respectively. The absolute risk for hospitalisation due to diving accidents in this cohort was 0.01% per dive. Two divers suffered from severe permanent health issues resulting from their diving accident (1.0% of the divers' population).

RISK ASSESSMENT FOR DIVING ACCIDENTS WITH HOSPITALISATION

We conducted a risk assessment for the divers reporting to have been hospitalised due to a diving accident, regardless of the need for HBOT. There were 13 divers reporting a total of 18 hospitalisations (Table 4).

Table 4

Diving incidents and accidents among subjects in the study cohort; MD – medical doctor; * twelve divers had accidents necessitating hyperbaric oxygen treatment (HBOT). A total of 15 accidents necessitating HBOT were recorded (some divers had more than one accident); ** there were 24 divers seeking medical help without hospitalisation, three of them had two medical contacts, and one of these divers also reported being hospitalised because of a diving accident on separate occasions explaining why the total number of divers with at least one accident was 36; # 15/140,133 equals an absolute risk of 10.7/100,000 dives for requiring HBOT (see discussion)

Scuba diving events	210 divers *n* (%)	140,133 dives *n* (%)
Diving incidents (without MD consult)	55 (26.2)	116 (0.08)
Diving accidents (with MD consult)		
Hospitalisation (with or without HBOT)*	13 (6.2)	18 (0.01)#
No hospitalisation**	24 (11.4)	27 (0.02)
Diving incidents and accidents		
Decompression event	23 (11.0)	–
Barotrauma ear	25 (11.9)	–
Orthopaedic events	23 (11.0)	–
Recovery characteristics		
Major health issues remaining	2 (1.0)	–
Minor health issues remaining	5 (2.4)	–

Table 5

Characteristics of the study cohort stratified by those hospitalised and not hospitalised; IQR – interquartile range; * vocational or higher education; ** see Table 1 for diver qualification classifications

Characteristic	Not hospitalised *n* = 197	Hospitalised *n* = 13	*P*-value
Age category, *n* (%)			
< 50 years	92 (46.7)	2 (15.4)	0.04
≥ 50 years	105 (53.3)	11 (84.6)	
Sex, *n* (%)			
Male	165 (83.8)	11 (84.6)	ns
Female	32 (16.2)	2 (15.4)	
Body mass index, $kg \cdot m^{-2}$	27	28	ns
median (range; IQR)	(15–42; 24–29)	(19–35; 25–32)	
Education, *n* (%)			
Primary or secondary	63 (32.0)	6 (46.2)	ns
Postsecondary*	134 (68.0)	7 (53.8)	
Smoking, *n* (%)			
Never	117 (59.4)	5 (38.5)	ns
Active, passive or ex	80 (40.6)	8 (61.5)	
Diver qualification, *n* (%)			
Candidate D3**	108 (54.8)	1 (7.7)	< 0.001
D4 → Instructor	89 (45.2)	12 (92.3)	
Dives per year,	21	48	< 0.001
median (range; IQR)	(1–122; 14–42)	(41–186; 43–77)	
Specialties, *n* (%)			
Dry suit	97 (49.2)	11 (84.6)	0.02
Deep / decompression	101 (51.3)	12 (92.3)	0.004
Nitrox diving	148 (75.1)	13 (100)	0.04

Hospital admissions were predominantly for HBOT. In Tables 5–7, the characteristics of these divers are compared to those divers that were never hospitalised due to diving accidents. As shown in Table 5, divers with accidents necessitating hospitalisation were older (> 50 years, $P = 0.04$), had a higher-level diver qualification ($P < 0.001$) and in addition did more dives per year (48 versus 21, $P < 0.001$). In Table 6, the burden of comorbidities is compared. Divers with accidents necessitating hospitalisation had significantly more cardiac diseases (30.8% vs 8.1%, $P = 0.03$) or ENT pathology (38.5% vs 8.6%, $P = 0.006$). The medication use of the divers with

Table 6
Comorbidities among the study cohort stratified by those hospitalised and not hospitalised; ENT – ear nose and throat conditions; * if total count of patients with a comorbidity was ≤ five, the factor was not tested

Comorbidity	Not hospitalised, *n* = 197 *n* (%)	Hospitalised, *n* = 13 *n* (%)	*P*-value*
Cardiac diseases, High blood pressure	16 (8.1) 6 (3.0)	4 (30.8) 4 (30.8)	0.03 0.002
Pulmonary diseases Asthma	6 (3.0) 4 (2.0)	1 (7.7) 1 (7.7)	ns ns
ENT diseases	17 (8.6)	5 (38.5)	0.006
Gastro-intestinal diseases	6 (3.0)	0 (0)	ns
Diabetes	6 (3.0)	0 (0)	ns
Rheumatic diseases	14 (7.1)	3 (23.1)	ns
Cancer	9 (4.6)	1 (7.7)	ns
Orthopedic diseases Lower back problems Back surgery Arthrosis Prosthesis	22 (11.2) 8 (4.1) 7 (3.6) 8 (4.1) 6 (3.0)	1 (7.7) 1 (7.7) 1 (7.7) 1 (7.7) 1 (7.7)	ns ns ns ns ns
Allergies	60 (30.5)	4 (30.8)	ns
Claustrophobia	5 (2.5)	0 (0)	ns

Table 7
Use of medications among the study cohort stratified by those hospitalised and not hospitalised; * if total count of patients with a comorbidity was ≤ five, the factor was not tested; ** includes blood pressure medication, anticoagulants and antiplatelet agents including low dose aspirin, lipid lowering drugs, anti-arrythmia medication; *** inhalers

Medications	Not hospitalised, *n* = 197 *n* (%)	Hospitalised, *n* = 13 *n* (%)	*P*-value*
Cardiac medication**	35 (17.8)	7 (53.8)	0.005
Pulmonary medication***	9 (4.6)	1 (7.7)	ns
Diabetes medication	6 (3.0)	0 (0)	ns
Anti-allergy medication	30 (15.2)	2 (15.4)	ns
Neuropsychiatric medication	5 (2.5)	0 (0)	ns

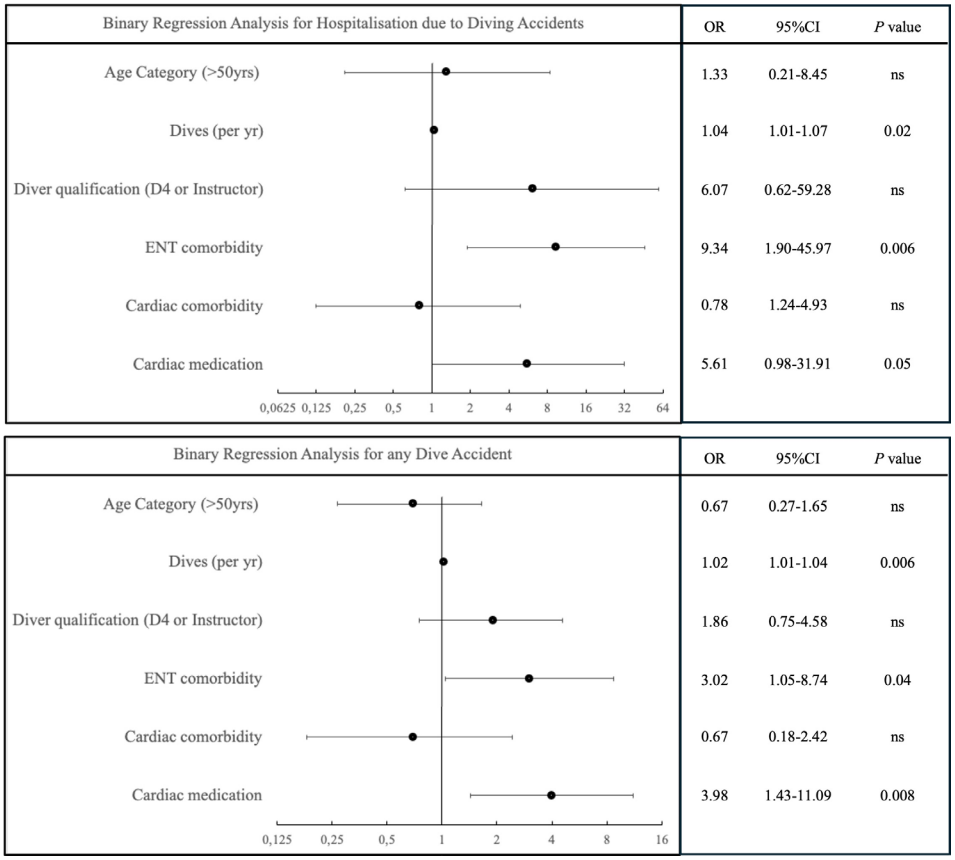
accidents necessitating hospitalisation is shown in Table 7. Those hospitalised for an accident were more likely to take cardiac medication (53.8% versus 17.8%, $P = 0.005$).

Based on the factors identified above, a binary multivariate logistic regression model was constructed to model the risk for a severe diving accident in the studied cohort. We selected age category, diving qualification (D4 or instructor vs all lower qualifications), number of dives per year, cardiac or ENT comorbidities and the regular use of cardiac medication as risk factors of interest. A forest plot is shown modelling the risk for 'any diving accident' (Figure 1 – lower panel) or a 'diving accident necessitating hospitalisation' (Figure 1 – upper panel). The OR for an accident leading to hospitalisation of the diver (with or without HBOT) was 9.34 (95% CI 1.90–45.97, $P = 0.006$) and 5.61 (95% CI 0.98–31.91, $P = 0.05$) if there was ENT comorbidity and if any cardiac medication was taken respectively. The OR for any dive accident leading to an urgent medical intervention was 3.02 (95% CI 1.05–8.74, $P = 0.04$) and 3.98 (1.43–11.09, $P = 0.008$) in case of ENT comorbidity or if cardiac medication was taken respectively. The number of dives per year is also significantly correlated (although the OR is close to 1) while age and diver qualifications are not.

DIVING MEDICAL EXAMINATIONS: EXPERIENCES AND PREFERENCES

Table 8 shows the medical examinations the scuba divers underwent. During the initial diving medical examination, targeted questionnaires were frequently used (58.1%), along with ECG (53.8%), ergometry (29.0%), thoracic imaging (9.0%), and spirometry (52.4%). In subsequent consultations, there is a trend towards less use of questionnaires and technical investigations. In 52.6% of those older than 50 years, an ergometry was performed in the last three years. In at least 76.7%, the diver estimated the knowledge of the medical doctor as appropriate; but at least 12.9% did not think their medical examiner was competent for diving medicine. Gauging the preference of divers about the way

Figure 1
Risk model for dive accidents; the lower panel pertains to 'any diving accident' and the upper panel pertains to a 'diving accident necessitating hospitalisation'



a medical examination should be performed, a minority (5.7%) indicated that systematic medical screening is not useful. The majority (61.4%) believed the combination of a questionnaire plus a doctors' visit, and ergometry in those aged at least 45 years, is appropriate. Two thirds prefered to have a yearly medical examination, while one in four indicated a two-yearly medical examination would be sufficient.

Discussion

The most important finding of our study is that one in six divers of the evaluated group reports to have needed professional medical help because of a diving related injury during their diving career. The presence of ENT comorbidities and the regular intake of cardiovascular medications were strongly associated with an increased risk of hospitalisation among Belgian divers.

Analyses of several recreational diving communities and their diving-related injuries is of utmost relevance to understanding and mitigating the risk factors. Not surprisingly, the identification of risk factors differs depending on the characteristics of the population, but also on the types of diving performed. These probably explain why risk factors for diving related injuries appear inconsistent across studies.[5,7,10,11] For example, age appears to be a risk factor for diving injuries in some studies[10] while it is a protective factor in others.[5–7]

Our study population can be considered 'old' (55% were aged 50 or older), 'male' (84%) with a considerable fraction (> 35%) of active smokers or former tobacco smoke exposure. They are apparently experienced divers (median 380 dives, 18 years of diving, and 36% were dive instructors). Only thirty-eight percent of respondents reported no comorbidities necessitating regular medical follow-up, which indicates comorbidities are prevalent in the studied population. We found that orthopaedic problems, cardiac diseases (mainly hypertension), ENT problems and allergies were the top four self-reported health issues for which the divers were followed-up regularly by a medical doctor. Lower back pain seems prevalent in divers and was related to higher weight belt loading.[12] In a recent review by Westerweel et al. it was reported that depending on the series, 12–33% of divers are reported to be hypertensive.[13] In our survey, we found that only 5% reported being followed-up by a medical specialist because of hypertension, however 20% did take medication related to the cardiovascular system. This was more than the 10% reported in a United Kingdom study[14] but lower than the 28% found in a Dutch study.[4] It has been suggested that the use of cardiac related medication in divers could be an

Table 8
Medical examinations undergone by the study cohort and related opinions and preferences; CT – computed tomography; ergo – ergometry; MD – medical doctor

Parameter	Initial examination *n* (%)	Last examination *n* (%)
Diving medical examination		
Consult with questionnaire	122 (58.1)	87 (41.4)
Consult without questionnaire	79 (37.6)	106 (50.5)
No consult	9 (4.3)	17 (8.1)
Tests administered during consult		
Clinical exam	192 (91.4)	177 (84.3)
Electrocardiogram	113 (53.8)	64 (30.5)
Ergometry	61 (29.0)	43 (20.5)
Spirometry	110 (52.4)	88 (41.9)
X-ray or CT scan of the chest	19 (9.0)	11 (5.2)
Ergometry in divers over 50 years	–	61/116 (52.6)
Appreciation of MD knowledge		
Good	79 (37.6)	–
Basic	82 (39.0)	–
Insufficient	27 (12.9)	–
Don't know / prefers not to tell	22 (10.5)	–
Divers' preferences initial medical exam		
Not necessary	1 (0.5)	–
Questionnaire + MD if abnormal	11 (5.2)	–
Questionnaire + MD always	59 (28.1)	–
Questionnaire + MD + ergo if ≥ 45 yr	129 (61.4)	–
None of the above	10 (4.8)	–
Divers' preferences follow-up exam		
Not necessary	–	5 (2.4)
Yearly	–	140 (66.7)
Two-yearly	–	52 (24.8)
Three-yearly	–	11 (5.2)
None of the above	–	2 (1.0)

argument for a more rigorous medical screening.[15] Problems with the upper airways and allergies necessitating regular medical contacts were quite prevalent in our population, a finding that was consistent with another report.[4]

The current survey evaluated self-reported diving-related incidents and accidents. Not surprisingly, the number of incidents largely exceeded that of accidents, the latter necessitating professional medical intervention. At the individual level, we found that one in six had needed at least one episode of professional medical help because of an acute diving-related injury. Although this seemed a very high number, it needs to be interpreted in the context of the high number of diving years (almost 4,000) or absolute number of dives (over 140,000) in our study population. In a Divers Alert Network (DAN) study there were 5.7 decompression accidents requiring HBOT per 100,000 dives.[5] In the current study, we found a higher figure: 10.7 per 100,000 dives. The absolute risk for permanent severe physical harm after a diving accident was 1% – a figure that compares to earlier reports describing severe residual symptoms in eight out of 799 divers.[7]

By identifying specific health conditions and behaviours that increase the risk of diving accidents, we can offer better recommendations for individual medical evaluations and interventions.[6] We presumed that the most accurate recordings would be those that caused hospitalisation (with or without HBOT). We therefore focused on those divers to construct a risk assessment model. In univariate analysis, we found that a higher age, a higher diver qualification and higher dive frequency as well as the presence of cardiac and ENT comorbidities and the regular use of cardiac medication all correlated with the risk for a diving accident with hospitalisation. Body mass index (BMI), sex or smoking behaviour did not. These findings differ with those of Ranapurwala et al. who found that greater age, more annual dives and higher certification levels were associated with less self-reported decompression symptoms.[5] Notably, their survey did not assess the risk of hospitalisation.

Multivariate risk models are however more relevant for estimating the risk for a diving accident. They account for the complexity and interplay of multiple factors simultaneously and provide a nuanced risk estimation. This leads to more

reliable and valid risk predictions, which are essential for making informed decisions and a more personalised risk management. Our binary logistic multivariate analysis reveals that the presence of ENT pathology and the use of cardiovascular medication indicate an increased risk for more severe diving accidents resulting in hospitalisation, while age or diver qualification no longer appear to be significant factors. Here, the question arises as to whether the influence of cardiovascular medication is explained by the role of beta-blockers and diuretics (commonly used antihypertensives) in the development of diving accidents such as decompression sickness and immersion pulmonary oedema.[13] A higher number of dives per year is also a significant factor, however, with a hazard ratio of 1.02 and 1.04 for any accident and for hospitalisation respectively, it cannot be seen as clinically relevant. To the best of our knowledge, this is the first integrated analysis of combined risk factors in a particular diver population and provides useful information to guide risk assessments and fitness of dive examinations.

We assessed how the current study population was evaluated for their first and last medical examinations. One in eight divers expressed concerns regarding the medical examiners' expertise during the fitness-to-dive evaluation, a concerning statistic. Standardised questionnaires as recommended by the Undersea and Hyperbaric Medical Society (UHMS) were used in less than 60%. This indicates there is room for teaching and standardisation.[9] It could be of help to indicate the websites where these questionnaires can be found on the medical cards issued by the dive federations. A wide range of medical tests was used with the majority of those done during the first-time medical exams. It is clear from the data that in daily practice, basic technical investigations such as electrocardiography or spirometry are not rigorously implemented.[16,17] The majority of participants endorsed the usefulness of a yearly medical investigation, preferably based on the use of a questionnaire, a clinical exam and an ergometry for those aged over 45 years. It has indeed been shown that the addition of a medical investigation on top of a questionnaire is more sensitive to detect those at risk for diving accidents.[18] A patient-centred approach, taking into account the risk-factors identified for the diving population the diver belongs to, and facilitating shared decision-making between divers and practitioners is always recommended to ensure an optimal assessment.[16]

The current analysis benefits from an adequate response rate to an online survey (30%) and the comprehensive evaluation of the participants' medical status, diving experience and accidents, enabling the development of a robust risk model. The fact that the analysis was done in one of the smaller diving federations in Belgium is subordinate. We argue the data are most probably of relevance for all recreational divers exposed to the blend of dives outlined in the methods. However, it is essential to emphasise certain issues that warrant cautious interpretation of the data. The diver sample exhibited selection bias, evidenced not only by a different distribution of dive qualifications compared to those who did not participate in the survey ($P < 0.001$, data not shown), but also by the fact that persons who terminated their membership after an accident were not considered. The impact of this bias on our conclusions is hard to estimate. The cross-sectional study design is subject to inherent limitations, including a notable risk of underreporting. The implication would be that the figures we report are even an underestimation of the reality. Comorbidities were defined as a medical problem that either led to hospitalisation or that still requires a regular medical follow-up. As a result, individual divers may incorrectly perceive some medical issues as not relevant. This may explain lower than expected values for cardiac comorbidities in comparison to the prevalence of cardiovascular medication use. An additional risk for underreporting, particularly for minor events, arises from considering a complete diving career spanning up to 54 years. Therefore, we focussed on severe accidents resulting in hospitalisation or HBOT, presuming that the likelihood of recall failure and underreporting would be reduced. However, this approach has the limitation of reducing the number of events available for risk factor analysis. Prospective data collection would effectively address these issues properly and appears to be a feasible approach.[11] Additionally, it could enable the evaluation of other potentially relevant variables, such as physical fitness, detailed diving profiles, geographical diving locations, and technical specifications of the diving equipment, which we didn't consider in this study.

Conclusions

By surveying a broad and diverse cohort of recreational divers in Belgium, we acquired valuable insights into common health issues and the frequency and characteristics of diving accidents. Our multivariate analysis identified cardiac medication and ENT disease as risk factors associated with dive accidents. These should require specific attention from healthcare professionals during medical evaluations. We suggest that similar studies be carried out in different settings as the identification of risk factors for dive accidents could improve fitness-to-dive assessments and contribute to overall dive safety.

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Meclizine seasickness medication and its effect on central nervous system oxygen toxicity in a murine model

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Cholinergic antagonists; Closed circuit rebreathers; Diving; Histamine antagonists; Seizures

Abstract
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Introduction: Diving utilising closed circuit pure oxygen rebreather systems has become popular in professional settings. One of the hazards the oxygen diver faces is central nervous system oxygen toxicity (CNS-OT), causing potentially fatal convulsions. At the same time, divers frequently travel by boat, often suffering seasickness. The over-the-counter medication meclizine is an anticholinergic and antihistaminergic agent that has gained popularity in the treatment of seasickness. Reports have shown the inhibitory effect that acetylcholine has on glutamate, a main component in the mechanism leading to CNS-OT seizure. The goal of the present study was to test the effect of meclizine on the latency to CNS-OT seizures under hyperbaric oxygen conditions.
Methods: Twenty male mice were exposed twice to 608 kPa (6 atmospheres) absolute pressure while breathing oxygen after administration of control solution (carboxymethyl cellulose solvent) or drug solution (meclizine) in a randomised crossover design. Latency to tonic-clonic seizures was visually measured.
Results: Mean latency to seizure did not significantly differ between the control group (414 s, standard deviation [SD] 113 s) and meclizine group (434 s, SD 174 s).
Conclusions: Based on results from this animal model, meclizine may be an appropriate option for divers suffering from seasickness, who plan on diving using pure oxygen rebreather systems.

Introduction

Military diving operations are becoming increasingly more complex and demanding. They often require long durations and compact gear, particularly in clandestine missions. Breathing systems that fulfil these requirements usually deliver oxygen (O_2) rich gas in a closed-circuit design. In addition, many divers suffer from seasickness while travelling to the dive site, and even during the dive. Thus, seasickness medications are in demand amongst divers.

Divers breathing pure O_2 are exposed to significantly higher partial pressures of oxygen (pO_2) than at sea level. Exposure to elevated PO_2 may impose toxic effects on the central nervous system (CNS) and lungs, which may be life-threatening. Thus, the likelihood for CNS O_2 toxicity (CNS-OT) must be considered during dive planning.[1,2]

The hallmark of CNS-OT is a generalised tonic-clonic seizure. There may be prodromal symptoms such as sensory anomalies (aura, blurred vision, tinnitus, tingling of extremities, dizziness) and changes in mood and mental status.[3,4] The seizures manifest with a sudden loss of consciousness and stiffening of the body (tonic phase) followed by twitching and jerking of the face, arms, and legs (clonic phase). These events will ultimately prove fatal if pO_2 is not reduced.[3] If such seizures should occur during a dive, death by drowning may occur. As the risk for CNS-OT is a function of exposure time and PO_2, efforts have been made over the decades to create predictive risk analysis tools.[1,5]

Divers must also cope with seasickness, affecting 25–75% of sea vessel passengers.[6] Meclizine, an anti-motion sickness medication, operates in the CNS as both a histaminergic receptor antagonist and a nonspecific muscarinic receptor antagonist.[7] Thus, it also blocks CNS acetylcholine receptors.[8,9] The neurotransmitter basis for oxygen toxicity seizures involves elevated levels of acetylcholine and glutamate in the CNS. Studies show that the first tonic phase is induced by over-stimulation of muscarinic acetylcholine receptors (mAChRs), while the second clonic phase is a result of high levels of glutamate.[10–12] The mAChR receptors have been found to downregulate glutamate activity,[13] while antagonists of mAChR prevent the tonic

phase seizures.[10] The inhibitory effect of acetylcholine on glutamate is probably mediated by GABAergic neurons.[14,15] This GABAergic population of cells appears to produce inhibitory control of glutamate's excitatory activity. Under these conditions, susceptibility to CNS-OT could be curbed. Compounds having antimuscarinic activity, such as scopolamine and meclizine, block the excitatory effect of acetylcholine receptors. In this manner, the GABA downregulatory effect on glutamate would be reduced. This cascade has the potential to hasten the onset of full tonic-clonic seizures. Nevertheless, uncertainty remains regarding how different acetylcholine receptor antagonists affect the risk of CNS-OT.[3]

Previous studies have tested the interaction of high PO_2 with other anti-motion sickness medications,[16,17] yet meclizine remains untested. Meclizine is becoming increasingly popular thanks to its global availability as an over-the-counter drug and minimal side effects. The present study aimed to evaluate the effect of meclizine on the latency to CNS-OT under hyperbaric conditions in a murine model.

Methods

ETHICAL APPROVAL

The Animal Research Committee of the Israel Ministry of Defence approved the experimental procedures, and husbandry and handling were in accordance with internationally accepted humane standards.

STUDY POPULATION

Twenty male C57BL/6 mice (Envigo RMS, Jerusalem, Israel) aged eight weeks and weighing between 16 and 20 grams were included in the study.

HUSBANDRY

Mice were housed in wire frame cages under standard conditions, with free access to drinking water, cardboard homes, and standard feed. They were kept in a 12-hour (h) light / 12 h dark cycle, and the ambient temperature was maintained at 24°C.

MECLIZINE PREPARATION

Carboxymethyl cellulose (CMC) solution 0.25% w/v, prepared by mixing CMC powder (MO512-100G, 4000 cP, Sigma) in single distilled water, served as the solvent and control solution. Meclizine solution was prepared with a single Bonine® tablet (25 mg meclizine hydrochloride, WellSpring Pharmaceutical Corporation) triturated by mortar and pestle and suspended in CMC solution via sonication. The final animal equivalent dose calculated and used was 5.2 $mg \cdot kg^{-1}$ per mouse, according to well-established methods.[18–20]

EXPERIMENTAL PROCEDURE

Each mouse was exposed twice to hyperbaric O_2 (HBO) in a randomised-crossover design. The exposure procedure was performed on each mouse individually. Since sensitisation to oxygen toxicity has been observed in small rodents after repeat exposures to HBO, the two sessions were performed a week apart to allow for any residual effects to dissipate.[21,22] Mice were denied food 12 h preceding HBO exposures, assuring more uniform drug pharmacokinetics.[23] Free access to fresh water was maintained at all times. Since the effects of drugs tend to be influenced by body mass, the body mass of the study groups was recorded and compared.

Mice were administered 0.2 mL of either control solution (CMC) or meclizine solution using a 20-gauge oral gavage needle, followed by placement in the test exposure box at standard ambient atmospheric conditions (101.3 kPa absolute pressure, 21% O_2). Mice were given 10 minutes to acclimatise in the exposure box, which is the time the drug was calculated to reach maximal blood concentration according to mouse equivalent pharmacokinetics.[24] The exposure box was placed in a hyperbaric chamber (Roberto Galeazzi, La Spezia, Italy) and pressure was increased at a rate of 101.3 $kPa \cdot min^{-1}$ (one atmosphere per minute) up to 608 kPa absolute pressure (six atmospheres absolute) with 100% O_2. While fully pressurised and breathing pure O_2, the mice were observed for clear signs of tonic-clonic seizures. Once seizures were evident and the time of exposure documented, the gas in the exposure box was replaced with air and the hyperbaric chamber was depressurised at a rate of 101.3 $kPa \cdot min^{-1}$ to avoid decompression illness. Mice were retested after seven days for the other treatment (control or meclizine). Every exposure session was recorded by video and subsequently further analysed to ensure the exact time of seizure onset was captured. A visual outline of the hyperbaric oxygen exposure procedure can be seen in Figure 1.

After completion of both sessions, mice were sacrificed by sedation with isoflurane vapour, followed by pentobarbital sodium overdose (200 $mg \cdot ml^{-1}$, CTS, Israel) and manual neck dislocation.

Data analysis was performed using GraphPad InStat 3.1 (GraphPad Software, San Diego, CA, USA) and KaleidaGraph 5.02 (Synergy Software, Reading, PA, USA).

Results

The mice exhibited the highly reproducible tonic-clonic seizures expected of CNS-OT, with an average (standard deviation [SD]) latency of 424 (SD 146) s. Comparing the latency to toxicity in control (414 [SD 113] s) versus meclizine-treated mice (434 [SD 174] s), no statistically significant difference was observed (paired Student *t*-test, $P = 0.37$). Additionally, plotting for the change in latency

Figure 1
Exposure profile of mice to hyperbaric oxygen conditions, and measurement of latency to onset of tonic-clonic seizures; during pressurisation, air is gradually switched to oxygen, and during decompression the oxygen is switched back to air. kPa – kilopascals; min – minutes; O_2 – oxygen

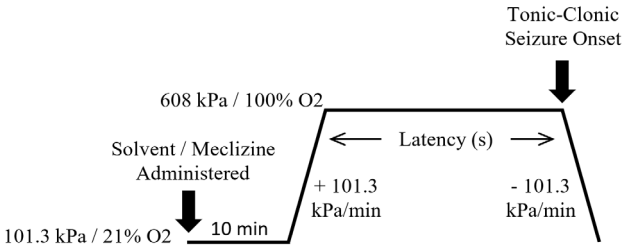

Figure 2
Latency for individual subjects for control and meclizine-treatment conditions. s – seconds

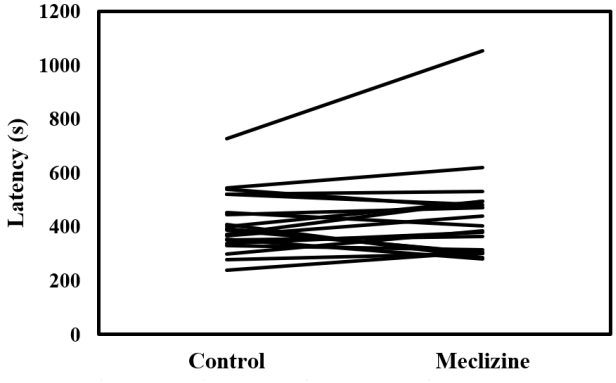

Figure 3
Latency versus mass of mice; oxygen toxicity latency did not vary with mass for either control (solid line) or meclizine-treatment (dashed line) groups. s – seconds; g – grams

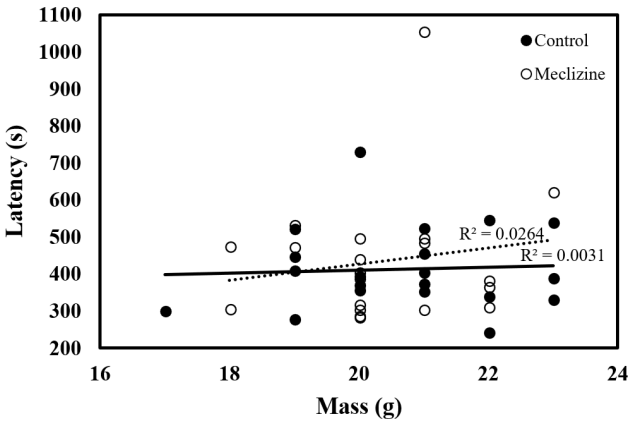

Table 1
Latency (s) to onset of seizures in control and meclizine-treated mice exposed to hyperbaric oxygen; s – seconds; g – grams

Group	Body mass (g)		Latency (s)	
	Mean (SD)	*P*	Mean (SD)	*P*
All sessions	20.5 (1.45)	–	424 (146)	–
Control	20.7 (1.60)	0.520	414 (113)	0.371
Meclizine	20.4 (1.31)		434 (174)	

for individual mice did not exhibit any clear trend, as may be seen in Figure 2.

Regarding the influence of body mass, there was no statistically significant difference between control and meclizine groups (mean 20.7 [SD 1.60] g and 20.4 [SD 1.31] g, $P = 0.52$). The mean chronological change in body mass between the sessions was + 0.50 (SD 1.96) g, $P = 0.27$. The dependency of latency to toxicity on body mass was also examined. Linear regression demonstrated no correlation between latency and body mass for either control or meclizine groups ($r^2 = 0.0031$, $P = 0.81$; $r^2 = 0.0026$, $P = 0.49$, respectively), as shown in Figure 3.

Latency and body mass data are shown in Table 1.

Discussion

Closed-circuit divers using pure oxygen rebreather apparatus have an elevated risk of CNS-OT. In many cases, these divers also develop motion sickness on their way to a dive location. To cope with seasickness, the use of medication is common. Some relevant drugs such as meclizine contain anticholinergic compounds, potentially increasing the risk of CNS-OT. The present study aimed to evaluate the effect meclizine may have on CNS-OT as defined by the clear appearance of tonic-clonic seizures in rats.

The main finding of the present study is that meclizine had no effect on the latency to CNS-OT resulting from high pO_2. To interpret this result, meclizine's pharmacology should be considered. Meclizine operates via different pathways to prevent motion sickness. It is considered to affect both histaminergic and cholinergic pathways.[25] Several studies have been conducted to examine the specific site of action of this drug. Although meclizine is defined as an antihistamine having additional antim uscarinic potency, it shows low affinity for the muscarinic receptors.[9] The antiemetic action of meclizine is attributed for the most part to blocking of the H1 histamine receptor.[26] Therefore, the mechanisms of action of this drug may explain the main result of the present study. Further support is provided by past studies which have also concluded that certain anticholinergic activity does not reduce the latency to CNS-OT.[16,17]

Since there was some variability in body mass, further analysis was undertaken to find out if this may have affected the results. The statistical analysis did not reveal a correlation

between body mass and latency to CNS-OT, in agreement with the literature. Arieli reviewed data of body mass and time to convulsion from several studies.[27] His analysis found that under hyperbaric conditions, time to convulsion did not correlate with body mass, either within a species or between species. Metabolic rate and free radical production, both at the basis of the biochemical mechanism for CNS-OT, increase with body mass. Arieli theorised that although free radical production increases, antioxidant production rates may also increase with body mass.[27] This would support the present study's main result.

Though the mouse and other small mammals are widely used in CNS-OT research,[3] the effects on humans may not be directly deduced due to differences in physiology. The acetylcholine antagonist scopolamine was tested for its effect on CNS-OT in rats over 30 years ago, and results did not show an increased risk.[16] Since then, scopolamine has become widely used by divers prior to oxygen diving. In this time, there have not been any reported CNS-OT cases involving this drug. With this knowledge and the results of the present study, a future human study can be conducted.

Conclusions

The results of the current study did not indicate any effect of meclizine in development of CNS-OT, as observed in mice. This may suggest that the pharmacological pathway and mechanism of this medication are not involved in the events leading to diving-related tonic-clonic seizures.

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Trends in competitive freediving accidents

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Keywords
Barotrauma; Blackout; Breath-hold diving; Central nervous system; Hypoxia; Loss of consciousness; Safety

Abstract
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Introduction: Understanding safety issues in competitive freediving is necessary for taking preventive actions and to minimise the risk for the athletes.
Methods: We analysed occurrence of loss of consciousness (LOC) and pulmonary barotrauma (PBt) in various freediving disciplines in 988 competitions over five years (from 2019 to 2023 inclusive), with 38,789 officially registered performances (starts): 26,403 in pool disciplines and 12,386 in depth disciplines.
Results: Average incident rate in competitive freediving (all cases: LOCs plus PBt, 2019–2023) was 3.43% (1,329 incidents / 38,789 starts). The average incident rate of LOC and PBt within five years were 3.31% and 0.38% respectively for all disciplines. Two disciplines present higher risk for LOC: dynamic without fins (DNF) (mean risk ratio (RR) = 1.48, 95% CI, 1.13 to 1.96, $P < 0.01$) and constant weight without fins (CNF) (mean RR = 2.02, 95% CI, 1.39 to 2.94, $P < 0.001$). The RR for PBt was not higher in any discipline. The overall risk of all types of incidents (LOC plus PBt) was also higher for DNF (mean RR = 1.55, 95% CI, 1.18 to 2.04, $P < 0.01$) and CNF (mean RR = 2.80, 95% CI, 1.70 to 5.04, $P < 0.001$).
Conclusions: The disciplines without fins in the pool (DNF) and at depth (CNF) appear to be the most dangerous in terms of LOC. We may recommend that organisers and safety teams should pay a special attention to no-fin disciplines as most risky for possible LOC.

Introduction

Freediving is an activity that dates back thousands of years. In the last ten years, there is a boom in competitive freediving worldwide. Understanding related safety issues is important, to take preventative measures for minimising the risk for athletes.

The competitions in freediving may be organised in a pool or in the sea. Pool disciplines are static apnoea (STA), dynamic no fins (DNF) and dynamic with fins (DYN). Static apnoea is a holding of breath at the surface of the water without any movement for as long as possible; dynamic apnoea consists of covering the greatest distance horizontally with a monofin (DYN), two fins (bifins) (DYNB) or without fins (DNF) on a single breath of air. Depth disciplines are constant weight no fins (CNF), free immersion (FIM), constant weight with a monofin (CWT) and constant weight with bifins (CWT_B). The objective of the depth disciplines is to descend as deep as possible with constant weight without external assistance, with the exception of FIM which involves descending and ascending pulling a rope by hand. Current freediving records in each discipline are shown on the AIDA International (Association Internationale pour le Développement de l'Apnée) website (https://www.aidainternational.org/).

At the end of a record attempt, on surfacing, a freediver needs to demonstrate cognitive integrity and good physical condition by presenting the 'surface protocol' to judges. An athlete must remove all facial equipment (mask, nose clip), show the 'OK' sign and say 'I am OK', all within 15 sec from surfacing (starting from the emerging athlete's airway being above water). The freediver's airway should remain above the water throughout the whole protocol. If a freediver does not perform the surface protocol appropriately, an attempt is not counted. There are several reasons why the physical and mental condition of an athlete may be compromised at the end of performance. For example, the excellent diving reflex pertinent to high-level freedivers enables them to save their oxygen reserves,[1] but even top sportsmen may find themselves in deep hypoxic situations and may lose consciousness[2] with drop in arterial partial pressure of oxygen (PaO_2) to dramatically low levels.[3,4] Loss of consciousness (LOC) may occur at surface or at depth, but tends to occur commonly near the end of the dive as the freediver nears the surface – referred to as 'shallow-water

blackout'. Despite of understanding that LOC leads to disqualification and willing to escape this scenario, almost 10% of freedivers were disqualified because of LOC at depth competitions between years 1998 and 2004.[5] Freedivers may also suffer from pulmonary barotrauma (PBt) due to the extreme pressure they have to cope with during deep dives.[6] Freedivers with PBt ('lung squeeze') may have a cough and a sensation of chest constriction accompanied with dyspnoea.[7,8] Symptoms of PBt have been reported in up to 25% of freedivers after repeated diving sessions.[9] Repetitive breath-hold diving may increase transpulmonary capillary pressure and this increasing could lead to non-cardiogenic oedema and alveolar haemorrhage.[8] Despite the importance of assessing the risk for the athletes, there are no recent data on freediving incidents observed at competitions. Long-term consequences of freediving injuries and their impact on athletes' health also are limited and are required further investigation in other studies.

The objective of the study was to evaluate the prevalence and risk of freediving-related incidents during competitions and to determine which competitive freediving discipline(s) are at higher risk of incidents. We intend that the results of the study should contribute into assessment of the overall situation with the safety of freediving competitions, and be taken into consideration by coaches, medical teams, organisers of competitions and support the improvement of the competition rules and regulations.

Methods

Data were obtained from open public sources. Approval of the Ethical Committee and the informed consent of subjects was not required. The Medical and Science Committee and the Board of AIDA International provided the authorisation to use the competition data for the purpose of this study.

PARTICIPANTS

Data were collected from the official results of all competitions worldwide organised by AIDA International from 2019 to 2023, inclusive, (https://www.aidainternational.org/). All competitors had a medical certificate allowing them to practice freediving and participate in freediving competitions. A competitor should announce planned performance (time of breath hold, distance, or depth; the official term is 'announced performance') before an attempt. If an athlete does not reach the announced result, penalty points are awarded and the diver is presented with a yellow card by the judge. The announced performance (time of breath hold or distance) may be exceeded in the pool but not in the depth disciplines (the rules prevent that).

GENERAL ASSESSMENT OF ATTEMPT BY JURY

The assessment of a performance by the jury through presenting a card of a different color (white, yellow, or red) doesn't reflect the medical condition of an athlete directly. If an athlete can't complete the surface protocol appropriately, an attempt is not counted (a red card is shown by the judges). Inability to perform the surface protocol which leads to a red card may be connected not only to hypoxia but, for instance, to the lack of competitive experience or to the activities of third parties (for instance, touch of an athlete by safety diver, which is prohibited by the competition rules). A serious deterioration of an athlete's health may lead not only to a failed attempt but to the disqualification from the whole competition. A yellow card typically means that some rules are violated, or the result of the performance is below the announced one. It doesn't reflect the medical condition of an athlete as well. A white card means that all rules and requirements have been met. We considered that the analysis of the jury assessments (cards) could be of interest.

DESIGN

Data were analysed for number of countries where competitions were performed, number of competitions, number of dives, and for success of performance (number of red, yellow and white cards). Frequency of freediving incidents (surface LOC, underwater LOC, pulmonary barotrauma) was analysed in the following pool freediving disciplines: STA, DYN, DYN_B, DNF (pool), and in the following depth freediving disciplines: CWT, CWT_B, CNF and FIM. The decision whether LOC occurred was made by judges who directly observed an athlete after surfacing. The judges were assisted with videotaping of the surfacing, to review any questionable cases. Decisions regarding the occurrence of PBt was made by groups of competition medics who performed the medical examination of an athlete if PBt was suspected.

SAMPLE SIZE

Sample size calculation wasn't performed. All available data were collected from the official results of all competitions organised by AIDA International from 2019 to 2023, inclusive.

STATISTICAL ANALYSIS

Differences in disciplines, type of card, or gender were tested using two- or 3-way ANOVA. For *post-hoc* tests, Scheffé's method with the Bonferroni correction were applied. Underwater LOCs and surface LOCs were analysed in combination and separately. Because of the lack of yearly data on the performances with bifins, data for DYN with a monofin was combined with DYN_B (DYN+B), and CWT with monofin were combined with CWT_B. The relative risk (RR) for the LOCs, PBt and all accidents were calculated by comparing the frequency of accidents for each discipline with the annual pool and depth incidents obtained for each discipline. The RR, its standard error and 95% confidence interval were calculated according to Altman.[10] For RR,

the *P*-value is calculated according to Sheskin.[11] Statistical analyses were performed with SPSS (Version 21.0). The data are presented as means and standard deviation (SD). Differences with *P*-value less than 0.05 were considered significant.

Results

POPULATION

From 2019 to 2023, AIDA International authorised 988 competitions (mean 197, SD 75 competitions per year). The competitions took place in 59 countries, athletes of 116 nationalities participated in these events. In all competitions, there were 38,789 officially registered performances (attempts) in all disciplines (26,403 in pool disciplines and 12,386 in depth disciplines); 23,331 men's starts (60.2%) and 15,458 women's starts (39.8%). All results from all competitions were analysed for the purposes of the study (no missing data).

ALL INCIDENTS

The average incident rate in competitive freediving (all cases: LOCs plus PBt) over five years (from year 2019 to 2023 inclusive) was 3.43% (1,329 incidents in 38,789 starts). Data for each discipline are presented in Table 1.

LOSS OF CONSCIOUSNESS

The average incident rate of LOC (surface plus underwater) over the five year period was 3.31% (1,282 incidents in 38,789 starts) for all disciplines: 3.22% (850 incidents in 26,403 starts) in pool disciplines and 3.49% (432 incidents in 12,386 starts) in depth disciplines. Surface LOC occurred in 2.51% (all disciplines); 3.00% in pool disciplines and 2.01% in depth disciplines. Underwater LOC occurred in 1.91% (all disciplines); 2.44% in pool disciplines and 1.38% in depth disciplines. Loss of consciousness data are presented in Tables 2 and 3. A Pareto chart indicates that CNF and DNF were respectively responsible for 35% and 17% of LOC events (Figure 1).

PULMONARY BAROTRAUMA

The incident rate for PBt is presented in Table 4, with an average occurrence of 0.38% over five years (47 incidents in 12,386 starts). A Pareto chart indicates that CNF was responsible for 58% of PBt (Figure 2).

RISK OF DIFFERENT DISCIPLINES

Two disciplines present higher risk for LOC occurring either underwater or at the surface: DNF (mean RR = 1.48; 95% CI, 1.13 – 1.96; $P < 0.01$) and CNF (mean RR = 2.02;

Table 1

Percentage of attempts resulting in loss of consciousness (LOC) (surface or underwater) or pulmonary barotrauma (PBt) by disciplines; CNF – constant weight without fins; CWT (+ B) – constant weight with a monofin or two fins; DNF – dynamic without fins; DYN (+ B) – dynamic with a monofin or two fins; FIM – free immersion; STA – static apnoea

Discipline	Percent incidents (LOC plus PBt)					
	2019	2020	2021	2022	2023	Total mean
STA	2.45	4.42	3.44	3.69	2.24	3.13
DNF	4.60	5.59	5.46	4.48	5.08	4.85
DYN (+B)	2.82	3.41	3.80	2.51	2.87	2.67
CNF	7.74	4.63	4.42	6.57	9.26	7.82
FIM	2.31	1.64	4.78	3.84	4.10	3.33
CWT (+B)	3.53	0.48	3.22	1.51	3.22	2.36
Total mean	3.89	3.36	4.79	3.70	4.39	3.43

Table 2

Percentage of attempts resulting in surface or underwater loss of consciousness (LOC) incidents in pool disciplines; DNF – dynamic without fins; DYN (+ B) – dynamic with a monofin or two fins; STA – static apnoea

Discipline	Percent incidence of LOC in pool disciplines					
	2019	2020	2021	2022	2023	Total mean
STA	2.40	4.42	3.01	3.63	2.16	3.13
DNF	4.53	5.59	4.90	4.27	4.98	4.85
Dyn (+B)	2.82	3.41	1.88	2.39	2.84	2.67
Total mean	3.25	4.48	3.26	3.43	3.33	3.55

Table 3
Percentage of attempts resulting in surface or underwater loss of consciousness (LOC) incidents in depth disciplines; CNF – constant weight without fins; FIM – free immersion; CWT (+ B) – constant weight with a monofin or two fins

Discipline	Percent incidence of LOC in depth disciplines					
	2019	2020	2021	2022	2023	Total mean
CNF	7.16	2.78	10.09	5.51	8.48	6.80
FIM	2.09	0.98	4.40	3.14	3.61	2.84
CWT (+B)	3.39	0.48	2.94	1.37	2.89	2.21
Total mean	4.21	1.41	5.81	3.34	4.99	3.95

Figure 1
Pareto chart of surface and underwater loss of consciousness (LOC) across freediving disciplines between 2019–2023; CNF – constant weight without fins; CWT (+ B) – constant weight with a monofin or two fins; DNF – dynamic without fins; DYN (+ B) – dynamic with a monofin or two fins; FIM – free immersion; Static – static apnoea

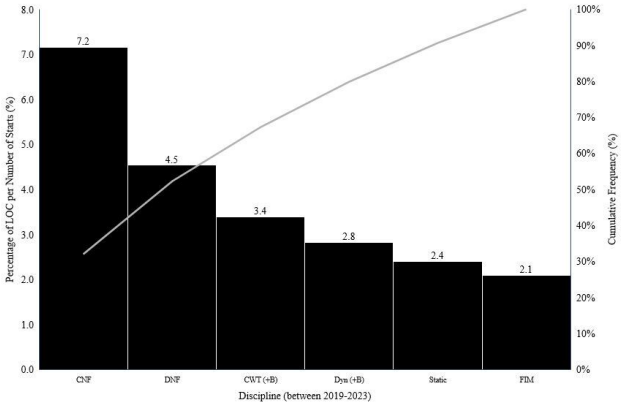


Figure 2
Pareto chart of pulmonary barotrauma (PBt) by freediving disciplines from 2019 to 2023; CNF – constant weight without fins; CWT (+ B) – constant weight with a monofin or two fins; DNF – dynamic without fins; DYN (+ B) – dynamic with a monofin or two fins; FIM – free immersion; Static – static apnoea

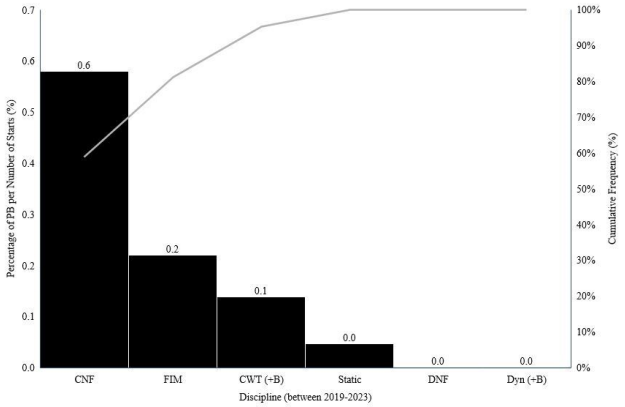


Table 4
Pulmonary barotrauma (PBt) incidents in depth disciplines; CNF – constant weight without fins; CWT (+ B) – constant weight with a monofin or two fins; FIM – free immersion

Discipline	Percent incidence of PBt in depth disciplines					
	2019	2020	2021	2022	2023	Total mean
CNF	0.01	0.02	0.01	0.01	0.01	0.01
FIM	0.22	0.66	0.38	0.70	0.49	0.49
CWT (+B)	0.14	0.00	0.41	0.14	0.28	0.19
Total mean	0.06	0.11	0.13	0.14	0.13	0.38

95% CI, 1.39–2.94; $P < 0.001$). The other disciplines did not present a greater LOC risk. The RR for PBt was not higher whatever the disciplines and years. The overall risk of all types of incidents also remains high for DNF (mean RR = 1.55; 95% CI, 1.18–2.04; $P < 0.01$) and CNF (mean RR = 2.80; 95% CI, 1.70–5.04; $P < 0.001$).

GENERAL ASSESSMENT OF ATTEMPT BY JURY

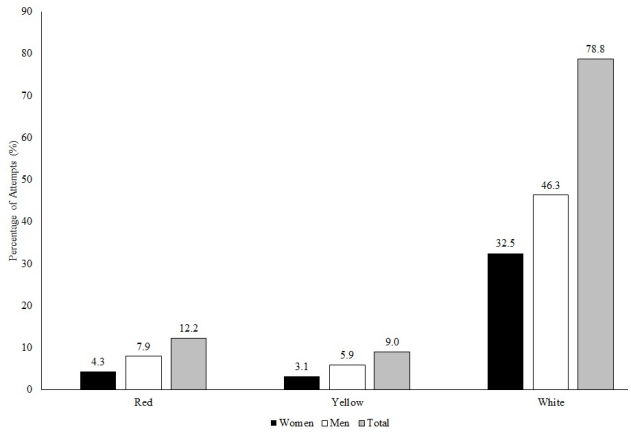
The average percentages of attempts receiving red, yellow, and white cards are shown in Figure 3. Static apnoea has a significantly higher proportion of red and yellow cards than other disciplines (F = 21.33; $P < 0.001$), with fewer cases in women (F= 34.21; $P < 0.05$). Next come CNF and DYN (+B), with no difference between men and for women.

Discussion

These results show that between 2019 to 2023, 1,282 out of 38,789 (3.31%) attempts in international freediving competitions were not counted due to the loss of consciousness or inability to perform the surface protocol

Figure 3

Mean percentage of red, yellow, and white cards issued across freediving disciplines from 2019 to 2023; results are presented by sex (men and women) and combined total; red – disqualified; yellow – rule violations; white – successful performance



through manifestations such as loss of motor control. During the same observational period, PBt occurred after 47 of 12,386 (0.38%) starts. The RRs of LOC were higher in no fin disciplines (DNF and CNF) with no difference for the other disciplines.

To our knowledge, this is the first study of the occurrence of freediving incidents during competitions occurring over an extended period and with the analysis of full set of data obtained from reliable official sources (all competitions under auspices of AIDA International, all disciplines, all incidents). Although we didn't perform direct comparison, it is interesting to note that there are less red and yellow cards in women than in men, especially in STA and CNF. This difference may be explained by different risk-taking between men and women, as anecdotally observed by our team.

If we compare the results of our study with the results of a similar study,[5] our study found a lower incidence of LOC (3.31% vs 9.7–11.1% respectively). That study did not include PBt. The different result for LOC may relate to the difference in sample size (596 in Lindholm's study[5] versus 38,789 in our study). We may conclude that most risky disciplines are those without fins, both in depth disciplines and in the pool. This observation may be speculatively explained on the basis that no-fin disciplines require a greater oxygen consumption due to the recruitment of a greater number of muscle groups. This increased oxygen consumption leads to more rapid desaturation of arterial oxygen.[12] The rapid drop in oxygen levels likely predisposes to syncope and may impair the efficiency of the dive reflex, creating a conflict between oxygen delivery to active muscles and the brain.[13]

There is some evidence that repeated hypoxic events can progressively alter neurocognitive functions. Billaut et al.[14] observed mild executive dysfunction positively correlated with the duration (years) of practicing apnoea. Potkin and Uszler[15] used brain imaging and suggested abnormalities in the brain functions in five elite breath hold divers. In contrast, Doerner et al.[16] found no mid-term morphological changes in the brains of 17 elite freedivers. Other researchers attempted to assess the impact of hypoxia on brain with several brain markers. Liner and Andersson[17] observed high levels of S100B (a serum marker of cerebral ischaemia and brain damage) within five days after a dynamic apnoea attempt ending with LOC, and this observation may suggest long-term negative consequences of severe brain hypoxia in freedivers.[18] Gren et al.[19] showed that the amyloid precursor protein (Tau) associated with neuronal damage or dysfunction, is accumulated in plasma after long static apnoea. In trained freedivers, dynamic cerebral autoregulation is acutely impaired during maximal breath hold attempts.[20] A decrease in cerebral oxidative metabolism and disruption of the blood-brain barrier may also occur.[21–23] Bailey et al,[24] found the persistence of functional-structural destabilisation of the blood-brain barrier (BBB) in elite freedivers periodically exposed to extreme hypoxia. Thus, some evidence indicates that repeated, prolonged apnoeas may lead to minor BBB disruption and neuronal parenchymal damage, increasing the possibility of at least mild neurocognitive sequelae. Thus, a risk to freediver's brain can be hypothesised. Since disciplines without fins appear to present a higher risk than other disciplines, it could be of interest to investigate elite freedivers holding the records in DNF and CNF.

Our study demonstrates the relatively low risk of PBt, and this condition was found to occur only in depth disciplines, not in the pool. It is possible that risk may be underestimated because while clinically significant cases of PBt are recorded, milder cases, when only the athlete is aware of symptoms, often go unnoticed by the event medics. Athletes do not report these symptoms to the physician due to fear of being disqualified from the competitions. The medical consequences after mild or moderate PBt may be important. A suggestion that PBt increases the risk of LOC by limiting oxygenation during ascent should be investigated. Another sign of PBt is haemoptysis after alveolar haemorrhage due to cardiovascular changes that occur during deep apnoea dives. High ambient pressure in depth and exposure to a cold environment increase intrathoracic blood volume and cardiac output, as well as pulmonary capillary pressure. The risk of alveolar haemorrhage is also increased by negative pressure inside the alveoli due to involuntary breathing movements during the late phase of an apnoea dive. Haemoptysis has been self-reported in one-fifth of freedivers.[9] Blood clots which probably arrived from the lower respiratory tract were observed by laryngoscopy in freedivers who dived to a depth of 6 m after complete exhalation to residual volume to simulate thoracic squeeze.[25] Haemoptysis after deep dives may present as a single symptom or occur together with cough and dyspnoea, which are the symptoms of pulmonary oedema.[26,27]

LIMITATION

Unfortunately, we have no access to the full data set by gender and discipline. For this reason, we propose not to include the percentages for men and women separately for each discipline.

Conclusions

The study presents first analysis of five-year prevalence of two important freediving adverse medical events, LOC and PBt, in different freediving disciplines. The disciplines without fins in the pool (DNF) and at depth (CNF) appear to be the most dangerous in terms of LOC. We may recommend that organisers and safety teams should pay a special attention to no fin disciplines as most risky for possible LOC. Athletes should carefully consider announced performance and freediving training technique: movements of the legs and arms should not only be effective, but also as relaxed as possible to minimise oxygen consumption and, most importantly, to prevent sharp reduction of arterial PO_2.

The international freediving federations, AIDA International and CMAS, have competition rules with a section about assessment of the incidents and follow-up actions. The statistics about freediving incidents should be taken in consideration when the competition rules are under periodic revision. If the number of freediving incidents is increasing, the rules should be amended, for instance, to tighten the conditions for further participation of athletes in competitions after serious incidents.

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Divers treated in Townsville, Australia: worse symptoms lead to poorer outcomes

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Keywords
Decompression illness; Decompression sickness; Hyperbaric oxygen treatment; Oxygen; Recompression; Scuba diving

Abstract
(Blake DF, Crowe M, Lindsay D, Turk R, Mitchell SJ, Pollock NW. Divers treated in Townsville, Australia: worse symptoms lead to poorer outcomes. Diving and Hyperbaric Medicine. 2024 20 December;54(4):308–319. doi: 10.28920/dhm54.4.308-319. PMID: 39675739.)
Introduction: Hyperbaric oxygen treatment (HBOT) is considered definitive treatment for decompression illness. Delay to HBOT may be due to dive site remoteness and limited facility availability. Review of cases may help identify factors contributing to clinical outcomes.
Methods: Injured divers treated in Townsville from November 2003 through December 2018 were identified. Information on demographics, initial disease severity, time to symptom onset post-dive, time to pre-HBOT oxygen therapy (in-water recompression or normobaric), time to HBOT, and clinical outcome was reviewed. Data were reported as median (interquartile range [IQR]) with Kruskal-Wallis and chi-square tests used to evaluate group differences. Significance was accepted at $P < 0.05$.
Results: A total of 306 divers (184 males, 122 females) were included with a median age of 29 (IQR 24, 35) years. Most divers had mild initial disease severity ($n = 216$, 70%). Time to symptom onset was 60 (10, 360) min, time to pre-HBOT oxygen therapy was 4:00 (00:30, 24:27) h:min, and time to start of HBOT was 38:51 (22:11, 69:15) h:min. Most divers (93%) had a good (no residual or minor residual symptoms) outcome and no treated diver died. Higher initial disease severity was significantly associated with shorter times to symptom onset, oxygen therapy, and HBOT, and with worse outcomes. The paucity of cases receiving HBOT with minimal delay precluded meaningful evaluation of the effect of delay to HBOT.
Conclusions: Most divers had mild initial disease severity and a good outcome. Higher initial disease severity accelerated the speed of care obtained and was the only factor associated with poorer outcome.

Introduction

The Great Barrier Reef (GBR) is one of the most popular places to dive in Australia. It is the largest living structure on the planet and extends for 2,300 kilometres along the Queensland coastline. With 2.4 million visitor days per year, the GBR provides 64,000 jobs and contributes $6.4 billion to the Australian economy in annual revenue.[1] Diving is a relatively safe sport, but 483 fatalities were reported with the activity in Australia from 1970 to 2018, 116 in Queensland.[2] Although death is relatively rare from diving, many divers are injured each year requiring treatment in a hyperbaric facility. In 2018, 112 divers were treated for decompression illness (DCI) in Australia, 34 in Queensland (Hyperbaric Technicians and Nurses Association, unpublished data). Decompression illness is a collective term embracing decompression sickness (DCS) caused by bubble formation from dissolved gas, and arterial gas embolism (AGE) caused

by pulmonary barotrauma.[3] In this paper 'DCS' is used when the goal is to specifically refer to the consequences of bubble formation from dissolved gas, and the collective term 'DCI' is used to refer to both DCS and AGE.

Hyperbaric oxygen treatment (HBOT) is currently considered the definitive treatment for DCI.[3,4] Access to HBOT may be delayed due to the remoteness of a dive site and limited access to hyperbaric chambers. Delay to HBOT greater than three hours has been associated with poorer outcomes in severely injured divers.[5] The Townsville University Hospital operates the only hyperbaric chamber in north Queensland, providing physician advice and HBOT for injured divers from the Whitsunday Islands north to the Torres Strait as well as for divers from some of the surrounding Pacific Islands. However, no recent literature has been published on injured divers in north Queensland.

The aim of this retrospective review was to outline the incidence, care and outcome of injured divers referred to Townsville University Hospital hyperbaric medicine unit.

Methods

Ethics approval was granted from the Townsville Hospital and Health Service (LNR/2019/QTHS/51229) and James Cook University (H7767). The Townsville Hospital relocated to its current site in October 2001, with the installation of a new multi-place rectangular chamber (Fink Engineering Pty Inc., Warana, Queensland, Australia). Data from the first two years of service at the new site were presented at a diving medicine workshop.[6] This retrospective review includes all injured divers treated at the Townsville hyperbaric unit after the previous report, from 4 November 2003 through 31 December 2018. Yearly patient logs and electronic discharged summaries were reviewed to identify cases for inclusion.

Retrieval Services Queensland databases (Queensland neonatal emergency transport service, clinical coordination retrieval information system, and Brolga) were searched using key words and relevant diagnoses (cerebral arterial gas embolism, decompression [including illness and sickness], drown*, snorkel*, and scuba), hyperbaric med*, and offshore retrievals by rotary wing asset to identify cases. Identifying data (name, date of birth, and date of incident) were collected so that cases could be linked with hyperbaric unit data to ensure that no cases were missed or duplicated.

Individual charts were reviewed, and data extracted to pre-formatted forms. Where available, the Queensland state-wide diver injury assessment form provided valuable information (Appendix A [#]). Diver age, sex, region of origin, body habitus, medical history and known medication use was collected. Body habitus was classified using body mass index if height and weight data were available otherwise from clinical descriptions in the medical charts, passport photos, or staff memory. Diving history including qualification, reported number of previous dives, years of diving, previous DCI as well as a description of the incident dive (day of week, month, and place of incident, nature of the dive, dive team, breathing gas and circuit type, dive computer use, potential contributing factors, maximum depth, and total dive time) and symptom profile were obtained from medical records and dive logs. Due to the complexity of dive profiles and the lack of dive computer downloads, only maximum depth, and total dive time (also known as surface-to-surface or run time) were documented. If a dive log had depths recorded as fractions of a metre (e.g., 24.3 m) it was recorded that a dive computer had been used. Time of symptom onset was defined in two ways. First, using a binary definition: during the dive (symptom onset underwater during the dive) or post-dive (after arriving at the surface). Second, calculating an actual time duration from the time the injured diver arrived at the surface after the incident dive to the time of symptom onset. Due to the unavailability of details on time to symptom onset underwater, the time of arrival at the surface was used as the starting time point for calculating time to treatment for all divers.

Initial disease grade was classified as mild, moderate, or severe using a system developed in Townsville (Table 1).[6] This grading system is subtly different to the widely accepted paradigm that arose from the 2005 remote DCI workshop,[3] but it was adopted here for having been applied to the Townsville patients in 'real time' over the study period. Treatment details were collected including time to commencement of pre-HBOT oxygen therapy (if administered) after symptom onset.

Retrieval details were collected including platform (boat, rotary wing, fixed wing, or road) and type of retrieval (primary, secondary, or tertiary). Primary retrievals were classified as retrievals from a pre-hospital location. If a dive boat called for medical advice and was directed to return to shore, this was classified as a primary retrieval by boat. If the dive boat returned to shore without any urgency after completing their trip, this was not considered a retrieval. Secondary retrievals were defined as retrievals from a place of medical care to a second facility providing higher care. This may be a second retrieval leg after a primary retrieval or the transfer between two health care facilities after diver self-presentation. Tertiary retrievals were defined as transfers from a secondary site to a third facility. Road retrievals included ambulance, bus, or car. Time to start of HBOT following symptom onset, final diagnosis, and clinical outcome at completion of HBOT (characterised as in Table 2)[7] were determined.

[#] Appendix A can be found on the DHM Journal website: https://www.dhmjournal.com/index.php/journals?id=344

Table 1
Initial disease severity grade using the established Townsville Hospital categories;[6] mild and moderate symptoms are invariably decompression sickness (DCS) while arterial gas embolism events would be classified as severe

Severity	Definition
Mild	Symptomatic DCS with no objective signs except: Minor skin rash Lymphatic DCS Sharpened Romberg test less than 30 seconds
Moderate	Symptomatic DCS with subtle signs: Impaired higher function Impaired Romberg test Subjective sensory changes Minor weakness due to pain Cutis marmorata
Severe	Symptoms threatening life or mobility: Loss of consciousness Cardiopulmonary DCS Spinal DCS

Table 2
Clinical outcome classification at the end of hyperbaric oxygen treatment[7]

Well, no residual signs or symptoms
Minor symptoms, no functional significance
Residual symptoms, moderate impairment
Major incapacity
Dead

Two researchers (DB and RT) performed the data extraction. Forms were compared and consensus reached. Individual Retrieval Services Queensland records were accessed to clarify retrieval information not apparent in the hospital medical records. All collected data were de-identified and entered into an Excel worksheet, and subsequently exported into Statistical Package for the Social Sciences version 28.0.0 (SPSS®, IBM® Corporation, Armonk, New York, USA) for analysis.

ANALYSIS

Data are presented using frequencies and percentages for categorical variables, and median and interquartile range (IQR) for continuous variables as all data were not normally distributed as assessed using the Shapiro-Wilk test. The Mann-Whitney U test was used for analysis comparing time to symptom onset, oxygen commencement, and HBOT between divers with onset of symptoms at depth versus onset of symptoms after the dive. Comparison of initial disease grade with time to event data (symptom onset, oxygen delivery, and HBOT) and oxygen duration was completed using the Kruskal-Wallis test for analysis. Dunn's test was used for *post hoc* analysis with Bonferroni correction. Statistical significance was accepted at $P < 0.05$ for all tests. As data were missing from some medical records, the *n* presented throughout the results denotes the number of records for which the information was documented.

Figure 1
Number of injured divers by sex presenting for hyperbaric oxygen therapy at the Townsville hyperbaric medicine unit per calendar year during the study period 4 November 2003 through 31 December 2018

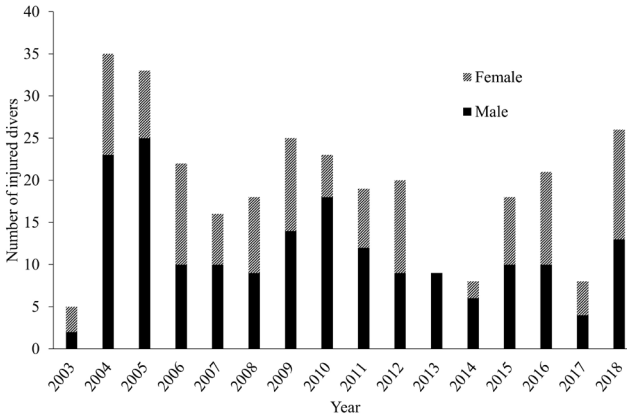
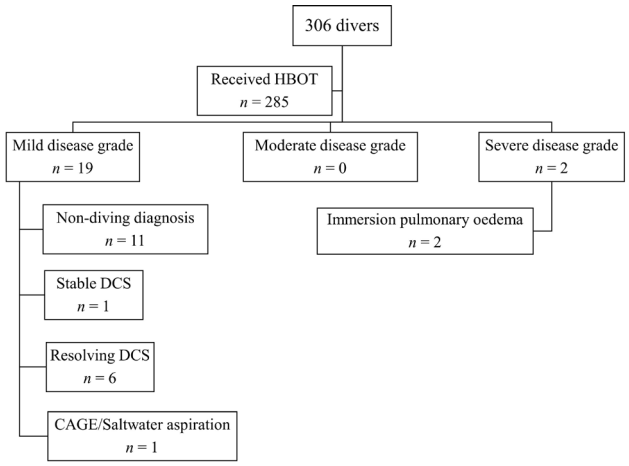


Figure 2
Breakdown of cases by initial disease grade of divers who did not receive hyperbaric oxygen treatment (HBOT); CAGE – cerebral arterial gas embolism; DCS – decompression sickness



Results

DIVER DEMOGRAPHICS

A total of 310 injured divers were identified during the study period. Four divers were excluded as their paper medical records had been destroyed following national medical record guidelines, one in 2003 and three in 2004, leaving

Table 3

Injured diver characteristics; data are *n* (%) unless otherwise specified; [a]World Health Organization regions; *n* = number of divers for which the data was documented in each category; DCI – decompression illness; IQR – interquartile range

Characteristic (*n* = 306)	
Male	184 (60)
Female	122 (40)
Median age (years)	29 (IQR 24, 35) (Range 14–74)
Region of origin[a] (*n* = 306)	
Western Pacific	159 (52)
Australia	143 (47)
Europe	103 (34)
Americas	43 (14)
Africa	1 (< 1)
Eastern Mediterranean	0
South-east Asia	0
Body habitus (*n* = 165)	
Underweight	5 (3)
Normal	135 (82)
Overweight	14 (8)
Obese	11 (7)
Relevant medical history	
Medical/Surgical history = yes (*n* = 292)	148 (48)
Medication use = yes (*n* = 248)	107 (43)
Diving qualification (*n* = 216)	
Uncertified	19 (9)
Student	25 (12)
Open water	57 (26)
Advanced	19 (9)
Rescue	13 (6)
Divemaster/Assistant instructor	16 (7)
Instructor	47 (22)
Commercial/Military	20 (9)
Relevant diving history	
Median number of previous dives (*n* = 198)	55 (IQR 9, 325) (Range 0–17,000)
Median years of diving (*n* = 75)	8 (IQR 2, 13) (Range 0–53)
Previous DCI = yes (*n* = 207)	41 (20)

306 divers for the analysis, displayed by year and sex in Figure 1. The term 'injured divers' is used intentionally to reflect the fact that some divers did not receive HBOT and had diagnoses other than DCI including cases considered to be suffering severe symptoms arising from immersion pulmonary oedema. (Figure 2). Most of the divers were from overseas, young, and certified with a wide range of reported previous diving experience (Table 3). Over half were male and the majority were of normal body habitus. A small number of divers reported having a previous incident of DCI (Table 3). Just under half of the divers had a history of a medical or surgical condition and many used a medication (acute or chronic) in the 48 hours before or after the incident dive (Table 3).

INCIDENT DIVE AND CONTRIBUTING FACTORS

Most incidents occurred during recreational dives (Table 4), during the summer (Southern Hemisphere) months, and on weekends (Friday through Sunday). Less than one-third of the incidents were in occupational divers and incidents were rare in scientific divers (Table 4). Just over half of the divers were diving with a buddy and the vast majority were breathing compressed air with open-circuit equipment. Only

Table 4
Incident dive characteristics; *n* = number of divers for which the data was documented

Characteristic	*n* (%)
Nature of dive (*n* = 260)	
Introductory	16 (6)
Certification course	55 (21)
Recreational	116 (45)
Occupational	69 (27)
Scientific	4 (1)
Technical	0
Dive team (*n* = 130)	
Solo	5 (4)
Buddy	68 (52)
Threesome	2 (1)
Group > 3	41 (32)
Surface support	14 (11)
Breathing gas (*n* = 177)	
Air	164 (93)
Nitrox 32%	6 (3)
Nitrox other %	6 (3)
Oxygen	1 (< 1)
Breathing circuit (*n* = 181)	
Open	158 (88)
Surface supply	22 (12)
Freediving	1 (< 1)
Dive computer (*n* = 160)	
Computer used	133 (83)

half of the medical records had documentation of whether a dive computer was used. In those with documentation, the majority used a computer (Table 4). The maximum dive depth recorded in 301 cases ranged from 1.8 to 50 metres of seawater (msw) (median 18, IQR 14, 25 msw). The total dive time recorded in 279 cases ranged from one to 210 minutes (min) (median 37, IQR 29, 45 min). Medical record documentation was poor for pre/post-dive contributing factors. However, dehydration and seasickness were commonly noted in those charts with documentation (Table 5). Possible contributing factors were varied, with repetitive dives and inadequate surface interval being the most frequently reported (Table 5).

SYMPTOMS AND TREATMENT AT SCENE

Most symptoms commenced after the incident dive (*n* = 275, 90%) rather than during the dive. The diagnoses of the divers with symptom onset during the dive were: 17 DCS, 10 cerebral AGE, one inner ear barotrauma and three immersion pulmonary oedema. All divers diagnosed with DCS had performed multiple dives often over several days. The median time to symptom onset post-dive (*n* = 269) was 1 hour (IQR 0:10, 6:00 h:min). One extreme outlier was identified. This diver had a time to symptom onset of 384 h. This diver was exposed to altitude by flying after diving, so the diagnosis of DCS was considered plausible, and the diver was recompressed, but the final diagnosis was

Table 5
Possible contributing factors for decompression illness pre-, during and post-dive; *n* = number of divers for which the data was documented; [a]factors listed on the Queensland diver assessment form; [b]cold or overheated combined as a single factor on the Queensland diver assessment form

Possible contributing factor	*n* (%)
Dehydration (*n* = 153)	88 (58)
Seasickness (*n* = 64)	37 (58)
Rough seas (*n* = 61)	32 (53)
Alcohol/Drug use (*n* = 133)	61 (46)
Possible contributing dive factor[a] (*n* = 306)	
Multiple repetitive dives (> 3 / day)	138 (45)
Surface interval < 120 min	136 (44)
Multi-day diving (> 3 consecutive days)	97 (32)
Rapid ascent	95 (31)
Excessive exertion	93 (30)
No safety stop	75 (25)
Reverse profile	72 (24)
Ear problems	67 (22)
Equipment problems	44 (14)
Violated computer/table guidance	35 (11)
Altitude exposure	32 (11)
Buoyancy problems	20 (7)
Thermal stress[b]	16 (5)

non-diving related. Most divers were classified as having mild initial disease grade (*n* = 216, 70%). Paraesthesia was the most common presenting symptom followed by arthralgia/myalgia and poor balance/ataxia (Figure 3). Half of the divers received treatment at the scene (*n* = 155/304), most commonly oxygen (143/155, 92%). Twenty-four divers (*n* = 24/155, 15%) had analgesia, 32 (*n* = 32/155, 21%) had fluids (30 oral, one intravenous and oral, one intravenous only) and two (*n* = 2/155, 1%) had antiemetics at the scene. Time to symptom onset was shorter for divers treated at the scene (*n* = 130) (median 20 min, IQR 00:05, 1:30 h:min) compared to the group of injured divers not treated at the scene (*n* = 139) (median 4 h, IQR 1, 16 h).

RETRIEVAL

One-third of the injured divers (*n* = 104) were primarily retrieved, half by boat (*n* = 52/104). More than three quarters of the injured divers required a secondary retrieval (*n* = 236), half by road. Only 24 injured divers had a tertiary retrieval.

RECOMPRESSION

A total of 285 (93%) of the injured divers received HBOT. Figure 2 depicts, by initial disease grade, the divers that did not receive HBOT. Nineteen of 216 injured divers initially classified as having a mild disease grade were not

Figure 3
Frequency of presenting symptoms of the injured divers who may have exhibited more than one symptom; the 'other' category consists of 26 discrete symptoms. LOC – level of consciousness

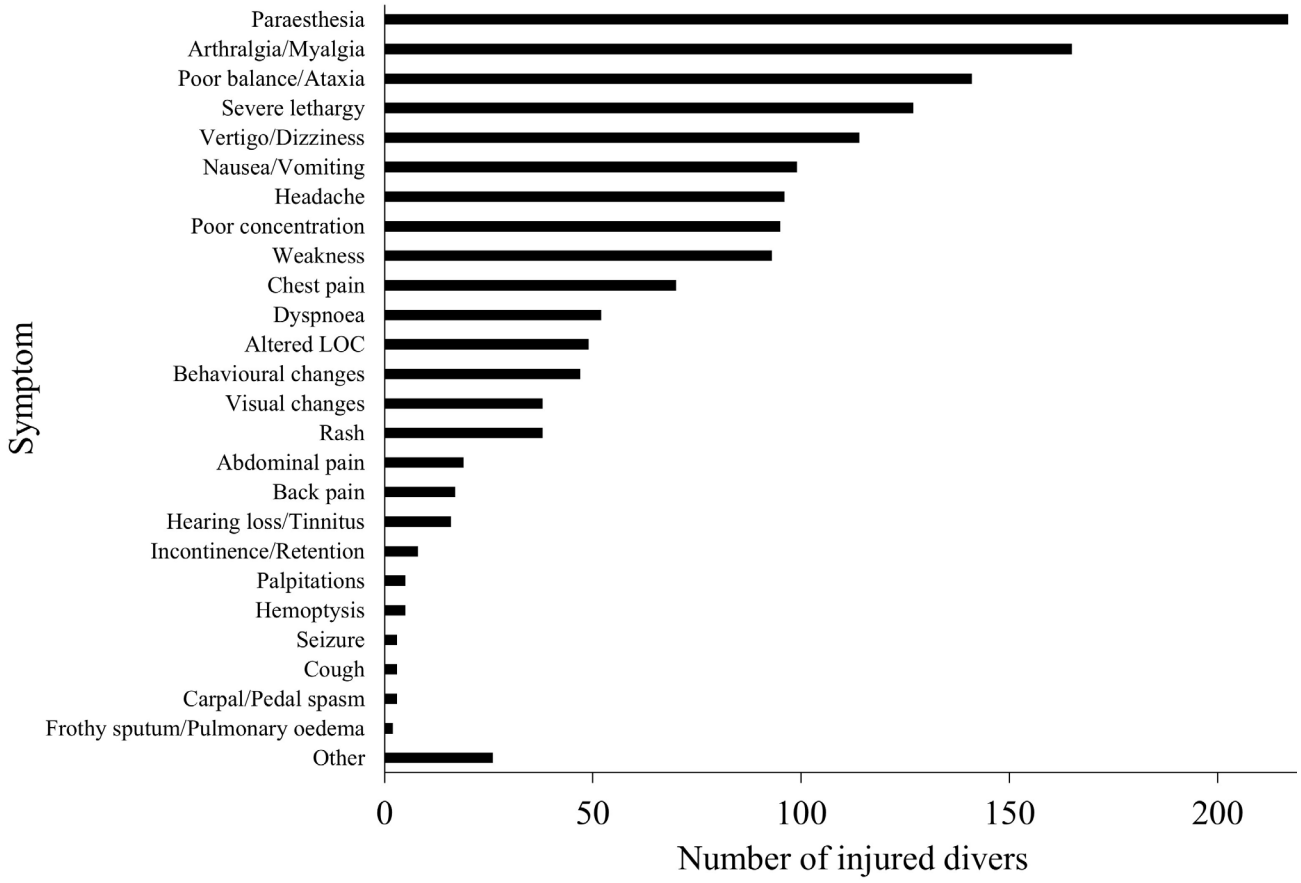


Table 6
Time to hyperbaric oxygen treatment (HBOT) post-symptom onset for all divers and subgroups; *P = 0.001 vs divers with symptom onset during the dive; IQR – interquartile range

Parameter	Median (IQR) (h:min)
Time to HBOT all injured divers, n = 283	38:51 (22:11, 69:15)
Time to HBOT for divers with symptom onset post-dive, n = 256	41:30 (22:26, 70:37)*
Time to HBOT for divers with symptom onset during the dive, n = 27	23:48 (9:45, 31:06)
Time to HBOT for divers treated at scene, n = 145	26:12 (17:20, 49:48)
Time to HBOT for divers primarily retrieved, n = 97	21:40 (10:30, 38:10)

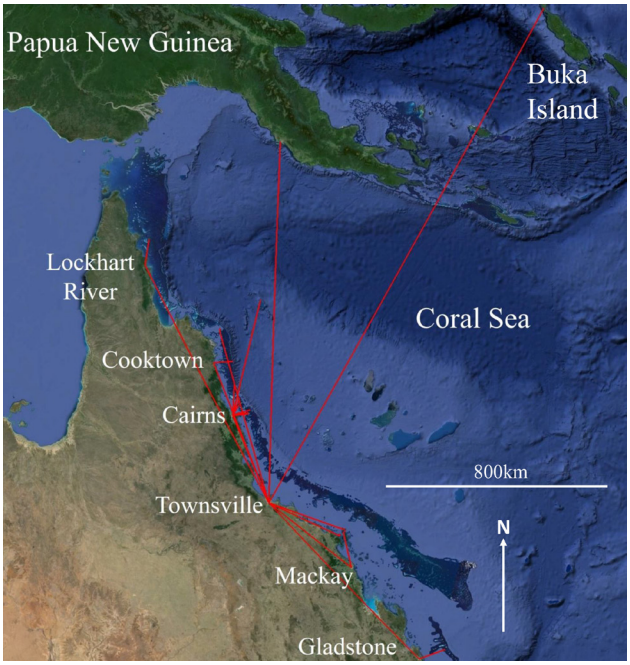
recompressed and for two the time to HBOT could not be calculated. Of these 19 non-recompressed divers, 11 had a non-diving related final diagnosis, seven had a final diagnosis of decompression sickness with one a disease evolution of stable and the other six resolving. One injured diver had a final diagnosis of possible cerebral AGE and saltwater aspiration. Due to a previous medical condition and resolution of symptoms, it was decided not to recompress this diver. All injured divers with initial disease grade of moderate (n = 57) were recompressed. Two injured divers classified as having initial severe disease grade (n = 33) were not recompressed. The final diagnosis in both cases was immersion pulmonary oedema. Divers with symptom onset during the dive had shorter times to HBOT compared to those with post-dive symptom onset (Table 6). Time to HBOT decreased as initial disease grade severity increased.

Of the 283 divers that underwent HBOT, none had HBOT commenced under three hours and only eight had HBOT commenced under six hours. Only 35 divers (12%) commenced HBOT under 12 hours and only 93 divers (33%) commenced HBOT under 24 hours. Three extreme

Table 7
Reasons for delays to hyperbaric oxygen treatment (HBOT); IQR – interquartile range; NBOT – normobaric oxygen treatment; * retrieval pathways for these cases are shown in Figure 4

Reason for delay	*n* (%)	Median (IQR) time to HBOT (h:min)	Initial disease severity		
			mild	moderate	severe
Delayed presentation for medical review	100 (35)	48:30 (29:58, 91:53)	83	15	2
Extreme retrieval distance (500 to > 1,700 km)	91 (32)	22:03 (11:48, 41:15)	42	27	22*
NBOT overnight then transferred	24 (8)	31:18 (23:11, 45:23)	20	4	0
Kept diving	22 (8)	84:45 (56:36, 244:11)	18	2	2
Initial misdiagnosis	15 (5)	73:32 (49:50, 134:56)	12	2	1
No delay	10 (4)	5:39 (3:32, 6:54)	3	3	4
NBOT with symptom reoccurrence	10 (4)	48:16 (30:08, 78:03)	9	1	0
NBOT overnight with morning HBOT	9 (3)	22:22 (19:34, 24:39)	7	2	0
Refused initial transfer	2 (< 1)	68:03 (24:31)	1	1	0

Figure 4
Retrieval pathways for divers with severe initial disease grade and extreme retrieval distance; *n* = 22



outliers were identified with a time to HBOT of greater than 373 h from symptom onset. All three divers were occupational divers who presented late for initial medical review. Reasons for delay to HBOT are listed in Table 7. Initial misdiagnosis and the lack of knowledge of the need for HBOT led to a delay in the referral and transfer of some divers to Townsville. Extreme retrieval distance was the most common reason for delay to HBOT for divers with severe initial disease severity. Retrieval pathways for these divers (*n* = 22) are shown in Figure 4. Only one of these divers was directly transferred to Townsville with the remaining 21 requiring more than one retrieval leg.

The initial recompression treatment table used was most often a modified Royal Navy (RN) 62 (US Navy treatment table 6), with only a small number of table extensions required (Table 8). Most divers required only a few treatments and had a good outcome (Table 8). Sixteen divers required more than 10 treatments, only one of these had mild initial symptoms (symptoms initially resolved on normobaric oxygen therapy) and only three had complete resolution of symptoms. Seven of these divers had a modified RN 62 as their first follow-up treatment table, four of whom had a Comex 30 as their initial treatment table. The other follow-up treatments were a combination of 180 kPa (100 min with 2 x 5 min air breaks) and 140 kPa (120 min with 2 x 5 min air breaks) treatment tables.

There were statistically significant differences between initial disease grade and time to symptom onset, time to oxygen commencement, and time to HBOT (Table 9). Divers with more severe initial disease grade had a shorter time to symptom onset, oxygen commencement, and HBOT. There was no statistically significant difference for duration of pre-HBOT oxygen therapy between the three initial disease grade groups ($P = 0.408$).

POSSIBLE CONTRIBUTING FACTORS FOR CLINICAL OUTCOMES

The small group of injured divers with major incapacity at the completion of hyperbaric treatment (*n* = 2) had severe initial disease grade, short times to symptom onset post-surfacing (1 min) and to oxygen commencement (5 min), and HBOT (6 h 49 min and 7 h 3 min). Due to small numbers, the clinical outcome groups 'moderate impairment' (*n* = 19) and 'major incapacity' (*n* = 2) were combined into one group for further analysis. There were no statistical differences between the clinical outcome groups for time to symptom onset, time to oxygen commencement or time to HBOT in the group of divers with severe initial disease grade (Table 10). There was

Table 8
Initial hyperbaric treatment table, number of treatments and clinical outcome; IQR – interquartile range; *n* = number of divers for whom the data was documented

Treatment parameter	*n* (%) or median (IQR), range
Initial treatment table (*n* = 285)	
Royal Navy 62	262 (92)
Comex 30	12 (4)
Other	11 (4)
Extensions and treatment numbers	
Table extension (*n* = 283)	30 (11)
Median (IQR) treatments (*n* = 285)	3 (2, 4), range 1–37
Clinical outcome (*n* = 306)	
Well, no residual signs or symptoms	147 (48)
Minor symptoms, no functional significance	138 (45)
Residual symptoms, moderate impairment	19 (6)
Major incapacity	2 (1)
Death	0

Table 9
Comparison of initial disease grade with timelines as specified (median IQR hours:minutes [h:min]); * Kruskal-Wallis test; # does not include injured divers with symptom onset during the dive; [a]significant difference between mild and moderate; [b]significant difference between mild and severe; [c]significant difference between moderate and severe; IQR – interquartile range; HBOT – hyperbaric oxygen treatment

Timeline	Mild	Moderate	Severe	*P*-value*
Time of symptom onset post-dive# (h:min)	2:00 (0:15, 8:00)[a,b] *n* = 199	0:15 (0:02, 2:00)[a] *n* = 51	00:10 (0:01, 1:00)[b] *n* = 19	< 0.001
Time to pre-HBOT oxygen start post-symptom onset (h:min)	9:00 (0:39, 31:57)[a,b] *n* = 169	1:27 (0:15, 11:48)[a] *n* = 54	00:15 (0:06, 3:40)[b] *n* = 31	< 0.001
Time to HBOT (h:min)	46:55 (26:10, 79:15)[a,b] *n* = 195	24:31 (12:10, 43:16)[a,c] *n* = 57	11:28 (7:57, 23:48)[b,c] *n* = 31	< 0.001

Table 10
Comparison of clinical outcome after completion of hyperbaric oxygen treatment (HBOT) with timelines as specified (median IQR hours:minutes [h:min]) for divers with initial severe disease grade; *Kruskal-Wallis test; [a]does not include injured divers with symptom onset during the dive; [b]one extreme outlier excluded due to a non-DCI final diagnosis

Timeline	No residual symptoms	Minor residual symptoms	Moderate / major residual symptoms	*P*-value*
Time to symptom onset post-dive[a] (h:min)	0:01 (< 0:01, 0:20) *n* = 6	0:01 (0:01, 3:00) *n* = 5	00:45 (0:01, 2:00)[b] *n* = 7	0.322
Time to oxygen start post-symptom onset (h:min)	0:10 (0:10, 3:00) *n* = 11	2:22 (0:07, 6:22) *n* = 12	00:11 (0:05, 5:16) *n* = 8	0.462
Time to start HBOT (h:min)	8:08 (6:57, 66:59) *n* = 9	16:53 (8:16, 23:33) *n* = 12	13:42 (7:40, 41:18) *n* = 10	0.347

a statistically significant association between initial disease grade and combined clinical outcome ($P < 0.001$, df 4). *Post hoc* analysis showed that divers with moderate or severe initial disease grades had poorer outcomes.

Discussion

Divers with higher initial disease grade had earlier time to symptom onset, oxygen commencement, shorter time to HBOT and poorer outcomes. These findings appear to be consistent with other studies where initial disease severity is

related to outcome, but other contributing factors are difficult to determine.[5,8,9] However, almost all the divers in this study had a substantial delay to HBOT, precluding meaningful evaluation of the effect of time to HBOT on clinical outcome.

Many factors (Table 5) have been proposed as possibly contributing to the risk of DCS and outcomes.[3,10,11] Numerous retrospective reviews[5,8,9,12–14] have reported the incidence of these factors, with one study reporting that 76% of injured divers had one or more contributing factors.[15] Despite ongoing attempts, there appears to be no consistent association between these proposed contributing factors and DCS risk or outcome. The retrospective nature of these studies probably greatly contributes to the difficulty in delineating pertinent risk factors. Incomplete documentation often leads to exclusion of cases[16,17] or possibly missing pertinent negatives in data sets as only positive responses are often recorded. Self-reporting would only include the items a diver believed to be a possible risk factor.[13,18] A prospective study collecting information on possible risk factors would greatly improve our understanding of risk and help focus educational opportunities for divers, dive operators, and dive medical personnel.

The divers in our study were largely young (Table 3), possibly reflecting the Australian backpacker (younger people travelling overseas, often on a working visa, staying in hostels) commonly taking scuba lessons and diving on the GBR. Injured divers are often young[12,14,16,19–21] especially compared to deceased divers. Divers Alert Network (DAN) fatality data from 2018 found a median age of 56 years of age[22] in deceased divers and Queensland data for 2000–2019 found a median age of 48 (IQR 32, 57) years.[2] Older divers are more likely to have medical conditions and poor physical fitness. Previous medical conditions are frequently listed in fatality reviews[2] but infrequently documented in retrospective reviews for divers treated for DCI. Health surveillance of recreational divers has been an issue discussed in the diving medicine fraternity;[23] however, any recommendations would be difficult to enforce. Divers are encouraged to be reviewed by a medical practitioner after a change in health.[23] Despite this recommendation, an online survey completed by DAN found divers with diabetes, cardiovascular, or respiratory disease rarely modified their diving practices or sought specialist advice.[24] Identifying medical or surgical conditions when divers are treated for DCI could provide an opportunity for discussion with a diving physician, potentially decreasing the risk of death in later years.

Many of the injured divers treated in Townsville were from overseas, possibly due to a regional phenomenon reflecting the high load of visitors who often participate in scuba courses to dive on the GBR. The percentage of overseas divers seems to be even higher than described in other tourist areas.[14,20] This is also reflected in the seasonality of presentations with more cases in the Australian summer months when the ocean water is warmer. In the northern hemisphere, higher call volume for advice is also found in the summer months.[22,25] More injured divers presented over the weekend days. This is unsurprising as dive trips are often planned around other commitments as weekend getaways.

The median time to symptom onset of an hour post-dive in our study was similar to that described in previous reports.[17,19] Other studies reported time to symptom onset of: 30 min,[13] 41 min,[16] and 90 min.[12] Divers in our study with a severe initial disease grade had shorter times to symptom onset. This was consistent with other studies focusing on divers with spinal cord DCS, the time to symptom onset from surfacing being considerably shorter: 5 min,[8] 10 min,[5] and 15 min.[9] Longer times to symptom onset have been associated with better outcomes,[5] while severe initial symptoms are associated with poorer recovery.[26] Together, short delays to symptom onset and severe symptoms should lead to prompt initiation of first aid treatment and arrangement for transport to a recompression facility.[3]

The most common presenting symptoms of paraesthesia and arthralgia/myalgia in our study are in keeping with previously published data.[3,16,21] These symptoms may be mild and vague, often making DCS difficult to diagnose by an inexperienced practitioner. The DAN America 'hotline' was established in 1980 to help injured divers by providing advice for both pre-hospital and hospital care.[22] An Australian hotline, called the Diver Emergency Service (DES), started operation in 1983 providing similar advice in the Asia-Pacific region. DAN World assumed responsibility for the Australian hotline in 2019. Phone advice can be obtained from DAN as well as directly from diving physicians around the world assisting with the diagnosis of diving related injuries, and guidance on treatment and disposition. This is a valuable service especially for centres that may not frequently care for injured divers.

The median time to HBOT in our study was considerable. Other studies have reported median times to HBOT of 6 h (Switzerland),[13] 24 h (Turkey),[16] 32.5 h (Poland),[19] and 2 days (New Zealand).[12] Consistent with our study, two studies in France found that divers with severe initial disease had shorter times to HBOT, 3 h[8] and 2 h 44 min.[5] Delay to recompression seems to increase the risk of incomplete recovery, but only in severely injured divers.[5,26] Previous research found an improvement in outcomes when divers with severe disease received HBOT within six hours.[26] A more recent study has found that divers with spinal DCS treated with HBOT within three hours of symptom onset had less sequelae at time of discharge.[5] None of the divers in our study had HBOT starting within three hours and only eight divers had HBOT commenced within six hours. Most divers in our study presented late for HBOT. Delayed HBOT, greater than 48 hours, has still been found to alleviate symptoms,[27,28] therefore delayed presentation should not preclude HBOT.

Many factors contributed to the delay to HBOT in our study (Table 7). Time to HBOT not only varies with initial disease severity but also by geographical location and distribution of hyperbaric facilities.[20,28] The Townsville hyperbaric unit covers a large geographical area with divers often in remote locations requiring long and complex retrievals. Further in-depth analysis of retrieval pathways will provide information on the factors leading to long retrieval times, identifying areas for improvement.

During our study period no formal follow-up occurred. Follow-up of divers with incomplete recovery is infrequently documented in retrospective reviews and often commented on in the limitations.[15,20] One study presented clinical outcome at one month post-injury in divers with spinal cord DCS, but the details of how this was done were not included.[8] Another study contacted divers treated over a two-year period, 1.5 to 3.5 years later.[21] In this study, 13 divers "*had reduced but lingering symptoms at discharge from hospital*".[21] Out of the 30 divers treated over the two-year period, 24 were contacted, one having died in a subsequent diving accident.[21] Six divers had residual symptoms at the time of contact, but interestingly three of these did not report having symptoms upon completion of their HBOT.[21] One diver suffered a concussion in the intervening years and it could not be determined if the reported symptoms were from DCS or the concussion. No other information was provided on possible reasons for recurrence of symptoms in the divers who had been free of symptoms on discharge.[21] In the current era of electronic communication, it would seem easier to contact previously treated divers whether they were local or tourists, though securing responses is likely to remain challenging. Historically, the Townsville HMU sent out follow-up letters to divers requesting information on clinical outcome and recurrence of symptoms during air travel. This information led to the changing of the advice on flying after hyperbaric treatment for DCI, decreasing the time to three weeks post completion of treatment from previous advice to wait for 4–6 weeks. Despite the reduction, this remains a very conservative recommendation. Follow-up letters are no longer sent to divers, and follow-up information was not documented in any of the charts in this review. Follow-up questionnaires could provide valuable data on recovery of divers especially those discharged with residual symptoms. At the time of discharge, divers are presented with a treatment summary and discussion ensues around returning to diving and flying. This would be an ideal time to verify electronic contact details and discuss the sending of a follow-up questionnaire. This would provide continuity of care for the divers and help with organising clinical review if necessary. Active follow-up of all treated patients would improve the knowledge of the incidence of ongoing permanent sequelae and allow for better prognostication and advice to patients on discharge.

RECOMMENDATIONS

Queensland has a state-wide diver injury assessment form (Appendix A[#]). The form contains information on assessing potential diving related injuries and a fillable section to enter information on dive profiles, risk factors, symptoms, physical assessment, and treatment provided. This form was designed to provide guidance for facilities infrequently encountering injured divers, providing a template of pertinent factors to be collected and discussed when referring to the hyperbaric facilities. Recommended changes to this form have been identified from this study. Thermal stress should be divided into cold and overheated and include the phase of dive at which this occurred.[4] There was poor documentation of thermal stress in the current study perhaps indicating a lack of knowledge of the role it may play in DCS risk. Current diving practice would indicate that a 60 min surface interval between dives is now considered standard, therefore, this item should be changed from 120 to 60 min. Lastly, documenting the incident dive location would assist in identifying high risk dive sites and allow for improved analysis of retrieval pathways and time to HBOT.

LIMITATIONS

This study was retrospective and limited by incomplete records and missing data. Missing data may have contributed to the difficulty to detect correlations between initial disease severity, contributing factors, timelines, and clinical outcomes. Time to treatment for divers with symptom onset during the dive may have been longer than reported as arrival at the surface was used as the starting point for timeline calculations. Divers are encouraged to return to the hyperbaric unit for review should symptoms reoccur, however, there was no attempt at follow up of divers after completion of their hyperbaric treatment, therefore final outcome is unknown. It is unknown if any injured divers were treated with normobaric oxygen therapy either on dive boats or at other health care facilities and not transferred to Townsville for treatment. Therefore, the true incidence of DCI in the Townsville catchment area is unknown.

Conclusions

This review describes 15 years of activity at the Townsville hyperbaric medicine unit. Most divers had mild initial disease severity, required few hyperbaric treatments and had a good outcome. Higher initial disease severity accelerated the speed of care obtained and was the only factor associated with poorer outcome. Improved documentation may enhance the ability to understand the impact of contributing factors on clinical outcomes.

[#] Appendix A can be found on the DHM Journal website: https://www.dhmjournal.com/index.php/journals?id=344

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Technical report

Dive medicine capability at Rothera Research Station (British Antarctic Survey), Adelaide Island, Antarctica

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Abstract

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Rothera is a British Antarctic Survey research station located on Adelaide Island adjacent to the Antarctic Peninsula. Diving is vital to support a long-standing marine science programme but poses challenges due to the extreme and remote environment in which it is undertaken. We summarise the diving undertaken and describe the medical measures in place to mitigate the risk to divers. These include pre-deployment training in the management of emergency presentations and assessing fitness to dive, an on-site hyperbaric chamber and communication links to contact experts in the United Kingdom for remote advice. The organisation also has experience of evacuating patients, should this be required. These measures, as well as the significant infrastructure and logistical efforts to support them, enable high standards of medical care to be maintained to divers undertaking research on this most remote continent.

Introduction

The first recorded dive under Antarctic ice was in 1902 to carry out ship repairs and, for the last six decades, diving has been integral to facilitating scientific study in the region.[1] The British Antarctic Survey (BAS) has undertaken scientific diving since the early 1960s.[2] In the mid-1990s, a marine science facility was completed at Rothera Research Station (RRS), where the BAS diving programme is currently located. Some of the environmental and technical considerations related to diving in Antarctica have previously been reported[3–6] and, in 1994, Milne and Thomson briefly summarised the medical care available to divers at Signy Research Station, where the majority of BAS diving was undertaken at the time.[7] While many of the issues encountered and risk mitigation measures are similar, our aim is to provide a comprehensive account of the current dive medicine capability at RRS.

Primarily the diving at RRS is for scientific research but other activities (e.g., hull inspections) are occasionally necessary. The hostile environment and remote location of RRS create challenges which must be met to ensure that the risk to divers is minimised as far as possible. Part of mitigating this risk includes the medical response to a dive incident and access to an on-site hyperbaric chamber.

Rothera Research Station

The station is located in the British Antarctic Territory on the Antarctic Peninsula at 67°34'8" S, 68°07'29" W (Figure 1). It is the largest BAS station and has been continuously occupied since 1975. Over recent years, construction workers have boosted the summer population to approximately 150 and over the winter this falls to about 25. The station serves as a key hub to deploy fieldwork projects across a large part of the continent, as well as hosting its own science programmes.

Diving at RRS

We examined the dive logs submitted to the BAS database for five years up to 1 June 2024. These recorded 651 dives. As the team do not dive alone, the number of person-dives is at least 1,302. This risk is not distributed evenly across the year, with more dives occurring during the austral summer (Figure 2).

Figure 1
Location of Rothera Research Station relative to potential evacuation routes. Figure produced by the Mapping and Geographic Information Centre, with data from the SCAR Antarctic Digital Database, 2024

Figure 2
Monthly dives over a five-year period

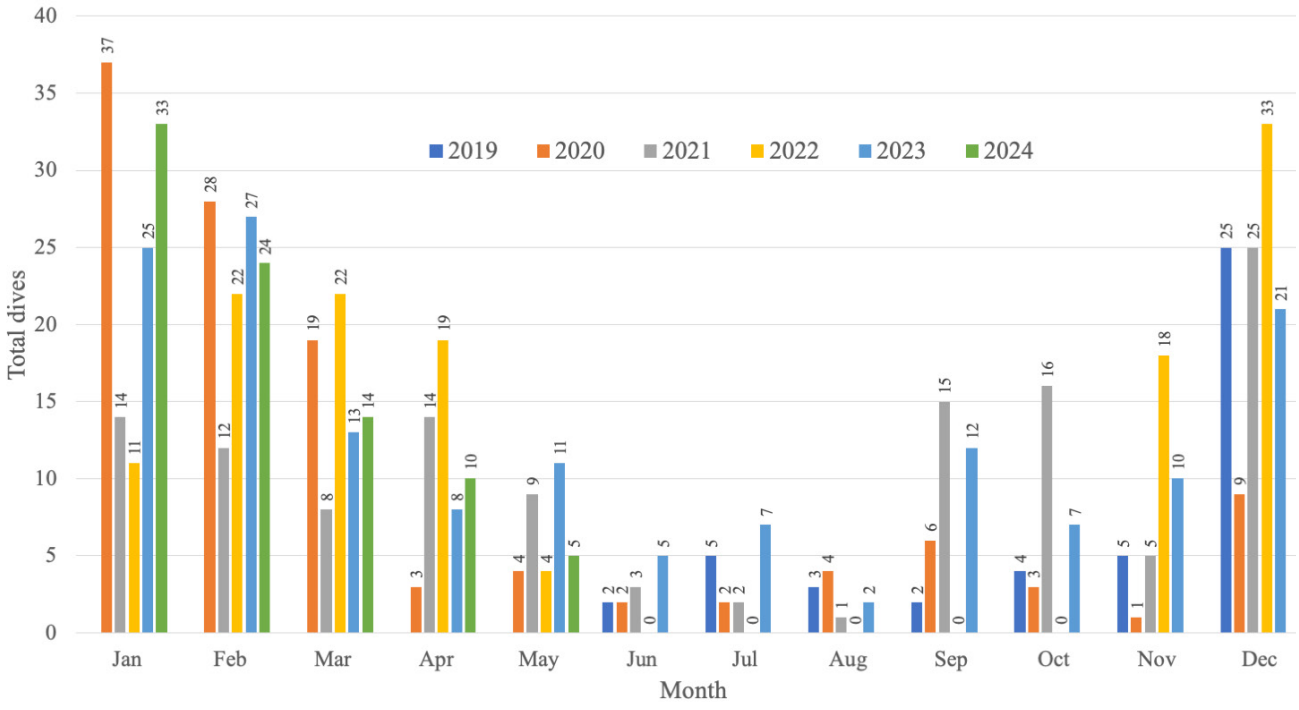
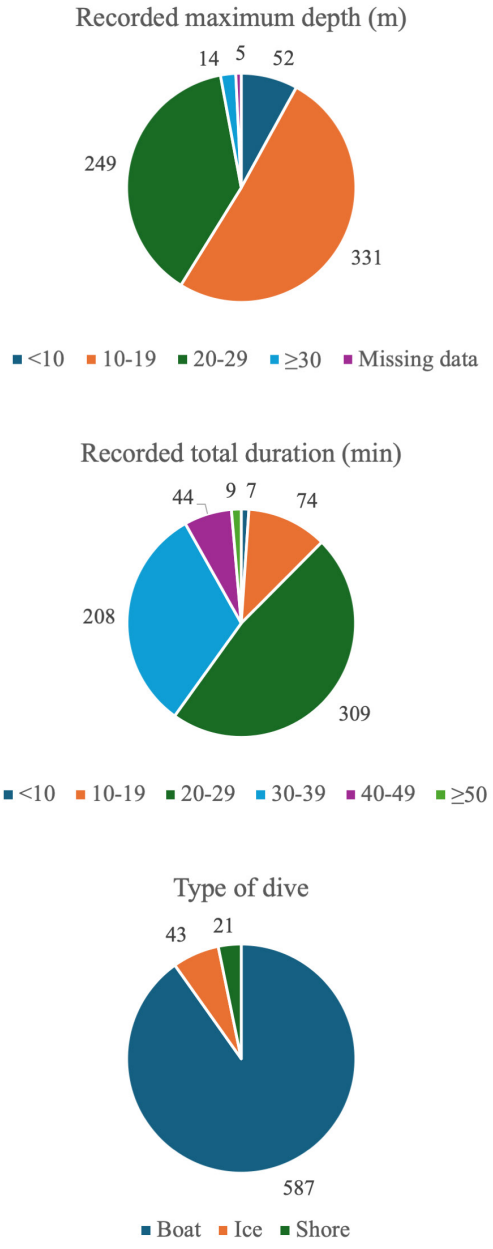


Figure 3
Characteristics of the 651 recorded dives from May 2019–May 2024



The collated dive characteristics are shown in Figure 3. The majority of dives were < 40 min and only 14 had a documented maximum depth of 30 m or more.

While most (> 90%) diving was from rigid inflatable boat, 43 ice dives are recorded. These were distributed as follows:

- 6 July – 11 October 2019: 14 dives.
- 27 July – 8 October 2020: 13 dives.
- 3 September – 17 September 2021: 4 dives.
- 12 September – 22 September 2023: 12 dives.

(From June–October 2022, there were insufficient staff to dive safely, so the programme was paused.)

The value of the scientific diving undertaken at RRS is enhanced by the long-standing nature of the programme. Many projects are part of a multi-year programme of work that allows year-on-year comparison and contributes to BAS's monitoring of long-term trends. Under-ice diving further enhances this value, allowing seasonal variation to be captured.

As an illustrative example, during the 2022–3 season, scientific diving projects at RRS included:

- Sampling of soft sediment assemblages from benthos at 6, 12 and 20 m using an air lift bag and corers; two to five dives each week, all year round.
- Sediment traps deployed at 10, 28 and 33 m; left for one season then retrieved; total of approximately 10 dives over the season. Since the deeper traps are below the usual diving limits, special permissions are required with simple deployment procedures and conservative dive times.
- Annual assessments of anemone growth, 18–24 m, a few dives every quarter.
- Organism sampling. Any depth up to 18 m; up to five dives monthly.
- Fish collection; up to 18 m with multiple dives until all collected.
- Monthly survey of wall life at 24 m.
- Photography and maintenance of IceBergs Impact Study[8] grids at 5, 10 and 25 m. Multiple dives in early summer.

In recent years, divers have also been called upon to survey the new wharf (maximum depth 12 m) and perform survey work on the Sir David Attenborough research ship.

Undertaking the work outlined above, the dive team at RRS usually consists of four people. Each has relevant qualifications for occupational diving and experience from scientific diving or other relevant fields.

All diving is on scuba, with 12 L compressed air tanks and a 3 L bailout. Full face masks reduce the amount of skin exposed to cold water and allow divers to communicate with each other and with the surface. Drysuits are worn. Currently, divers wear wet gloves and, anecdotally, cold hands are felt to be a limiting factor for dive duration. The dive team are part way through the process of procuring dry gloves as a potential alternative. Divers use a bottom timer which shows depth, time, maximum depth and temperature.

Defence and Civil Institute of Environmental Medicine (DCIEM) tables[9] are used to plan dives. A safety stop at 6 m for three minutes is standard practice for all dives deeper

than 9 m. This may be a dedicated safety stop or incorporated into the dive plan if there is work to do in the 5–6 m depth range, after deeper work has been completed. The average sea-level atmospheric pressures at RRS are lower than much of the rest of the world. Atmospheric pressures ≤ 980 hPa require adjustments to bottom times, equivalent to diving at 300 m altitude. As such, corrections are made for dives > 18 m in this circumstance.

In good weather, it may be expected to have one pair diving twice in a day (with an appropriate interval to warm and recover). The second pair may also be expected to dive once, so all four divers on station may have been diving in a single day. This is important when considering who may be able to act as internal tenders in the event of an emergency.

The majority of diving is undertaken from rigid inflatable boat. A dive may only be undertaken if the time taken to reach the dive site is less than 20 min. However, depending on conditions (wind, brash ice, etc.) the return journey may take considerably longer and this is not always predictable.

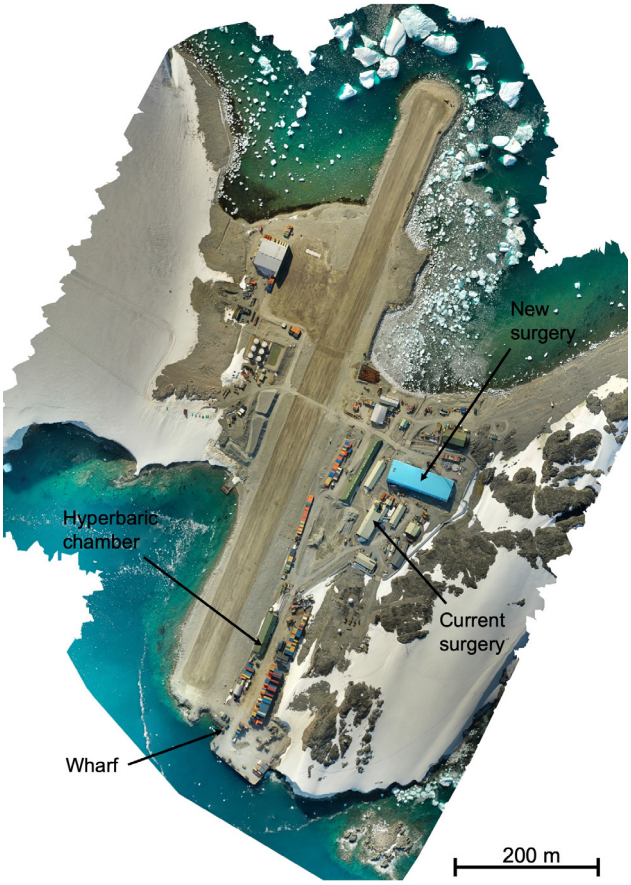
If there is sea ice cover during the winter, then diving may be undertaken if certain conditions are met to ensure the ice is safe to transit across and work on. Ice diving involves the cutting of a primary and a backup hole 30 m apart. Communication lines act as a surface tether for each diver. Each line has a dedicated attendant on the surface and is anchored to the body to be used as a safety line, which can help pull a diver back to the entry point, if required. If verbal communications fail, it can also be used for line signals. Under ice, divers aren't directly tethered as it adds another potential source of entanglement. However, the nature of under ice diving means generally good visibility (> 20 m) so divers can readily remain in visual contact.

Specific hazards and mitigations

The water temperature in which RRS divers operate may be as low as -1.5°C but relatively short dive times, drysuits and surface rewarming are effective at preventing hypothermia.[4] The risk of non-freezing cold injury when diving in these conditions is unknown. A condition of significance in its own right, non-freezing cold injury also has the potential to be mistaken for decompression sickness. It has been proposed that dry gloves may offer better protection than wet gloves during longer dives.[10]

Only no decompression diving is planned using DCIEM tables[9] and a safety stop is incorporated into dives deeper than 9 m. Pooled data has previously been published from Australian, New Zealand, United States and British programmes (1985–2007). From 17,647 person-dives, there were five reported cases of 'mild decompression sickness', five cases of minor barotrauma and no serious diving incidents.[11] This gives an estimated incidence of decompression sickness during Antarctic scientific diving of 2.8/10,000 person-dives. This is higher than other scientific diving.[12] It has been proposed this difference may be due to an increased risk of diving in the cold or to a cautious approach that favours treating equivocal cases early due to the remote setting.[11]

Figure 4
An aerial view of Rothera Research Station



In July 2003, marine biologist Kirsty Brown, was attacked and drowned by a leopard seal while snorkelling at RRS. While leopard seals are known to display curiosity towards divers, aggression is very rare.[13] Following this tragic incident, snorkelling is no longer undertaken. It is felt to engender greater risk than diving given that leopard seals normally hunt prey on the surface. Orca also pose a potential risk to divers. Diving does not commence or is aborted if either of these species is seen in the water. Additionally, divers carry a seal prod to deter advances, if required.

Medical facilities at RRS

Given the remote and hostile setting, provision of medical care is important for all at RRS. The station is served by a single medical facility which can be configured to give two resuscitation bays. Equipment is available to provide essential lifesaving interventions (e.g., chest drains) and basic diagnostics (e.g., X-rays but not ultrasound). Figure 4 is an aerial view of the site. The location of the current surgery is shown with its anticipated new location

in a nearby building, currently under construction. These locations are approximately 300 m from the hyperbaric chamber, which is in the marine laboratory near the wharf.

A medical kit is also available in the marine laboratory and oxygen, as well as basic first aid supplies, are available on the dive boat.

Medical personnel and training

Medical provision on station is the domain of the British Antarctic Survey Medical Unit (BASMU). Their permanent staff are based in Plymouth, United Kingdom (UK), and each year they employ doctors for the upcoming season to cover the BAS stations and the royal research ship Sir David Attenborough. The main doctor at RRS will usually deploy for about 18 months to cover most of a summer season then over winter, returning part-way through the next summer season. Successful candidates will typically have several years post-qualification experience including emergency medicine and some expedition experience. There is a package of pre-deployment training to develop primary and emergency care skills as well as, for example, basic dentistry and radiography. During the peak summer season, there will usually be a British military emergency medicine registrar to support the station doctor.

The doctors typically run a morning and afternoon clinic with pre-arranged and open access appointments. The majority of consultations are primary care or minor injury complaints but the pre-deployment training is crucial to prepare for less common presentations. A significant amount of time is spent training and preparing for less common emergency scenarios (including diving-related emergencies) and when these do occur they require a significant amount of resource to manage the clinical, logistical and myriad other aspects. The medical team must also manage all other aspects of running the surgery (e.g., dispensing medications, stock resupply, equipment checks and basic maintenance) that would likely not be part of their core role in the UK. Lastly, the doctors carry out a number of other duties contributing to station life but not related to medical matters.

Since experience in dive medicine is not a pre-requisite for employment, this is also covered in the pre-deployment training. Preparation for the 2022–3 season included:

1. Twenty-minute session on the background to dive medicine.
2. One hour shadowing dive medicals.
3. Five-day chamber operators' course.
4. One hour discussing deployed process for Health and Safety Executive (HSE) medicals.

This training package is currently under review to ensure it provides the best preparation for the deploying doctor in the time available.

In addition to the core medical staffing, a number of station personnel are trained, prior to and during deployment, in advanced first aid techniques so they can act as medical assistants. Lastly, the dive team includes at least two people trained as International Marine Contractors Association Diver Medical Technicians.

Hyperbaric chamber

A hyperbaric chamber is located in the marine laboratory, which is the base for most dive related activity at RRS. It is a Hytech 60 (Hytech-Pommec, Raamsdonksveer, The Netherlands) with space for three people inside (two stretchered with one sitting). It is serviced by visiting technicians, as required to ensure reliability and regulatory compliance. The chamber must be left ready to use when diving is undertaken. It is used periodically for training, which also serves to check that it is in working order.

A decision to utilise hyperbaric treatment in a given set of circumstances must consider the potential risk to the patient and attendants as well as the intended benefit. If the chamber was unusable (e.g., due to technical failure) or felt to be unsuitable, then other management options should be considered (e.g., surface oxygen and supportive care).[14]

Provision of oxygen

Oxygen in the RRS medical facility may be provided via an oxygen concentrator at 10 $L \cdot min^{-1}$. Apart from this all oxygen is shipped to RRS in cylinders via annual resupply as necessary.

The medical facility plan to start the winter season with a supply of approximately 15,000 L of oxygen mainly in 340 L and F 1,360 L cylinders.

Two cylinders are carried on the dive boat (3 L at 140 bar giving circa 50 min of O_2 at 15 $L \cdot min^{-1}$ via a non-rebreather mask), which is deemed sufficient for the anticipated 20 min journey to shore.

The oxygen stored at the marine laboratory at the start of winter is 12 x 50 L cylinders at 140 bar. This is calculated to be sufficient to complete a series of all of the following tables:

- 1 x fully extended United States Navy Treatment Table 6.
- 1 x United States Navy Treatment Table 6 without extension.
- 6 x Royal Navy Table 66.

This is the maximum treatment likely to be required by one diver. If a second diver (e.g., the buddy required treatment, it is felt that there is still sufficient oxygen for adequate treatment in most likely scenarios). Approximately one 50 L cylinder is used for training and refills annually. If

Figure 5
Diver transported on boat and trailer during training exercise

Figure 6
Piston Bully Antarctic transport vehicle

the oxygen stores were to be significantly depleted (e.g., following treatment of a diver) then urgent resupply may be possible in the summer months but would be unlikely during winter. An impaired ability to provide emergency hyperbaric oxygen therapy, may necessitate curtailing the dive programme prior to resupply.

Oxygen ready for emergency use is stored in the marine laboratory and surgery with the remainder in lockers outside to reduce fire risk.

Transport around RRS

During summer when there is minimal ground snow cover, in the event of a stretchered casualty on the boat, there is the ability to crane the boat from the water onto t he trailer which is then towed by tractor to the marine laboratory (Figure 5).

If required, stretchered casualties may otherwise be transported around station by a variety of vehicles. For much of the year, the roads around the site are covered in snow and ice. In adverse conditions, the most appropriate vehicle is likely to be the Piston Bully (Figure 6) or a skidoo

and Nansen sledge. These are used for a variety of roles around station and, in the event of a medical emergency, a vehicle would likely have to be re-tasked to undertake patient transport.

Evacuation

In the event that medical evacuation was required, this would most likely be by air in the summer months. This would typically involve flying to Punta Arenas, Chile, or to the Falkland Islands. Depending on wind conditions and aircraft type, this could entail a flight time of seven hours or more. Additionally, there may be further substantial delays due to factors such as:

- Aircraft / aircrew location and availability.
- Runway snow clearing.
- Weather.

Evacuation by sea is also an option but this would require a suitable vessel in the vicinity and even a direct passage to the destinations above would take several days.

During the winter, evacuation is likely to be substantially delayed. As an example, in recent years a patient (not a diver) was evacuated from Halley Research Station during the winter season. This involved flying two Twin Otter aircraft from Canada to RRS, which was used as a staging post for the rescue effort. The journey to RRS took almost a week, even with favourable weather.

The expectation would be that a casualty with decompression sickness would complete a course of hyperbaric oxygen therapy prior to flying unless there was another pressing reason for evacuation. Any decision to evacuate must balance the potential risks to the patient and crew with the intended benefit.

Communication

Communication among personnel at RRS is primarily via free net VHF radio. Communication outside the immediate area is via a satellite link that enables email, WhatsApp and telephone traffic to the UK. Until recently, it would not be possible to rely on video calls due to bandwidth limitations. Recent improvements to connectivity mean that video calling could now be considered. A back up option is to call the UK via Iridium satellite phone (Iridium Communications, McLean, Virginia, United States) but this may be time-consuming depending on connectivity.

Remote advice

Remote advice is available as necessary via DDRC Healthcare, Plymouth, UK, (who are contracted to provide advice in the event of a dive emergency and are co-located with BASMU) or by BASMU directly. The communication cascade is published internally to ensure the appropriate response. If it is not possible to contact senior support but the doctor and dive supervisor agree that treatment is in the patient's best interests then this should be commenced and contact made when possible.

Relevant personnel at RRS in the event of a dive emergency

While diving is ongoing, a station doctor must be within the local travel area and their presence physically on station is preferred.

The dive team and boating officer (five people in total) all undertake the chamber operator course in the UK prior to deploying. They have varying levels of previous chamber experience, though this is not a pre-requisite to employment. The team is augmented by non-diving personnel who are trained on station in the roles of chamber operator and internal tender, having had diving medicals prior to deployment. This redundancy is vital to enable the prompt treatment of a diving casualty as the dive team may have other roles following an incident or have been diving themselves. Chamber training and medical scenarios are run periodically to ensure that personnel are familiar with their allocated roles and each day the dive plan is emailed with a nominated person for every role.

Medicals

Occupational diving in the UK is regulated by the Health and Safety Executive (HSE). They specify that divers must have an annual medical performed by an approved medical examiner of divers (AMED). Divers and potential internal tenders have an HSE medical in the UK prior to deploying. If someone is deployed for more than one year, as is frequently the case, their HSE medical will expire. However, the station doctor is not an AMED. The deployed doctor will undertake a history and examination (in-line with their pre-deployment training above) and any tests required (as specified below). They will then discuss their findings with the medical director at DDRC Healthcare. Assuming the medical and fitness standards specified by the HSE[15] are met, the medical director can then issue a temporary re-certification for deployed diving only. Divers should be aware that this does not constitute an HSE medical for diving when back in the UK.

All divers undertake the first three of these tests annually, with the remaining only performed if indicated following history and examination.

Cases

To our knowledge, two divers have been treated for decompression sickness at RRS in the last two decades. The decisions to treat were largely precautionary based on

mild symptoms potentially consistent with decompression sickness. The first case was a diver who developed tingling in one leg while showering 30 min after exiting the water. The second case was a diver who felt nauseous soon after surfacing, which was unusual for them. On examination, they were felt to have unilateral lower limb hyperreflexia. In both cases, the symptoms were fully resolved following a single treatment with hyperbaric oxygen therapy.

Conclusions

Diving is important to facilitate the marine scientific programme at Rothera Research Station. We have described the medical measures in place to mitigate the risk to those diving in this extreme environment. In the event of severe decompression illness, prompt hyperbaric oxygen therapy and remote guidance regarding initial and ongoing management are available with the intent to minimise lasting morbidity. Supporting these measures in such a remote setting requires a significant effort in terms of training, equipment, logistics and UK-based on-call expertise.

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Review article

Development of myopia in scuba diving and hyperbaric oxygen treatment: a case report and systematic review

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Keywords
Myopization; Ophthalmology; Oxygen toxicity; Side effects; Recreational divers; Repetitive diving; Safety

Abstract
(Sokolowski SA, Räisänen-Sokolowski AK, Lundell RV. Development of myopia in scuba diving and hyperbaric oxygen treatment: a case report and systematic review. Diving and Hyperbaric Medicine. 2024 20 December;54(4):328–337. doi: 10.28920/dhm54.4.328-337. PMID: 39675741.)
Introduction: A 54-year-old, previously healthy Caucasian male diver was on a 22-day liveaboard diving holiday. During this time, he performed 75 open-circuit dives, of which 72 were with enriched air nitrox. All dives were within recreational length and depth. After the trip he noticed a worsening of vision and his refraction had changed from the previous -3.75/–5.75 to -5.5/–7.75 dioptres. Hyperoxic myopia is a well-known phenomenon after hyperbaric oxygen treatment (HBOT), but related literature in recreational divers is scarce.
Methods: A systematic literature review on the effect of a hyperoxic environment on the development of myopia was done according to the PRISMA guidelines. Three databases were searched: Ovid MEDLINE, Scopus, and the Cochrane Library. A risk of bias analysis was done on all articles, and the GRADE approach was used to evaluate the quality of evidence. Articles that had sufficient data were used to synthesise a visualisation of oxygen exposure and changes in refraction.
Results: Twenty-two articles were included in this review. These included five case reports, two case series, nine cohort studies, one randomised controlled trial and five reviews, of which one was systematic. Most articles described HBOT patients' ocular complications, although four articles were diver centric. The synthesis of results suggests that divers tend to get a greater myopic shift with a smaller exposure. However, the data were too heterogeneous to perform meaningful statistical analyses. This review is the first to focus on divers instead of HBOT patients.
Conclusions: The case presented led to a systematic literature review on the effects of hyperbaric oxygen on refractive changes in both HBOT patients and divers. The data were too heterogeneous to make meaningful suggestions on a safety limit to prevent myopisation in diving.

Introduction

In recent decades, the use of enriched air nitrox (EAN) has become increasingly popular in the recreational diving community. Previously, such gas mixtures were only used by technical divers. However, due to its benefits in prolonging the bottom time and decreasing the nitrogen load during diving holidays, its popularity has grown, and it is, therefore, available nowadays at almost any dive centre. Unfortunately, human physiology is not adapted to a constant hyperoxic environment, and whereas EAN can make diving safer from a decompression stress perspective, it also predisposes to some less-discussed adverse effects of oxygen toxicity, such as possible myopia or the maturation of cataracts. This phenomenon is well known in hyperbaric medicine and, to some extent, in technical diving and occupational diving. Regardless, authors of this article are not aware of literature on myopia in purely recreational diving. Due to the increased use of EAN in recreational diving, it should be discussed in greater detail.

Myopia and cataracts are common eye pathologies that are well understood. The physiology of the eye changes when it is exposed to a hyperbaric environment and even more so when the partial pressure of oxygen increases. The effect this has on the lens has been previously studied in animal models.[1,2] The hyperoxic environment causes oxidative stress in the eye metabolism by oxidising glutathione, which leads to changes in the opacity of the lens, and thus contributes to the formation of cataracts.[3] Additionally,

oxidative stress creates free oxygen radicals that damage the crystalline structures of the lens,[4] as well as other water-soluble proteins.[2] These are suggested to cause the refractive change in the lens, which then manifests as a shift towards myopia.[5] The myopic shift is suggested to be a precursor of the development of cataracts.[1]

The oxygen exposure limit leading to the ocular changes is not known. Divers are well acquainted with oxygen toxicity in terms of pulmonary toxicity and central nervous system toxicity. These are evidently more severe manifestations of the toxic effects of oxygen, as they may lead to convulsions, loss of consciousness, and, in an underwater setting, death.[6] The National Oceanographic and Atmospheric Administration (NOAA) has developed safety limits for divers to follow. Oxygen toxicity unit (OTU) and central nervous system percent (CNS%) scales were developed to estimate (respectively) the pulmonary and cerebral effects of hyperoxia.[6] These are taught to divers who want to use EAN during their dives.

In this paper, we report a case where the diver's myopia deteriorated during a diving vacation using primarily EAN as a breathing gas. The popularity of EAN in recreational diving has raised the question of whether it is needed on frequent but shallow dives. As this was not well described in the literature, a systematic review was performed to assess the effects of a hyperoxic environment on the development of myopia in divers and HBOT patients. The secondary objective was to compare the development of myopia in divers and HBOT patients to see how the case presented aligns with the literature.

Case description

Written informed consent for publication of his case was received.

A 54-year-old, previously healthy Caucasian male diver was on a 22-day liveaboard diving holiday. He performed 75 open-circuit dives on consecutive days. Of these, 72 were with enriched air nitrox 32% (EAN32) breathing gas and the remaining three with air. The daily number of dives was as follows: four dives/day for thirteen days, three dives/day for six days, two dives/day for two days and one dive/day for one day. The detailed dive log including the oxygen toxicity parameters was available only for the last 35 dives due to memory limitations of the old model dive computer (Suunto Vyper). The summary of the dives is shown in Table 1. Development of the daily central nervous system toxicity (CNS%) is shown in Figure 1, and oxygen toxicity units (OTU) in Figure 2. These were calculated by the diving computer for each dive using the NOAA rules,[6] which are commonly taught to divers. The cumulative CNS% calculations were slightly modified, as the a generic formula for half-life (Equation 1), was used instead of a less accurate constant half-life of 90 minutes.

$$N(t) = N_0 \left(\frac{1}{2}\right)^{\frac{t}{t_{1/2}}} \quad \text{Eq 1}$$

After the trip, he noticed that he had developed impaired vision and therefore visited an ophthalmologist. His refraction had changed from the previous -3.75/–5.75 dioptres evaluated two years earlier by an ophthalmologist, to -5.5/–7.75 dioptres. Before the trip he had not reported any new refractive problems. In addition, an ophthalmologist diagnosed early cataracts that were not seen previously. Other diseases, like diabetes and hypertension, were excluded. He had no history of ocular trauma, use of topical steroids or other ocular medications, nor exceptional exposure to sun. It was noted that he had suffered a retinal detachment twice in both eyes six- and seven-years prior that was adequately treated with no residual complications. During follow-up time of one month the refraction improved to -4.75/–7.00 but thereafter remained stable for six months, after which further improvement was not observed. During the following year the vision worsened to -5.25/–7.25 dioptres, even though the patient did not dive during that year. Subsequently he took a short diving holiday and dived 11 times over five days using air, not immediately noticing any difference in his vision. However, two months later an ophthalmologist measured his refraction at -6.00/-7.50 dioptres. There was no change in his cataracts.

Method

The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines were followed for the literature search and writing process. The PRISMA checklist is provided as a Supplementary file 1*. The protocol was registered on PROSPERO (CRD42023450396) before the analysis.

Table 1
Specifications of all the dives (n = 75) during the 22-day-long diving trip; msw – metres of seawater

Parameter	Dive time minutes	Maximum depth msw	Average depth msw
Mean	68	26	16
Median	68	27	17
Range	49–93	12–36	7–22

* Supplementary files 1–5 can be found on the DHM Journal website: https://www.dhmjournal.com/index.php/journals?id=345

Figure 1
Daily central nervous system percent oxygen exposure (CNS%) based on NOAA limits over the last 11 days of diving; the CNS% accumulated during the dive was recorded from the diving computer. The daily limit for CNS% is 80%, represented by the red line

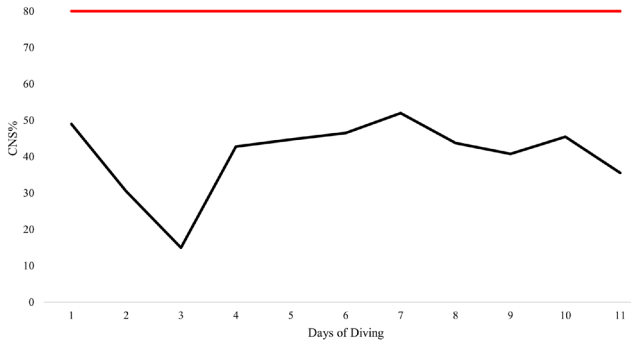
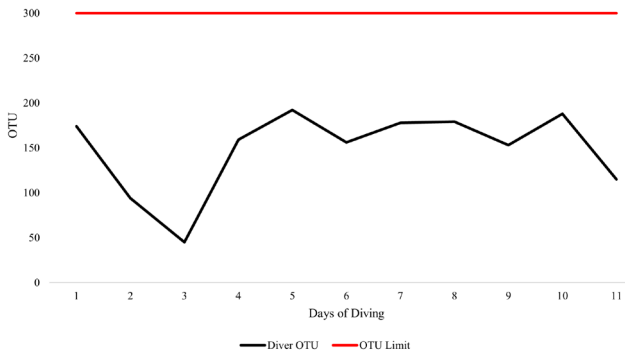


Figure 2
Diver's daily oxygen toxicity units (OTU) for the last 11 days of diving; the safety limit over nine days of diving is 300 OTU, which was respected throughout the diving vacation



SEARCH STRATEGY

The literature search was conducted on 3 October 2023, and the databases searched were Ovid MEDLINE, Scopus, and the Cochrane Library. The following search string was used:

((Myopia OR Myopic* OR Cataract* OR "*Vision chang**" OR "*Visual acuity*" OR "*Visual chang**") AND ("*Hyperbaric oxygenation*" OR "*Hyperbaric oxygen*" OR Diving OR Hyperoxia OR Hyperox* OR "*Oxygen* toxic**" OR "*Oxygen* poison**"))

The full search strategy can be found as a Supplementary file 2*.A medical librarian was consulted in the development of the search strategy and helped perform the final search. Two researchers (SS, ARS) went through the elimination process independently and any disagreements were discussed until a common understanding was reached.

ELIGIBILITY CRITERIA

Studies investigating the changes in refraction after exposure to hyperbaric and hyperoxic environments were sought. The scope of this review was on humans, so any non-human studies were excluded. However, there were no restrictions as to the age or sex of the patient populations. The only restriction in the health status of patients was an existing eye pathology before HBOT (e.g., vision loss due to arterial occlusion), as this was considered to be a confounding factor with the aim of this study. The hyperoxic environment was defined as HBOT or diving. Caisson workers were excluded. This study only involved peer reviewed and published work. Clinical articles, case studies, and reviews were all included, but expert opinions and commentaries were not. Additionally, only English works were included, and papers published before the year 1970 were excluded.

RISK OF BIAS AND CERTAINTY OF EVIDENCE

Joanna Briggs Institute (JBI) Critical Appraisal Tools[7] were used for each type of article separately (case study, case series, cohort study, systematic review, narrative review, randomised controlled trial). This tool consisted of a checklist including 8–13 questions that had "*yes*", "*no*", "*uncertain*", and "*not applicable*" options. It was used to assess the methodological quality of each study. This was done by analysing the possibility of bias in the study design, how the study was conducted and what analysis was used in each research article. The assessment was done at a study level, and any study failing to get a minimum of 50% of the total "*yes*" answers was excluded due to evident bias present in the article. The quality assessment was performed by two researchers independently (ARS, SS). The disagreements were discussed between the two researchers until a common understanding was found.

The certainty of evidence of each article was assessed using the grading of recommendations, assessment, development, and evaluation (GRADE) approach.[8] This approach takes into consideration whether a study is a controlled trial or an observational study and then upgrades or downgrades the quality of evidence based on study quality, imprecision, indirectness, inconsistencies, and effect size. This was done by three researchers, two of whom were working together (ARS, SS) and one independently (RVL). The results were discussed until all three agreed on the grading.

* Supplementary files 1–5 can be found on our website: https://www.dhmjournal.com/index.php/journals?id=345

SYNTHESIS OF RESULTS

Studies used for data synthesis must have clearly stated the oxygen exposure and the change of the refraction in dioptres. All studies failing to do so were excluded from the synthesis. The data including oxygen exposure, change in refraction, oxygen toxicity units, the pressure at which oxygen was breathed, the number of participants, and whether they were divers or HBOT patients were extracted. If the oxygen percentage used in HBOT was not specified, it was assumed to be 100%, and if 'air breaks' were not mentioned, it was assumed there were none. If the change in refraction was given separately for the left and right eye, the average change was calculated. The 'standard' HBOT treatment plan was assumed to be 90 min at 240 kPa of 100% oxygen. To make the oxygen exposures comparable between different studies, HBOT patients and divers, the oxygen exposure was calculated in hours of 100% oxygen exposure at one atmospheric pressure (101 kPa). These calculations apply exclusively to periods of diving or HBOT. The formula used is shown below:

> Exposure = number of treatments or dives x inspired fraction of oxygen x time (hours) x average pressure (atmospheres absolute)

These exposures were plotted against the refraction change in dioptres. All studies measuring only visual acuity or other measurements, such as intraocular pressure, eye axial length, or keratometry, etc. were excluded from the synthesis. Patients treated with HBOT were reported separately from divers. Additionally, the number of participants in each study was taken into consideration. The data extraction was done by one author (SS) under the supervision of the two senior authors (RVL, ARS). However, due to the observational nature of the study topic, no statistical tests were done, as they would not be meaningful and would bring very little additional value to the synthesis.

Results

Figure 3 shows the selection process as a flow chart. Initially, 478 records were identified; after the removal of duplicates 454 records were left for the screening process. After the initial screening of titles and abstracts, two researchers working independently (ARS, SS) agreed on 51 full text articles to be sought for retrieval and assessed against the eligibility criteria. Twelve articles could not be retrieved due to unavailability, leaving 39 articles to be assessed. After assessment another 16 full-text articles were excluded. Of these, 14 articles were excluded due to insufficient information on myopia developed in study subjects, i.e., a brief mention of ocular side effects without any further information as to the severity or reversibility was not considered sufficient. One article was excluded due to existing eye pathology (phakic and pseudophakic eyes) before HBOT. Finally, 23 articles met the inclusion criteria of the search. However, one article was later excluded due to acquiring less than 50% of the "*yes*" answers in the risk of bias assessment.[9] Thus, the final number or included articles was 22.

A total of five case reports[10–14] and two case series[15,16] met the inclusion criteria. The most frequent study design was a cohort study with nine articles meeting the criteria.[17–25] Finally, one randomised controlled trial met the criteria.[26] Additionally, five reviews met the criteria, four of which were narrative in nature[27–30] and one systematic.[31] The summary of the articles included is presented in Supplementary file 3* from which reviews are excluded.

RISK OF BIAS

Figure 4 represents the summary of risk of bias analysis using JBI Critical Appraisal Tools. Starting from the top, first the case reports are presented, followed by case series, cohort studies, randomised controlled trial, systematic review, and finally, the narrative reviews. Each study design had its own checklist. The results are presented as percentages. There were five articles that scored a full 100%, of which three were narrative reviews and two case reports. Additionally, six cohort studies only got "*yes*" and "*not applicable*" answers, similarly demonstrating a low risk of bias. The average percentage of "*yes*" answers was 80%.

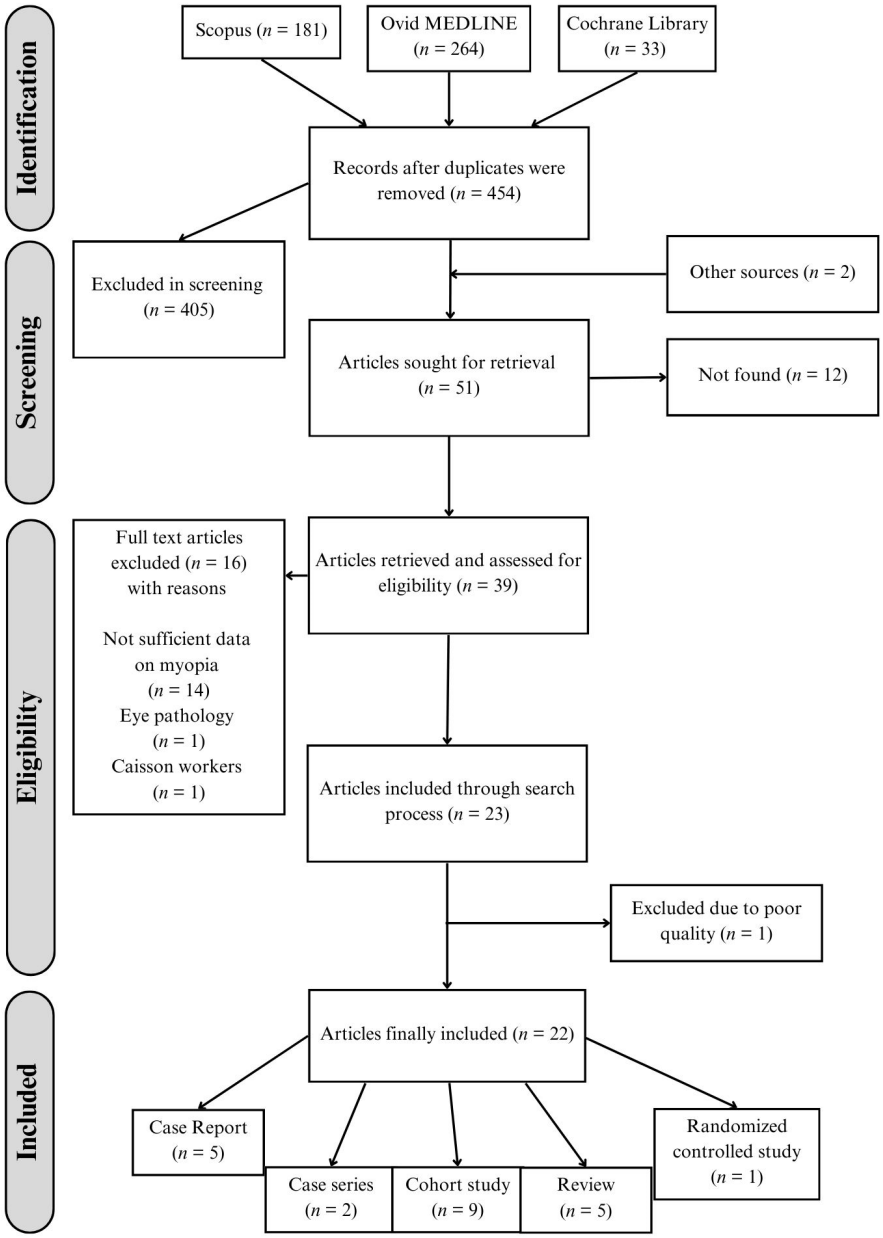
OCULAR CHANGES IN DIVERS

Only four articles described ocular changes in scuba divers including two case reports,[13,14] one case series[16] and one cohort study.[25] Marín-Martínez et al. presented two cases of occupational divers, both of whom complained of worsening vision after diving. One of them related this to a recent change to a closed-circuit rebreather (CCR). While there is a mention of use of HBOT after each dive, the reason for this treatment is unclear. It is also unclear what sort of dives they performed and how often, thus making it impossible to evaluate the oxygen exposure.[13]

Another case of hyperoxic myopia was reported by Butler et al. (1999). A 48-year-old male was participating in a film project requiring daily dives for 21 days. He was using a CCR with constant oxygen partial pressure of 130 kPa in an EAN mixture. He was exposed to a cumulative effect of hyperbaric oxygen during a total of 84.8 hours of diving at 130 kPa oxygen and started noticing a worsening of vision after 18 days. Once he had returned from his expedition, he was examined and found to have a myopic shift of -1.50 dioptres (D) in both eyes. After almost two months, his vision was restored and even turned slightly hypermetropic.[14]

* Supplementary files 1–5 can be found on our website: https://www.dhmjournal.com/index.php/journals?id=345

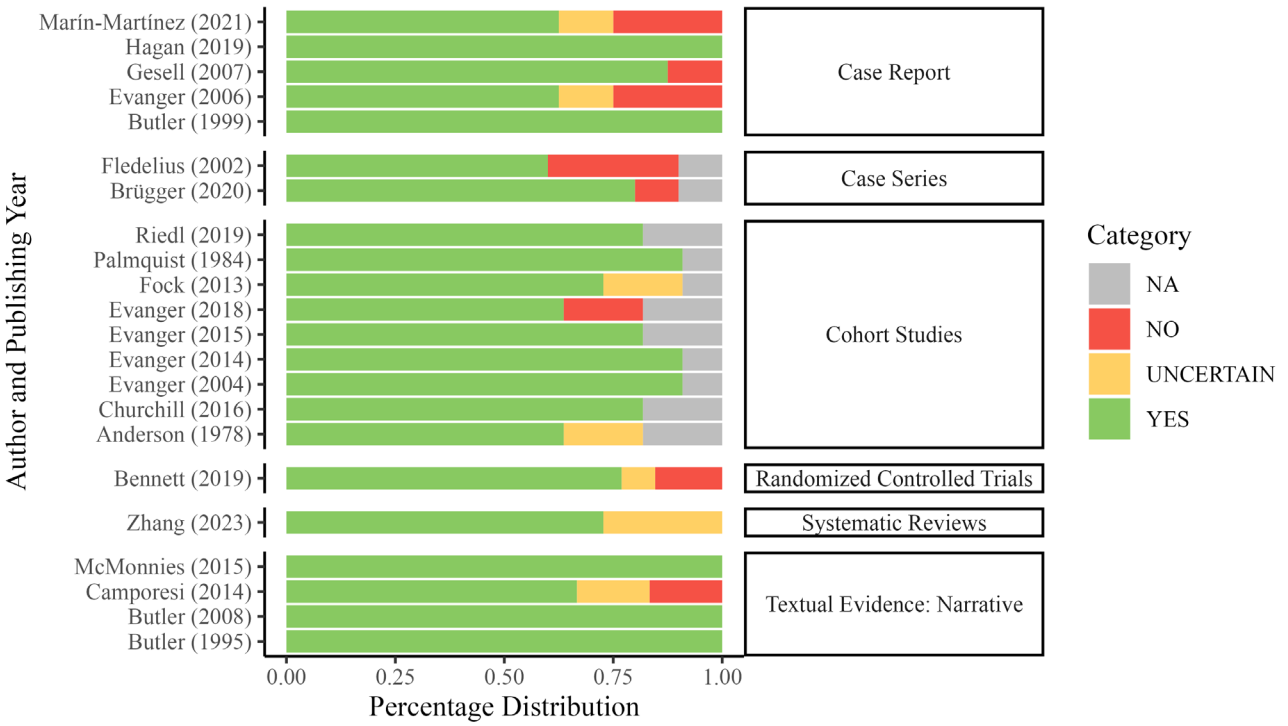
Figure 3
PRISMA flow diagram demonstrating the selection process of the articles for the systematic literature review



A case series of four military divers was presented by Brügger et al. (2020), wherein the subjects were exposed to 135 kPa of 100% oxygen via a MK20 Aga full-face mask with an open circuit regulator in a test pool. They were asked to perform light exercise on a bicycle for 30 minutes continuously every hour. Each dive was six hours long, and the participants dived for five consecutive days with an 18-hour surface interval in between dives; an equivalent exposure to 40.5 hours breathing 100% oxygen at 101 kPa. All subjects had an objective worsening of vision, measured with a Snellen chart, but recovered spontaneously seven to 30 days after onset.[16]

Finally, Fock et al. (2013) presented a cohort study with 14 male CCR divers and one OC diver who performed multiple day diving expeditions with an average of two dives per day, with a surface interval of approximately four hours between dives. The CCR divers maintained an oxygen partial pressure of 130–140 kPa for most of the dives. The mean duration of the dives was 112 minutes, and the average depth was 69 metres of seawater (msw). The mean change in visual acuity, reported in dioptres, was 0.4 on the 13th day of the expedition. Only one diver sought formal evaluation by an ophthalmologist, and his vision returned to baseline eight weeks after the expedition.[25]

Figure 4
The summary of risk of bias analysis



OCULAR CHANGES IN HBOT PATIENTS

The majority of the articles that met the inclusion criteria for this review were on HBOT patients. A total of three case reports,[10–12] one case series,[15] eight cohort studies,[17–24] and one randomised controlled trial[26] were included. The summary of the results is presented in Supplementary file 3*.

All three cases developed a significant (> -0.5 D) myopic shift after HBOT treatment. Two of the cases were female and one male. The age range was 49–58 years and the treatments varied from 21 to 48 treatments in total. All were treated with 90 min sessions at 200–240 kPa. All three subjects had their refraction measured, with the minimum myopic shift being -1.25 D, and the maximum -1.75 D. One patient's eyesight kept worsening, and at 11 months post-HBOT, the refraction in both eyes were measured -4.25 D.[10] Another patient developed hypermetropic shift four weeks after the completion of HBOT series. It continued worsening until 11 weeks after treatment, when the refraction was +1.62 D in the right eye and +1.50 D in the left eye. It remained stable at last follow at 1.5 years.[12]

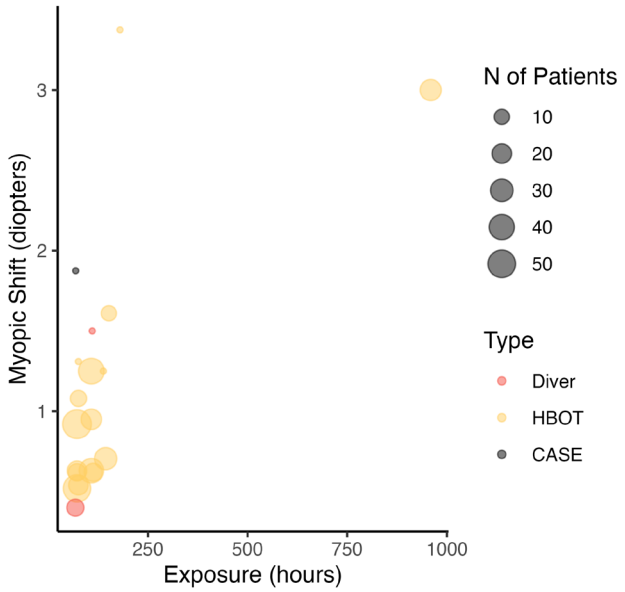
In the Fledelius et al. (2002) case series, 17 patients were treated with HBOT, mostly for post-radiation osteonecrosis of the mandible. Patients with cataracts were excluded from this study, and all patients received 30 treatments of 95% O_2 at 250 kPa in 95 min sessions. The oxygen was delivered via a mask system. The patients' visual acuity, refraction, and keratometry were measured, and the median change of refraction was -0.62 D, however, there was no change in visual acuity.[15]

The cohort studies form a heterogenous group of articles with varying results. The most common indications for HBOT were osteoradionecrosis, persisting leg ulcers, osteomyelitis, proctitis, or cystitis, but some studies did not specify the indication of HBOT.[22,24] One study included only patients having HBOT for the first time, or less than 40 treatments and no cataract surgery.[20] The mean age of patients varied between 55.1 and 61.7 years. The total number of treatments varied from 10 to 425. Most commonly, the treatment time was 90 min, however, longer treatments were also used.[17,18] Some had breaks for breathing air during the treatment, whereas others did not. The oxygen percentage breathed was not always mentioned but seemed to vary between 95 and 100%. Treatments were mostly given from Monday to Friday, or consecutively with no break days in-between. The Snellen Chart was commonly used to measure visual acuity, but most studies also examined the refractive error. The precision and equipment used to examine the eyes varied greatly, as a few articles also included ophthalmological measurements, such as keratometry, intraocular pressure, axial length of the eye, retinal thickness, and corneal thickness. All studies reported some myopic shift. In some articles, only some patients were affected (e.g., 60%),[24] but in others, all patients were reported to have visual changes.

VISUAL CHANGES IN RELATION TO OXYGEN EXPOSURE

Figure 5 was extrapolated from the articles reviewed, in order to compare oxygen exposure and the development of myopia in those studies. Out of the 22 articles that met the inclusion criteria, 14 works presented sufficient information

Figure 5
Synthesis of data from 14 articles showing the oxygen exposure, as hours of 100% oxygen at 101 kPa and vision change; HBOT patients, divers, and the case are demonstrated in different colours. The size of the population is represented in the size of the data point



on the number of patients, exposure, and myopic shift (in dioptres) to be included in a graph of exposure (as 100% oxygen-hours at 101 kPa) against myopic shift (in dioptres). Figure 5 is composed of articles listed in Supplementary file 3* all of which meet the inclusion criteria stated in the methods section. Articles that compared different administration methods or number of treatments[23,26] were given multiple data points, where one data point represents one group of patients (e.g., oxygen administered via hood). Supplementary file 4*shows the calculated exposures for each of the articles included. The weighted average of exposure was 155 hours in HBOT patients and 71 hours in divers. The weighted average of myopic shift was 1.0 dioptre in HBOT patients and 0.6 dioptres in divers. In contrast, the case report described in this article has a high myopic shift (1.88 D) with a relatively low exposure (68.6 hours). The cumulative exposure of the diver is calculated using the values in Supplementary file 5*. No statistical analyses were performed as these data are from a heterogenous group of original articles consisting of small sample sizes.

REVIEWS

Four narrative reviews met the inclusion criteria.[27–30] These reviews included most of the articles referenced in our systematic review. Two of these were by Butler, the first one dating back to 1995. That extensive review's focus was on optics in diving, but the ophthalmological complications related to decompression sickness (DCS) or HBOT were discussed as well.[27] In his second review, Butler focused on the ophthalmological indications for, and ocular complications of HBOT, including myopia. This review from 2008 included a greater number of articles, some involving divers.[28] McMonnies' review (2015) mentioned myopia only in a few sentences, with the focus on cataracts, keratoconus, and age-related macular degeneration.[29] Camporesi's review (2014) presented the general side effects of HBOT, but ocular complications were discussed in detail,[30] with most of our review's HBOT-related articles included.

Only one systematic review detailed the ocular complications and other side effects of HBOT. It followed the PRISMA guidelines and found that patients who underwent HBOT were significantly more likely to have ocular side effects compared to either sham therapy or other conventional treatments.[31] Nevertheless, the ocular side effects were not specified, thus potentially including ophthalmological conditions other than myopia. Furthermore, the review included only randomised controlled trials, thus, most of the articles included in this study were excluded.

Discussion

To the best of our knowledge, this is the first systematic review with the main focus on myopia in both divers and HBOT patients, in contrast to narrative reviews which have been written previously on the ocular complications of a hyperoxic and hyperbaric environment, focusing on HBOT.[27–30] One of these reviews took divers into consideration.[28] The systematic review included focused on the side effects of HBOT. However, ocular complications were briefly discussed.[31] The principal finding of this review is that even though hyperoxic myopia is a well-known phenomenon, especially in HBOT, the current evidence is not strong enough to suggest a safety limit of oxygen exposure to prevent complications. Nonetheless, guideline changes could be appropriate in the future with prospective and mindful study research.

It seems that some are more sensitive to a hyperoxic and hyperbaric environment. This is evident from multiple case reports on the subject.[10–14] Such subjects appeared to develop quite significant myopia compared to the cohorts. This could partly be explained by the nature of a case report, which generally describes an unusual presentation.[32]

Diving could potentially cause a greater myopic shift than HBOT, as divers appeared to develop myopic shifts at lesser exposures to oxygen. It has been previously suggested that the effect of oxygen is greater when submerged than in a 'dry dive'.[33] However, the data presented in this review are not reliable enough to support such a conclusion. Additionally, the intensity of exposure was different between HBOT patients and divers. The maximum partial pressure of oxygen the divers were exposed to was 140 kPa during the dive, or

* Supplementary files 1–5 can be found on our website: https://www.dhmjournal.com/index.php/journals?id=345

160 kPa during a decompression stop.[34] In contrast, HBOT patients were often exposed to a partial pressure of oxygen of 240 kPa. While patients undergoing HBOT were mostly subject to only one treatment daily, divers were observed to perform multiple dives within a day. Therefore, divers seem to be exposed to lower partial pressures of oxygen compared to patients undergoing HBOT, albeit at a higher frequency. Evanger et al. (2018) reported an improvement in the myopic shift after weekend breaks, with HBOT administered only during the weekdays from Monday to Friday.[22] This potentially indicates that divers tend to develop more myopia with a smaller exposure due to the frequency of the exposures, leaving less time for recovery between dives.

As there is no evidence of myopic shift in recreational divers, except for our case presented in this study, it is also unclear if the type of diving influences the development of myopic shift. The literature presented in this review portrays technical divers[13,14,25] and military divers,[16] who performed longer or deeper dives compared to recreational divers. It is more common for recreational divers to have a higher frequency of diving, e.g., on a diving vacation, but the dives are often shallower. Nevertheless, our case showed a relatively large myopic shift, and whereas this could be only a peculiarity, recreational divers should be investigated in greater detail in the future to investigate if the phenomena described in our case report is common or not.

Finally, based on our findings, it could be beneficial to discuss if the current oxygen toxicity limits presented by NOAA, taught early in divers' careers, are still relevant. These limits were developed to prevent the toxic effects of oxygen in the central nervous system (CNS%) and lungs (OTU),[6] both of which are more adverse than myopia. Regardless, the loss of visual acuity can also be debilitating. Technical divers pass the daily OTU and CNS% limits on their longer dives without any adverse effects, whereas the subject in our case report, whilst well below the recommended limits, still developed severe myopia that has not yet completely reversed. When compared to divers from DAN Europe's database, which includes 2,629 open circuit dives over a 5-year period, our case report patient had a shallower mean maximum depth (25.9 msw vs 27.1 msw), but a longer mean dive time (68 min vs 46.4 min).[35] DAN Europe's database includes technical divers, which can somewhat skew the results. Lastly, it is possible that unknown concomitant factors contributed to the worsening of vision of the diver in our case report.

STRENGTHS AND LIMITATIONS

The search process for this review was extensive. By broadening the scope to both HBOT patients and divers, more articles could be included. Additionally, no limitations were asserted on the type of study, thus a variety of literature was identified from case studies to randomised controlled trials. As reviews were included as well, a comparison of the articles included in this review and previous reviews showed that the search strategy was successful. The search found all the relevant articles from previous reviews, along with newer publications. Additionally, for a small field like diving medicine, a total of 22 articles seems an adequate review of the existing literature.

Nonetheless, partially due to the observational nature of diving and hyperbaric medicine and partially due to a long-time span of the research included (over 50 years), the overall quality of data cannot be considered scientifically very high. Furthermore, there were variations in the methodology and follow up times, which made comparison between studies difficult. To make a visualisation of oxygen exposure and refraction changes, some articles had to be excluded and others simplified. This was due to the great variation amongst the articles, including in their study designs. Because of this, no statistical analyses were done, as this would not give meaningful results due to the variations in the methods.

A weighted average was used, as it takes into consideration the number of subjects involved in the study. Therefore, case studies that tend to have severe myopic shifts with small sample sizes would not skew the results inordinately. Alternatively, studies such as Plamquist et al. (1984)[18] that involved very high exposure of a relatively large cohort altered the weighted average. Since no statistical analyses were performed, this measurement was given to clarify the difference between divers and HBOT patients. It is not possible to infer a relationship between oxygen exposure and the myopic shift. Hopefully, this encourages further research to determine reliably if there is a difference in the tendency to develop myopia.

The risk of bias analysis was done using different checklists for each study design in contrast to most systematic reviews, where one tool is used for all articles. This method was chosen because most of the general risk of bias tools give the greatest value to randomised controlled trials, and any other study designs are given lower scores. Randomised controlled trials are quite rare in diving medicine, thus, a tool taking into consideration the study design seemed optimal. Consequently, the risk of bias analysis shown in Figure 4 solely represents the degree of bias of the study in its own category. As a result, a comparison between categories is misleading, as different assessment criteria were used for each design.

FUTURE ASPECTS

In summary, this review demonstrates that more research is needed on the effects of hyperbaric oxygen, especially in diving, on the development of myopia. A carefully planned prospective study would be best suited to get useful data. Two issues should be investigated before any safety limits are suggested to divers. Firstly, whether shorter duration, but more frequent dives at lower partial pressures of oxygen are more problematic in terms of myopisation than longer

duration but less frequent dives at higher partial pressures. Secondly, more ophthalmological data should be recorded on recreational dives, as diving holidays are quite popular. Addressing these issues through well-designed research may contribute to the development of enhanced safety guidelines and limits on the use of EAN for dives less than 20 msw. This is especially with regard to the development of ocular complications in particular hyperoxic myopia.

Conclusions

A case of a recreational diver, who developed significant myopia after a diving holiday, led us to perform a systematic literature review on hyperoxic myopia. This is the first systematic review that takes into consideration both divers and HBOT patients, and focuses on the myopic shift after a hyperoxic environment. With the increased use of EAN as a breathing gas in recreational diving, a greater proportion of the diving population is exposed to a hyperoxic environment, with even higher exposure to oxygen than when diving with compressed air. Specifically, the use of EAN in more frequent, but shorter and shallower dives, is not well studied. Existing literature does not provide enough information for making any new safety limit suggestions to prevent myopisation. Consequently, more targeted research is needed to gain an improved appreciation of who is at risk, and at what level of exposure.

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Guidelines

South Pacific Underwater Medicine Society (SPUMS) position statement regarding paediatric and adolescent diving

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Abstract

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This paediatric diving position statement was developed from a targeted workshop at the 51st Annual Scientific Meeting of the South Pacific Underwater Medicine Society (SPUMS) on 8 June 2023. It highlights the factors that SPUMS regards as important when undertaking health risk assessments for diving by children and adolescents (defined as aged 10 to 15 years). Health risk assessments for diving should be performed by doctors who are trained in diving medicine and who are familiar with the specific risks which result from breathing compressed gas in the aquatic environment. Undertaking a diver health risk assessment of children and adolescents requires a detailed history (including medical, mental health, psychological maturity), a comprehensive diver medical physical examination and evaluation of all relevant investigations to exclude unacceptable risks. In addition, assessment of the individual's motivation to dive and reported in-water capability should occur, whilst engaging with their parent /guardian and instructor, where appropriate, to ensure that safety for the child is optimised. The guideline applies to all compressed air diving including scuba and surface supply diving provided in open and contained bodies of water.

Introduction

This paediatric and adolescent diving position statement was formulated through expert consensus from a targeted workshop at the 51st Annual Scientific Meeting (ASM) of the South Pacific Underwater Medicine Society (SPUMS) on 8 June 2023. Ten statements were developed and accepted in principle by workshop participants. Final editing and referencing were by the SPUMS Paediatric Diving Working Group. During this process, the age range was reclassified as paediatric and adolescent, and an additional statement was added (11 statements in total) that enhanced certain salient points to consider when assessing and consenting these prospective divers.

The society published its first guidelines for paediatric divers in 1990, setting a minimum age of 16 years before medical health risk assessments would be undertaken on prospective open water divers. This was revised in 1992 to a minimum age of 14 years, taking into consideration the level of psychological maturity, physical capability, and confidence for the candidate in managing the underwater environment.[1] It was also consistent with the now retired Australian Standards: Training and certification of recreational divers. Part 1: Minimum entry-level SCUBA diving. 4005.1(2000).[2]

The June 2003 edition of SPUMS Journal (Volume 33, Issue 2) was dedicated to children in diving. Many experts in the field weighed in and continued the discussion in that and subsequent journal issues that year.[1–7] The two decades since the 2003 publication have seen substantial increases in diving course options for children by training organisations. Children as young as 10 years have been completing diving courses allowing open water diving with well-established

training organisations around the world, albeit with defined restrictions and limits. Some of these commenced prior to 2000, with even younger participants.[7] It is noted also, that children aged 8–10 years can access introductory experiences using scuba equipment, directly supervised by instructors and confined to a pool environment.[8]

Evidence-based medical practice should be focused on the health and wellbeing of the prospective candidate and not the commercial interests of industry. It is widely acknowledged that a child's chronological age doesn't necessarily correspond to their physical, psychological, emotional, or intellectual level of development.[3,9]

The latest version of the SPUMS Recreational Dive Medical was published in 2020, with updated cardiovascular health guidelines.[10,11] At that time, SPUMS did not update the guidelines on medical health risk assessment of prospective child and adolescent divers.

Other medical societies, such as the Dutch Society for Diving and Hyperbaric Medicine and German Society for Diving and Hyperbaric Medicine recently revised their recommendations for this unique subset of the population.[12–14] There is a paucity of quality evidence regarding current best practice for assessment of children and adolescents seeking to undertake compressed gas diving, and the advice provided to them and their parents or guardians. Most information consists of case reports, retrospective event analysis, prospective cohort studies, and expert opinion. The South Pacific Underwater Medicine Society acknowledged that its existing guidelines required updating to support health risk assessment of children and adolescent prospective divers.

Definitions

In developing this current position statement, SPUMS has adopted the following definitions:

Paediatric (adjective) – refers to describing children from birth to 17 years of age.

Child or Children (nouns) – refers to individuals or groups including infancy through to puberty (puberty is achieved when the child reaches reproductive maturity: this includes a range of ages and is different according to the sex of the individual).

Adolescent (noun or adjective) – defined by the World Health Organisation as 10–19 years of age.[15]

It is also recognised that there may be individuals from the above descriptor groups for whom this SPUMS position statement is not relevant. The terms will be used in the text to refer to a narrower range of age groups defined in Statement 1.

Whilst acknowledging limited data in this field, it appears that diving amongst children and adolescents is relatively safe. This is likely due to experienced medical assessment, consent alongside their legal guardian, and engagement with a supportive and skilled instructor through a training organisation with appropriate, established procedures. Vandenhoven's retrospective study demonstrated that children had a high rate of medical issues incompatible with diving when medically assessed prior to diving.[7] One in eight children were excluded from diving on medical grounds.[7] Available evidence also suggests that even after medical clearance, children still have a high rate of ear, nose and throat (ENT) issues, specifically middle ear barotrauma.[4,7,9,12,16] Training organisations which vet their paediatric and adolescent divers by establishing pool skills and optimising ear equalisation techniques before proceeding to open water environments, appear to have a high degree of success with safe diving practices in their young trainees.[7]

Fatalities are infrequent in children and adolescents compared to adult recreational divers, although participant rates are far lower.[9] The Australasian Diving Safety Foundation data from 1966 to 2020 revealed five out of 531 scuba deaths (1%) were in children and adolescents (aged 8–15 years).[17] One series from DAN North America reported that children (defined as aged 12–17 years) made up 1.9% of all deaths reported between 2012–2015.[18] These data did not permit incidence to be calculated. Although infrequent, any child or adolescent diver death is unacceptable.[19–23] The cause of most paediatric deaths was arterial gas embolism from pulmonary barotrauma.[18] Anxiety and/or panic was a common precedent to rapid ascent in children and adolescent divers, which in turn resulted in pulmonary barotrauma.[9,22] Pulmonary barotrauma and subsequent arterial gas embolism can occur in water depths as shallow as one metre.[24] Asthma may also result in pulmonary barotrauma.[12,25] Decompression sickness was a less commonly confirmed diagnosis in one paediatric population.[9] Depth restrictions and less provocative diving may have reduced incidence of decompression sickness in this series. Fortunately, recompressing children with hyperbaric oxygen for decompression sickness under the current adult guidelines appears safe.[22]

Disclaimer

The advice contained in this SPUMS Position Statement is applicable to medical health risk assessment of children and adolescents aged 10–15 years who are seeking to undertake compressed air diving including scuba diving and surface supplied compressed air (e.g., 'hookah' diving).

The statements do not constitute a 'Standard'. The statements are based on analysis of available published evidence and expert opinion. They are expected to provide guidance to medical practitioners when undertaking health risk assessments on children and adolescent prospective divers.

The document should be used on a case-by-case basis utilising information on individual circumstances and as broad guidance for doctors. The society recommends engagement with the child or adolescent, their parent(s) / legal guardians, and the dive instructor when assessing 'fitness to dive' of the applicant. From the age of 15 years, adult guidelines apply.

Statement 1

The definition of a paediatric/adolescent diver for SPUMS diving medical assessment is from attaining the age of 10 years to less than 15 years of age.*[4]

* The society recognises that there is considerable individual variability of physical and emotional maturity in this age range, which needs to be taken into account by the assessing doctor. See Statement 11 for additional recommendations. The society also recognises that there are other definitions and published age ranges for this population.[12,18,22]

Statement 2

It is the society's position that all prospective children and adolescent divers should be medically assessed for health risks that may be incompatible with diving before commencing scuba diving training. It is recommended that doctors who perform diving medical assessments on children and adolescents have undertaken additional professional development in diving medicine and are up to date with specific risks for this population. Where there is doubt or the child has complex health issues, additional specialist (or specialist centre) advice should be sought.

For children (and those who are legally minors), such a medical assessment would also include consent from the parent(s) / legal guardian to confirm appropriate education regarding risk has been covered, understood and accepted by both parties.[1,4,23] The doctor should determine the reason why the child wishes to dive and their motivation and should be mindful of any excessive coercion from care givers.[4,6,12,16,23,25]

Statement 3

Dive medical assessments should be performed:

- prior to initial training for any compressed air diving, including scuba and surface supply diving, provided in open and contained bodies of water (from 10 years of age), and
- following any significant health event.

Statement 4

In addition to adult contraindications which preclude diving, children or adolescents should not dive if they have any of the following medical conditions:

- Epilepsy (any type including absence seizures);[10]
- Combined anxiety disorder and panic disorder;[7,9,12,22,23]
- Attention deficit hyperactivity disorder;[12,16,26,27]
- Asthma (including well controlled and exercise induced), cystic fibrosis, and other chronic respiratory tract illness;[6,7,10,12,16,22,23,27,28]
- Congenital heart disease despite correction;[10,12,27]
- Insulin dependent diabetes mellitus;[10]
- Migraine with aura;[10]
- Tympanostomy tubes present in either or both ears;
- Hereditary or acquired bleeding disorders;
- Any medical condition that could cause sudden incapacity.[10]

This list is not exhaustive and detailed specialist advice should be sought regarding any specific medical conditions which are identified in children and adolescents who seek to dive.

Statement 5

There is evidence of increased potential risk from diving in children / adolescents compared with adults, particularly relating to:

- cognitive and emotional maturity, attention and focus, and antecedent risk for panic underwater;[5,7,9,12,22,28]
- attention deficit and hyperactivity disorder and associated potential risks;[26]
- risk of ear nose and throat and respiratory tract infections;[7,16,28–30]
- immaturity of the paediatric airway;[6,7,12,22,27,28,31]
- risk of persistent (patent) foramen ovale (PFO);[6,7,9,12,16,27,28]
- risk of hypothermia;[5,6,12,16,25,27,28]
- limited physical capabilities.[3–5,9,12,25,32]

Statement 6

The assessing diving doctor needs to pay careful attention to the child or adolescent's:

- past medical history;
- psychological maturity and executive function*;[3–5,7,9,12,16,22,25,32]
- physical maturity;[3–5,9,12,25,32]
- ear nose and throat assessment;[5–7,9,12,16,23,25,27,28,32]
- asthma risk;[9,12,23,27]
- risk of PFO;[6,9,12,16,27]
- hypothermia risk;[5–7,9,12,16,23,25,27,28]
- reported in-water and swimming capability**;[6,7,8,33]
- motivation for diving including whether the child perceives they are under pressure to dive.[4,6,12,16,23,25]

Physical examination should include a comprehensive medical assessment as performed for an adult diving medical examination, including pulmonary function testing and audiogram.[10]

* Where this is unable to be assessed accurately at interview, the assessing physician should seek further information from reliable third-party sources (e.g., other clinicians, allied health personnel, teachers).

**If a child is unable to swim, then they should not dive.

Statement 7

The society considers that for a diving doctor to form an opinion about medical risk for children and adolescents intending to scuba dive, the discussion with the candidate, legal guardian/s, and diving instructor must include:

- the child's / adolescent's swimming ability / in-water capability;[7]
- assessment of their level of maturity and understanding of the risks involved with diving;[22]
- assessment of their physical capabilities;[3–5,9,12,25,32]
- additional acceptance of risk by the legal guardian/s;[7]
- consideration of Gillick competency (determining whether a child / adolescent diver is functionally competent to provide informed consent).[34]

Statement 8

Recommendations for child / adolescent diver safety during subaquatic activities should include:

- emphasis of the need for physical and psychological fitness during their training;[1,4,5,23]
- emphasis of the need for accessory diving skills, including snorkelling and buoyancy control;[4,23]
- counselling regarding the risk of pulmonary barotrauma and resultant arterial gas embolism and the avoidance of panic;
- ensuring that the child / adolescent and their parents / guardians are complicit in this understanding and sign the acceptance of risk on the SPUMS Statement of Health for Recreational Diving;[1,4,5,10,23]
- determining that the child / adolescent is complicit in the decision to dive and not being coerced;[4,6,12,16,23,25]
- where possible, include the dive instructor in the decision making;[1,7,27]
- when diving, ensure:
  - » that a minimum of two adult certified, competent divers accompany the child or adolescent when diving; one of whom knows them well (e.g., parent or sibling);[9]
  - » the focus of the adults is as supervisors to the child or adolescent only;[3–5]
  - » the child or adolescent should be within arms-length distance from the adult and in direct view at all times;[9]
  - » that the child or adolescent diver is not expected to rescue their adult supervisor(s).[3–5,9,12,25,32]
- encouragement for training agencies to develop specialised training modules (including on-line) to teach young divers and lead them on open water dives;[9]
- in addition to limitations in Statement 6, child or adolescent divers should not dive in hazardous marine environments as defined in AS/NZS 2815.6 (2013) Section 1.1.4 (a)–(g), listed in Appendix 1.[35]

These recommendations are best managed by training agencies who have a special interest in child and adolescent divers and can provide individualised support for the specific needs and unique behavioural aspects of this population.

Statement 9

Regarding garments and equipment for the child / adolescent diver, these should:

- be appropriately sized and fit;[7,16]
- be appropriate thickness of wetsuit for thermal protection in the planned water temperatures*;[33]
- be of a weight that the child can carry when walking;
- preferably have integrated weights in the buoyancy compensator device**.[12]

* Hypothermia is a greater risk in children due to higher surface area to volume ratio.

** This avoids the need for a weight belt which could more easily slip off a child, leading to a rapid ascent with subsequent pulmonary barotrauma / arterial gas embolism.

Statement 10

The society recognises that there is limited evidence of harm to children and adolescents who have undergone medical risk assessment by a doctor who has training in diving medicine, and who undertake compressed air diving in a controlled, supervised environment within current training systems.[7,9,18,20–22,27] However, available studies also provide limited evidence of safety and do not permit accurate assessment of risk or incidence of harm in the child / adolescent population of divers. The negative impact of fatalities and episodes of significant injury in children is of such magnitude that a conservative approach is warranted when providing health risk advice.

Statement 11

The society supports, in-principle, the position of other medical societies and experts to stratify children or adolescents by age, when considering the diving activity,

*** Footnote:** Appendix 1 can be found on the DHM Journal website: https://www.dhmjournal.com/index.php/journals?id=346

environment, water temperature and limitations on depth of diving, and number of supporting certified diving adults (minimum of 2), when the child / adolescent is diving.[4–7,12,16,28,33,36]

Recommendations

This guideline was based on expert opinion from SPUMS clinician members present at the 51st SPUMS Annual Scientific Meeting, Cairns, Australia, June 2023. Their expert opinion is based on lived experience and currently available literature, which is limited to expert consensus, case studies, prospective cohort studies, and retrospective analyses.

Conclusions

Children and adolescents are an important group within the diving population who have development-specific considerations. Close attention needs to be placed on the medical history and assessment of the ear, nose and throat, and respiratory systems, in-water capabilities, and neurodevelopmental evaluation due to antecedent risks in the subaquatic environment.

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Joint position statement on immersion pulmonary oedema and diving from the South Pacific Underwater Medicine Society (SPUMS) and the United Kingdom Diving Medical Committee (UKDMC) 2024

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Fitness for diving; Guideline; Immersion pulmonary edema; Scuba; Swimming induced pulmonary edema

Abstract

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This joint position statement (JPS) on immersion pulmonary oedema (IPO) and diving is the product of a workshop held at the 52nd Annual Scientific Meeting of the South Pacific Underwater Medicine Society (SPUMS) from 12–17 May 2024, and consultation with the United Kingdom Diving Medical Committee (UKDMC), three members of which attended the meeting. The JPS is a consensus of experts with relevant evidence cited where available. The statement reviews the nomenclature, pathophysiology, risk factors, clinical features, prehospital treatment, investigation of and the fitness for future compressed gas diving following an episode of IPO. Immersion pulmonary oedema is a life-threatening illness that requires emergency management as described in this statement. A diver with previous suspected or confirmed IPO should consult a medical practitioner experienced in diving medicine. The SPUMS and the UKDMC strongly advise against further compressed gas diving if an individual has experienced an episode of IPO.

Introduction

This joint position statement is the product of a workshop held at the 52nd Annual Scientific Meeting of the South Pacific Underwater Medicine Society (SPUMS) from 12–17 May 2024, and following consultation with the United Kingdom Diving Medical Committee (UKDMC), three members of which attended the meeting. The joint position statement is a consensus of experts with relevant evidence cited where available.

The purpose of the statement is to provide medical practitioners with guidance regarding immersion pulmonary oedema (IPO) and diving, and in particular the emergency management of IPO and return to diving following a diagnosed episode of IPO, or an event in which there is a high degree of suspicion.

The statement must be interpreted in consultation with a medical practitioner experienced in diving medicine and will be subject to review based on new evidence becoming available.

Definition

Immersion pulmonary oedema is acute pulmonary oedema that occurs in divers, snorkellers and swimmers whilst immersed. Some individuals have developed pulmonary oedema when surface swimming and on other occasions when scuba diving, which suggests that the main pathogenic mechanisms are related to immersion.[1,2]

It may affect the following people when immersed:

- Divers breathing compressed gas using scuba, 'hookah', other surface supply, and rebreather apparatus
- Snorkelers and breath-hold divers
- Swimmers, particularly triathletes, open water swimmers and combat swimmers with head out immersion

Nomenclature

Immersion pulmonary oedema is also known as:

- Immersion pulmonary edema (IPE) (US spelling)
- Swimming induced pulmonary oedema/edema (SIPO/SIPE)
- Scuba divers pulmonary oedema/edema (SDPO/SDPE)

Pathophysiology

The hydrostatic effect of head out immersion on peripheral veins causes redistribution of blood centrally, which increases heart size and significantly increases cardiac filling pressures, including pulmonary capillary pressure.[3] At the same time, because the lung centroid is below the surface of the water, respiration is with a continuous negative airway pressure equal to the vertical distance between the lung centroid and the surface of the water.[4]

This combination of increased pulmonary capillary pressure and negative airway pressure creates a pressure gradient for transudation of fluid from pulmonary capillary blood into the alveoli. However, given these physiological changes when immersed are essentially ubiquitous while IPO is relatively uncommon, it seems that for frank pulmonary oedema to develop additional factors are usually required (see risk factors below).

In a diver, the hydrostatic effects of immersion on redistribution of blood are identical to head out immersion, but the effects on intrapulmonary pressures are dependent on the type of breathing equipment used, the relation between the diver's lung centroid and the breathing gas source, and gas density.[5] An increase in external inspiratory resistance from breathing equipment and increased resistance to flow through airways due to dense gas will cyclically exaggerate any negative airway pressures during inspiration. As in those with head-out immersion, additional factors are usually required before frank pulmonary oedema develops.

There is individual predisposition to immersion pulmonary oedema as indicated by the fact that individuals can get recurrent episodes.[1,2,6–11]

In addition, some studies have shown that divers affected have haemodynamic differences compared with divers who have never had IPO.[1,10]

Occasionally individuals that have had IPO experience pulmonary oedema in other extreme circumstances, which is in keeping with an increased susceptibility.[1,12]

It is also clear that IPO can be fatal.[8,13]

Risk factors

A number of risk factors for IPO have been identified:

Intrinsic:
- Previous episode of immersion pulmonary oedema[1,2,6–11]
- Female sex[11,14]
- Older age[11]
- Hypertension and / or pre-existing cardiovascular disease[1,2,13,15–17]

Extrinsic:
- Colder waters[1,14,17,18]
- Equipment – causing excess negative inspiratory pressures from regulators, rebreathers[2,5,19–23]
- Severe exertion[5,7,9,24]
- Excessive hydration[9]

Ascent: In divers, particularly those using open circuit, symptoms related to hypoxia may occur or worsen on ascent and/or after surfacing as the inspired partial pressure of oxygen (PO_2) decreases.

Symptoms, signs and diagnosis

The following are the main clinical signs and symptoms of IPO, but the diagnosis does not require the presence of them all:

- Cough
- Dyspnoea (shortness of breath)
- Expectoration of frothy sputum (which may be blood stained/ pink) or haemoptysis
- Moist or rattling breath sounds and wheeze
- Chest tightness
- Cyanosis and hypoxaemia
- Confusion and agitation
- Unconsciousness
- Cardiorespiratory arrest

NOTE: Signs and symptoms are predominantly respiratory with secondary effects due to respiratory failure and hypoxia e.g., chest tightness, weakness, vomiting. There is a wide range of severity of symptoms in affected individuals.[21,22,25–27]

The clinical diagnosis of IPO takes into account the temporal relationship during immersion, history and onset of signs and symptoms of the affected individual:

- Suspect if there has been a previous episode of IPO
- Suspect if there is an episode of dyspnoea during

immersion (in particular without aspiration), with any of the above signs and symptoms, and when the casualty has 'moist' breathing and crepitations on auscultation, evidence of arterial hypoxaemia clinically (i.e., cyanosis) or on oximetry. Because swimming is usually in the prone position, auscultatory signs of IPO are often predominantly or entirely in the anterior chest.[28] IPO may be unilateral and if so, the right lung is most commonly affected[28]

Differential diagnoses include aspiration and near drowning, pulmonary decompression sickness (the 'chokes'), and pulmonary barotrauma.

Sometimes, the diver or swimmer has recovered before being medically assessed.

INDICATIONS OF IPO NOTED BY DIVE BUDDIES:

- Diver is coughing
- Diver appears to be more breathless, is breathing more rapidly (may be apparent from exhaled bubbles when on open circuit) or is using breathing gas at a faster rate than is appropriate for the degree of exertion involved in the dive
- Diver mistakenly believes they are out of breathing gas, or their breathing equipment is malfunctioning (may be apparent if a diver switches to own back up regulator, flushes their equipment or requests gas supply from buddy)
- Agitation, panic, and compulsion to ascend

Recommended pre-hospital treatment of IPO

- Immediately terminate the dive and leave the water as soon as possible
- Ascend safely but omit 'safety' decompression stops and, if the casualty is very breathless or distressed, consider omitting compulsory stops. If compulsory stops are omitted, it is important to give normobaric oxygen on the surface, observe for signs of decompression sickness and inform the local recompression facility
- At surface, establish positive buoyancy but avoid over inflation of buoyancy compensator
- Rescue and remove affected individual from the water as quickly as possible[21]
- Targeted ABCD assessment
- Provide O_2 – highest concentration possible[21]
- Maintain chest upright / supported propped-up position for breathing efficiency
- Remove tight diving gear and / or wetsuit
- Keep the casualty warm
- Transfer to a hospital emergency department
- Intravenous access but restrict intravenous fluids
- Non-invasive ventilation with constant positive airway pressure (CPAP) if possible or positive end-expiratory pressure (PEEP) (early if available)[29,30]
- If the casualty is severely affected, consider providing assisted ventilation in the field via bag-valve-mask with PEEP valve if available
- Vasodilators (provided blood pressure is normal or high)
- Patients with IPO are not usually fluid overloaded. Therefore, diuretics are not a first line treatment of IPO and should only be considered a second line treatment after use of non-invasive ventilation and vasodilator medication

Investigations

The following investigations may support the diagnosis of IPO.

- Oximetry confirmation of hypoxaemia[18,28]
- Point of care ultrasound findings consistent with pulmonary oedema[5,18,28]
- Chest X-ray findings of pulmonary oedema[18]
- High resolution CT chest findings of pulmonary oedema
- Echocardiography soon after admission with IPO may show left ventricular dysfunction, particularly in older individuals.[31] Occasionally an individual with IPO will have echocardiographic or other evidence of Takotsubo cardiomyopathy[32,33]

Future suitability for diving

When assessing future fitness to undertake compressed gas diving or open water swimming following an episode of IPO, the following must be considered.

THE POSSIBILITY OF UNDERLYING CARDIOVASCULAR DISEASE

Immersion pulmonary oedema could be an indication of underlying cardiovascular disease especially in older divers, and the diagnosis warrants a detailed cardiovascular and respiratory assessment by specialists in conjunction with specialists in diving medicine.[2,15,31]

Detailed cardiovascular assessment is advised, including 24-hour blood pressure monitoring and echocardiogram, to identify treatable pre-existing cardiovascular disease.

If hypertension is present, further tests to exclude a primary cause such as renal artery stenosis should be considered.[2]

Other tests to exclude myocardial ischaemia and dysfunction, such as stress echocardiogram, exercise stress test, myocardial perfusion scan, CT coronary angiogram and stress cardiac magnetic resonance imaging scan should be considered, as should a test of fitness that includes peripheral oxygen saturation monitoring.

RISK OF RECURRENCE

IPO has a high risk of recurrence.[1,2,6–11] Fatal recurrence is documented.[8,13]

Recurrent episodes can occur even when thorough investigations have found no abnormal results. However, what constitutes a thorough work-up before clearance to undertake diving has not been clearly defined.

Any decisions about returning to compressed gas diving require careful consideration of the risks applicable to the specific individual and should be made in consultation with a diving medicine physician and / or a cardiologist with an interest diving medicine. It is recognised that some centres have significant experience and expertise in assessing these individuals.[4,13,21,22,34]

SPUMS/UKDMC statements regarding returning to compressed gas diving after an episode of IPO

STATEMENT 1

SPUMS and UKDMC strongly advises against further compressed gas diving if an individual has experienced an episode of IPO.

STATEMENT 2

All divers who have had an episode of IPO should be fully investigated to identify any disease that predisposed to the condition because it may have implications for the individual unrelated to future diving. (e.g., IPO has occurred in divers with significant coronary disease, cardiac valve disease, cardiomyopathy, renal artery stenosis, etc).

Investigations should be overseen by physicians and cardiologists experienced in diving medicine.

STATEMENT 3

If divers choose to dive again despite the advice in Statement 1, they must be fully informed of the risk, including that a recurrent episode of IPO may be fatal.[8,13,35]

If divers choose to dive again despite the advice in Statement 1, they should only do so after satisfactory treatment / resolution of any disease or risk factors identified during the full investigation recommended in Statement 2.

If divers choose to dive again despite the advice in Statement 1, they should be made aware of potential risk mitigation strategies such as: wearing high quality well-fitting thermal protection, avoiding heavy exertion, avoiding overhead environments or virtual ceilings (decompression diving), only diving if O_2 is immediately available after surfacing, avoiding dive locations remote from tertiary medical services, avoiding pre-dive overhydration, avoiding back-mounted counter lungs (rebreather divers).

STATEMENT 4

Depth limitation is not an acceptable risk mitigation strategy to prevent IPO. There is no known association between IPO and decompression sickness. It should be noted that if a diver develops IPO during a deep dive, it will take longer to surface and exit the water, particularly if decompression stops are required. In addition, an ascent from depth involves additional risks to casualties suffering from IPO, particularly when the diver is using open circuit breathing apparatus. First, the inspired pO_2 will decrease during the ascent. Second, ascents are usually performed head-up, which results in negative pressure breathing and that will exacerbate the development of IPO.

STATEMENT 5

A diver who has had IPO should be advised that there is a known association between experiencing IPO and subsequently developing hypertension.[1,2,36] Therefore they should receive life-long regular blood pressure checks – due to the risk of developing hypertension.

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Dr Turner acts as a consultant and proctor for St Jude Medical, Medtronic and Edwards Lifesciences, as a consultant and lecturer for Gore Medical and performs PFO closures on private patients. The other authors declare that they have no conflicts of interest.

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Case reports

Anaesthetic and surgical management of gastric perforation secondary to a diving incident: a case report

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Keywords
Hypoxia; Trauma; Surgery; Anaesthesia; Treatment; Ascent

Abstract

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Gastric perforation secondary to barotrauma is a rare surgical condition which may manifest as an acute abdomen and potentially lead to complications such as pneumoperitoneum. A 50-year-old, healthy, experienced diving instructor was transported to our emergency department for an acute abdomen and severe dyspnoea after a diving incident. Clinical suspicion combined with computed tomography scanning lead to the diagnosis of linear rupture of the stomach. Exsufflation of the abdominal cavity was performed in the emergency department and then the patient was sent to the operating room for emergency laparoscopic gastric repair. Post-operative management was focused on decompressing the stomach with a nasogastric tube and abdominal radiography with barium ingestion was performed to confirm the absence of leakage. The patient was discharged at postoperative day four. We found 16 similar cases in the published literature. Gastric perforation secondary to a diving accident is rare but requires rapid diagnosis and surgical treatment.

Introduction

Gastric perforation secondary to a diving accident is a rare event[1] that should be suspected in any patient who presents with abdominal pain after rapid ascent. The expansion of air in the stomach during ascent usually causes abdominal discomfort or pain, but in extreme cases, it can be responsible for a gastric or intestinal rupture with the development of a huge pneumoperitoneum. In most of the cases found in the literature[1,2] the perforation is secondary to a rapid ascent or dysfunction in the diving equipment, and occurs at the level of the lesser gastric curvature. Prompt diagnosis and surgery are needed to avoid serious complications such as peritonitis and sepsis.[3] In this article, we report a case of gastric rupture following barotrauma and the specifics of our anaesthetic and surgical management.

Case report

The patient consented to deidentified publication of his case details and images.

A healthy 50-year-old man, weighing 96 kg, was admitted to the emergency room after a diving accident with abdominal pain and severe dyspnoea. The patient was an experienced diving instructor, having over 600 dives to his credit. The medical history consisted of high blood pressure treated with perindopril and amlodipine. He had a meal and a glass of champagne about six hours before the dive and nothing but clear water afterward. At 40 m depth he felt dizzy and panicked, deciding to begin a rapid ascent towards the surface, while exhaling. He was conscious for the first 20 m of ascent, then passed out after experiencing sudden abdominal pain. His team pulled him out of the water and he spontaneously regained consciousness about two minutes later. At this point, he complained of severe abdominal pain and dyspnoea.

Emergency services were immediately contacted, and when the ambulance arrived 100% oxygen first aid was provided based on an initial diagnosis of decompression sickness. The patient was first taken to a recompression centre, where doctors determined he did not require recompression therapy and referred him to our hospital for treatment of the suspected gastric rupture. Thoracic and abdominal computed tomography (CT) (Figures 1 and 2) showed severe pneumoperitoneum with suspected gastric perforation. The images also revealed a significant elevation of the diaphragm

Figure 1
Computed tomography scan (axial view) showing severe pneumoperitoneum and gastric perforation at the lesser curvature of the stomach

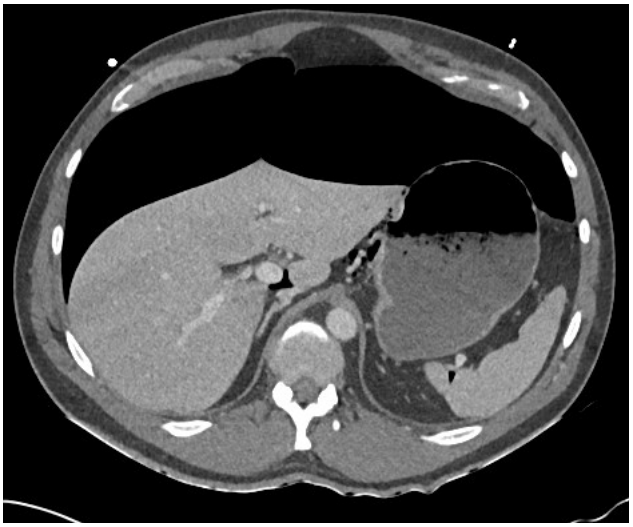
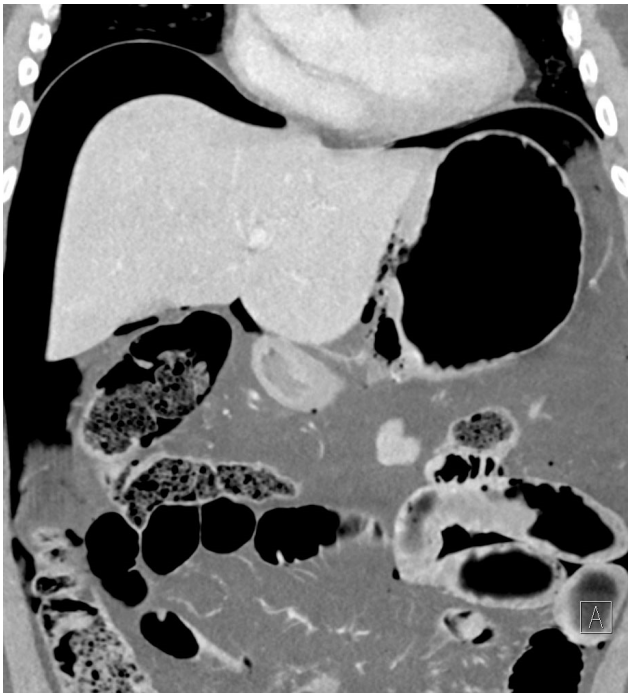


Figure 2
Computed tomography scan (coronal view) showing severe pneumoperitoneum and gastric perforation at the lesser curvature of the stomach



limiting lung expansion. The patient's respiratory condition worsened, with symptoms of respiratory failure indicated by mild hypoxaemia and tachypnoea, but he remained stable haemodynamically. After a discussion between surgeons, anaesthesiologists, and emergency physicians, it was decided to perform a bedside exsufflation of the abdomen under local anaesthesia. The patient was also administered 500 ml of intravenous NaCl. He then rapidly improved clinically and his respiratory parameters were better as oxygen supplementation was no longer required. However, abdominal distension and discomfort were still present. A gastric tube was placed, and prophylactic intravenous antibiotics were administrated. The patient was then sent to the operating room.

ANAESTHETIC CONSIDERATIONS

Laparoscopic exploration was planned to be performed under general anaesthesia with orotracheal intubation. Due to the large volume of gastric contents on the scanner and to protect the airways, we decided to perform a rapid sequence induction without ventilation after three minutes of pre-oxygenation with 100% oxygen. For the induction we used sufentanil, ketamine, lidocaine, and propofol, along with a muscle relaxant (rocuronium). For haemodynamic support, the patient was preloaded with 500 ml of IV crystalloid fluid. Our main concern for induction was the inferior vena cava, which was invisible on the CT scan, being compressed by the pneumoperitoneum. The consequent decrease in preload and thus the decrease in cardiac output posed a risk of cardiac arrest during induction due to the additional hypotensive effect of the drugs used. Despite the risk being at least partially eliminated[4] by the exsufflation performed preoperatively, we preferred to consider the patient at risk and took precautions to minimise the reduction of preload during induction. Additionally, divers are often relatively dehydrated, which prompted us to administer fluids to the patient prior to induction. In addition, surgeons were ready to perform an urgent laparotomy during induction if necessary, to relieve pressure on the inferior vena cava. Fortunately, no complication was reported with the induction of general anesthesia. Blood gas analysis was performed shortly thereafter and showed only respiratory acidosis, possibly secondary to the restrictive syndrome. Since several studies[5,6] have failed to demonstrate the effectiveness of cricoid pressure in reducing complications such as gastric regurgitation and aspiration, it has become standard practice at our institution not to use cricoid pressure. We chose rocuronium at a dose of 1.2 mg / kg over succinylcholine because it has a longer duration of action (lasting approximately 30 minutes) and can be re-administered as necessary to maintain muscle relaxation throughout the procedure. Additionally, rocuronium was chosen for its benefit of having an antidote available to reverse paralysis.

SURGICAL CONSIDERATIONS

Laparoscopic exploration was performed. A 1 cm supraumbilical incision was made for the insertion of the first optical trocar. Carbon dioxide was insufflated into the peritoneal cavity, followed by the insertion of the camera. We initially insufflated CO_2 at a pressure of 9 mmHg (instead of 12 mmHg) with a low flow rate. Once approved with our anaesthesia team, we proceeded to increase the pressure to 12 mmHg. There were omental adhesions to the peri-and infra-umbilical part of the anterior wall of the abdomen and the suspensory ligament of the liver. Complementary

Figure 3
Laparoscopic view of the haematoma at the lesser gastric curvature with contained perforation

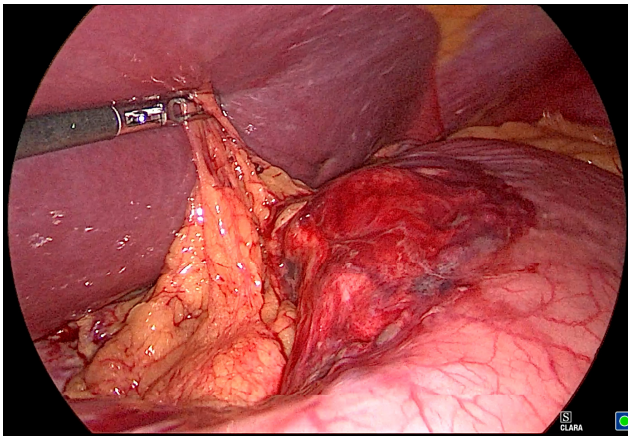
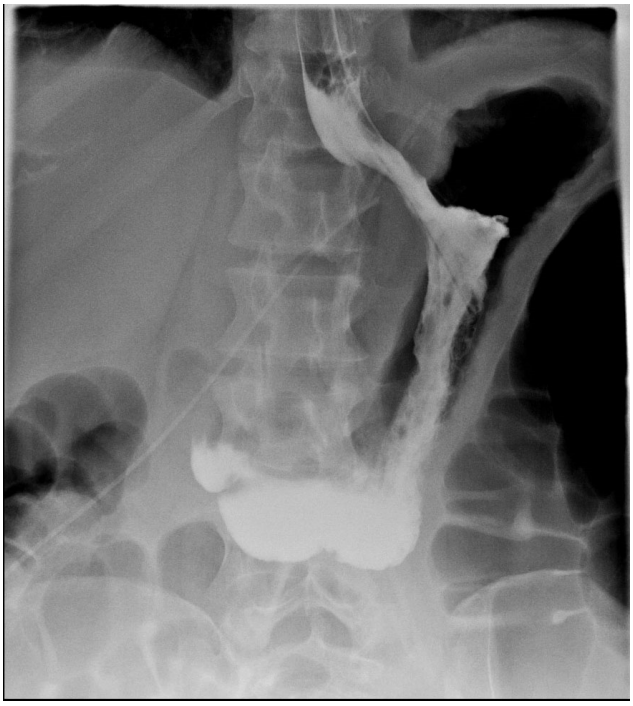


Figure 4
Radiographic examination with barium ingestion a day two after surgery confirming the absence of leakage



trocars were placed in the two hypochondria and then in the two flanks. The exposure of the stomach was made without difficulty. There was a haematoma (Figure 3) at the level of the lesser gastric curvature with suspicion of a contained perforation. Dissection along the medial border of the stomach confirmed the presence of a 6 cm long perforation, the exposure of which was improved by releasing its posterior border. A primary closure was then performed with a V-LOC 2-0 absorbable suture. Injection via a gastric tube of 700 ml of methylene blue-stained saline solution confirmed gastric wall integrity. The abdominal cavity was checked and widely washed, including the omental bursa and pelvic cavity. Two drains were positioned along the stomach. The patient was extubated at the end of the surgery and kept under close observation. A radiographic examination of the upper gastrointestinal tract with ingestion of gastrografin (barium) was performed two days after surgery to confirm the absence of leakage (Figure 4). Nasogastric aspiration was then discontinued and the patient was allowed to eat. He was discharged safely on the fourth day without any symptoms except for mild pain at the incision sites. His recovery progressed smoothly, and he achieved a full recovery by his six-week follow-up.

Discussion

Gastric rupture secondary to barotrauma during a diving accident is a rare event but should be suspected based on history. Quick diagnosis is required as potentially lethal complications such as peritonitis and sepsis are to be considered. Specific pathophysiological considerations must be understood (e.g., pneumoperitoneum) as they can be challenges for the induction of general anesthesia and the surgical procedure.

Our PubMed search using the terms 'diving' OR 'barotrauma' AND 'gastric' OR 'stomach' AND 'perforation' OR 'rupture' revealed 16 case reports. In virtually all the reported cases, gastric perforation typically occurs at the lesser curvature where the stomach is relatively fixed to adjacent structures and presents a single muscular layer and fewer mucosal folds, making it less elastic than the rest of the stomach. Rapid ascents lead to increases in airspace volumes, potentially resulting in barotrauma and decompression sickness,[7] which is why they are consistently discouraged. The pneumoperitoneum secondary to a gastric rupture will lead to a compression of other intra-abdominal structures, including the inferior vena cava, and push the diaphragm upwards, potentially causing an acute respiratory insufficiency. At sea level, the atmospheric pressure is 101 kPa, and each metre of descent increases the hydrostatic pressure by approximately 10 kPa.[8] The absolute pressure is the sum of the atmospheric pressure + the hydrostatic pressure, meaning that at 40 metres it is about 500 kPa, and at 20 metres around 300 kPa. We can then observe that the pressure increases by 100% between the surface and 10 metres and by only 20% between 30 and 40 metres. According to Boyle's law (pressure x volume = constant) a rapid ascent from 40 metres would result in up to a 5-fold increase in gas volume in the stomach (depending on its distensibility). Since our patient did not drink any soda beverage before the dive and had a presumed empty stomach (his last meal was taken six hours before the dive), the most likely origin of the gas in his stomach is aerophagia, either caused by panic or by reflex during the dive or ascent.

Under normal conditions, the oesophagus functions as a pressure release valve, opening when stomach pressure exceeds a certain threshold to prevent excessive buildup. In this case, however, two scenarios are possible: a pre-

existing weakness in the gastric wall could have led to rupture at a lower pressure than the oesophageal opening threshold, or the pressure required to open the oesophagus was unusually high, allowing the stomach to rupture without venting. Potential contributing factors to the gastric wall's vulnerability, prior to the dive, include chronic gastritis, peptic ulcers, or subclinical tears that compromised the stomach lining. Furthermore, previous reports have identified fundoplication, a surgical procedure to treat gastroesophageal reflux disease, as a risk factor for gastric rupture among divers.[9]

Stomach rupture has occurred after ascent from varying diving depths, with documented cases reported as shallow as 27 metres.[10] The majority of documented cases involving gastric rupture from barotrauma are typically treated through operative management.[11,12] However, there are a few instances where non-operative approaches have been successfully employed for small, localised perforations in otherwise healthy, minimally symptomatic patients.[13,14] Rapid ascents from moderate depths that are likely to cause gastric barotrauma may also result in pulmonary barotrauma and arterial gas embolism and decompression sickness.[7] It has been suggested that if arterial gas embolism or decompression sickness is present or suspected, the patient should be admitted to a recompression centre and hyperbaric oxygen treatment should be administered prior to surgery.[10]

Conclusions

Gastric rupture secondary to a diving accident is rare but requires rapid diagnosis and surgical treatment. Understanding this rare complication and its underlying pathophysiology is important because treatment should be administered emergently. Our case report differs by emphasising our use of pre-induction bedside abdominal decompression, which has not been prominently featured in prior reports. This preventive strategy before induction of anaesthesia aimed to reduce intra-abdominal pressure, thereby mitigating the risk of difficulty with ventilation, induction-related hypotension and cardiovascular collapse.

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Recurrent cutaneous decompression sickness in a hyperbaric chamber attendant with a large persistent foramen ovale

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Keywords
Bubbles; Echocardiography; Hyperbaric oxygen treatment; Migraine with aura; Oxygen; Working in compressed air

Abstract

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A 41-year-old female nurse had cutaneous decompression sickness on two occasions after acting as an inside chamber attendant for patients receiving hyperbaric oxygen. She breathed air during the treatments at pressures equivalent to 14 and 18 metres of seawater, but each time she decompressed whilst breathing oxygen. Latency was 2.5 hours and one hour. She was found to have an 11 mm diameter persistent foramen ovale. It was closed and she returned to work without recurrence of decompression sickness. Review of the literature suggests that shunt mediated decompression sickness is an important occupational risk for individuals with a large right-to-left shunt when working in hyperbaric air, but the manifestations of decompression sickness differ in those who decompress whilst breathing oxygen compared with those who decompress whilst breathing air.

Introduction

The incidence of some forms of decompression sickness (DCS), particularly cutaneous, neurological and cochlear-vestibular DCS, is increased in divers with a clinically significant right-to-left shunt.[1–5] It is believed that a right-to-left shunt permits paradoxical embolism of venous bubbles that form after decompression from some dives and, if those bubble emboli invade tissues supersaturated with inert gas (usually nitrogen), the bubbles are amplified as the dissolved gas in the tissue passes down the concentration gradient from the tissue into the bubble.[6,7]

When there is shunt-mediated DCS, a large persistent foramen ovale (PFO) is responsible in about 88% of cases, an atrial septal defect in about 5% of cases and pulmonary shunts in about 7% of cases.[8]

A PFO is present in 27% of the population, but only individuals with a PFO that is large enough to permit significant numbers of venous bubbles to shunt right-to-left are at risk of shunt-mediated DCS.[3,4] The median diameter of atrial shunts that cause shunt-mediated DCS is 10 mm.[8] In contrast, only 1.3% of the population have an atrial shunt that is 10 mm diameter or greater.[8]

Shunt-mediated DCS commonly occurs after a dive profile that is considered low risk and that rarely cause DCS in divers who have no right-to-left shunt, but the dive profile has to be one that liberates venous bubbles. [2,6,7] In contrast, in amateur divers who have decompression sickness but have no right-to-left shunt, the preceding dives are usually provocative and / or deep.[2–4]

There are also case reports that describe shunt-mediated decompression sickness after hyperbaric exposure in dry conditions when working as an inside attendant in a therapeutic hyperbaric chamber, in compressed air tunnel work and in hyperbaric factory work.[9–12]

This report describes recurrent cutaneous DCS after acting as an inside chamber attendant for patients receiving hyperbaric oxygen treatment.

Case report

This patient has consented to publication of this case report. She has reviewed the description below and agreed to its accuracy.

A female nurse aged 41 years, height 162 cm and weight 76 kg, was referred because she had two episodes of cutaneous DCS in early 2002. Each episode occurred after she had been an attendant in a hyperbaric chamber for patients receiving hyperbaric oxygen. She had worked at a hyperbaric unit for 18 months and she usually acted as an

attendant in a chamber twice each week. Her first episode of DCS occurred after one of the standard hyperbaric treatments given regularly in that institution to patients having hyperbaric oxygen therapy. The second episode occurred after a treatment used less routinely.

Her first episode of DCS occurred after a hyperbaric treatment at 243 kPa pressure (equivalent to 2.4 bar, 14 m of seawater [msw] pressure) for 90 minutes. Whilst the patients breathed oxygen at 'depth', she breathed air, but she switched to 100% oxygen during decompression. Decompression took eight minutes from 243 kPa (14 msw) to 132 kPa (3 msw), with a five-minute stop at 132 kPa (3 msw) and then one minute for the ascent to surface pressure. About 2.5 hours after decompression, she developed an itchy and sore mottled rash between her shoulder blades. A colleague who had seen divers with cutaneous DCS told her that the rash looked like cutaneous DCS, but she did not report it to senior chamber staff and she was not recompressed. The rash resolved in eight hours.

The second episode occurred after she acted as the chamber attendant for a single patient with carbon monoxide poisoning who was treated with Royal Navy Treatment Table 60. She breathed air during the hour at 284 kPa (18 msw) and until 15 minutes into the decompression when, at 192 kPa (9 msw), she switched to 100% oxygen for the remaining 15 minutes of decompression. One hour after decompression she developed itching over the right side of her body, which progressed to a florid mottled purple and pink rash typical of cutaneous DCS from her right hip to her right shoulder. Cutaneous DCS was diagnosed by a senior hyperbaric physician. There were no other symptoms or signs. She was rapidly recompressed using Royal Navy Treatment Table 61 (US Navy Treatment Table 5). There was rapid and almost complete resolution of itching and rash. After the treatment she had minimal residual skin discolouration over her hip.

She had a history of infrequent attacks of migraine with aura, but no family history of migraine. There was no other relevant medical history. She smoked an occasional cigarette (less than four per week).

Transthoracic echocardiography with bubble contrast showed a very large right-to-left shunt without provocative manoeuvres, with shunting seen during the inspiratory phase of normal respiration consistent with an atrial shunt. After counselling about options, she elected to have trans-catheter closure of her atrial shunt.

An 11 mm diameter PFO was closed using a 25 mm Amplatzer PFO device in September 2002. She was treated with aspirin for six months and clopidogrel for one month. Following the procedure she complained of palpitations which were the result of atrial ectopic beats. They resolved within two months after a short course of treatment with bisoprolol. Two months after the closure procedure, she had transthoracic echocardiography with six bubble contrast injections and provocative manoeuvres and there was no evidence of any residual shunt.

She returned to work as a chamber attendant for hyperbaric treatment and had no recurrence of DCS. In 21 years following the closure procedure, she has not had any attack of migraine but has had infrequent and minor visual aura.

Discussion

In the past, high incidences of DCS were described following occupational hyperbaric exposures which cannot be justified these days. For example, as late as 1971, Ghawabi and colleagues reported a DCS rate of 0.97% after caisson workers were exposed to air pressures equivalent to 28 msw for up to six hours and 25 msw for up to eight hours.[13] The authors reported that only seven of the 55 workers had no episode of DCS during the project, whereas 37 of the 55 (67%) of the workers experienced cardiopulmonary DCS ('the chokes') and 44% had radiological evidence of bone infarction.[13] The high incidence rates are consistent with unsafe profiles and, because nearly every worker had DCS at least once, there is no need to postulate the role of physical predisposition to DCS, such as right-to-left shunts.

Not surprisingly, occupational hyperbaric exposures have become more conservative, but DCS has not been entirely eliminated.

More recent publications report small numbers of episodes of DCS in workers in hyperbaric chambers, but most reports fail to provide detailed information about the pressure exposure profile or gases breathed by the affected employee or the clinical manifestations of DCS.[14] Very few reports provide the results of tests to detect whether those affected had a right-to-left shunt.

There are three reports of DCS after dry hyperbaric exposure when the individual breathed air during the hyperbaric exposure and also during decompression, and the affected individual had a right-to-left atrial shunt, either a PFO or an atrial septal defect.[9–11]

Johnson-Arbor reported a 50-year-old man who had numerous uneventful decompressions (sub-atmospheric and after diving during military service), but he had two episodes of DCS when working as an inside hyperbaric chamber attendant.[9] One was cutaneous DCS after treatment of a patient at 608 kPa (50 msw), but details of the profile and gases breathed are not provided. A second episode of DCS occurred after the chamber attendant breathed air at 223 kPa (12 msw) for two hours and also breathed air during decompression: he did not breathe oxygen at any time during the treatment. Within 10 minutes of surfacing, he became irritable and then had progressive ascending weakness and paraesthesia of both legs with a sensory level at T7. Definite spinal and probable cerebral DCS was diagnosed. There was recovery following treatment with US Navy Treatment

Table 6. Subsequently transthoracic bubble echocardiography showed a large atrial shunt.

Diederich and colleagues reported a 32-year-old man who worked packaging materials in a tank pressurised to 223 to 243 kPa (12 to 14 msw) for three to four hours, four times each week.[10] The decompression procedure used is not stated. There is no comment about oxygen breathing and that seems to be unlikely. The report said "*When brought back to atmospheric pressure, he developed headache, chest tightness, nausea, arthralgias, and vision changes, which he described as 'looking through a kaleidoscope'*." This visual disturbance is consistent with a migraine visual aura. Unfortunately, the intervals between surfacing and onset of different symptoms are not stated but it appears to have been soon after decompression. He found himself stumbling due to acute right-sided weakness, which spontaneously resolved. Upon returning home, he noticed an extensive rash overlying his torso consistent with cutaneous DCS. An echocardiogram showed the presence of a PFO. The precise type of echocardiogram is not stated and no information is provided about the size of the shunt.

Kütting and colleagues reported that in 2002 a 44-year-old tunnel worker had neurological DCS with onset 10 minutes after 42 minutes at a pressure equivalent to 375 kPa (27 msw).[11] None of his colleagues had DCS. It is also reported that he had recurrent episodes of DCS in the previous 15 years as well as episodes of 'blurred vision' after hyperbaric exposures. The visual disturbances may have been migraine aura. All these episodes of DCS occurred in years when it was very unlikely that oxygen was breathed during decompression. He was found to have an atrial septal defect.

The prominent manifestations of DCS in the three individuals, who decompressed whilst breathing air, were neurological though some also had cutaneous DCS. Where stated the onset of neurological DCS was soon after surfacing: in two cases onset was about ten minutes after surfacing. This is consistent with the peak latency of shunt-mediated neurological DCS in divers.[4]

In contrast, the patient described in this report is one of two where the casualty suffered DCS after decompression whilst breathing oxygen. In both cases, the casualty had cutaneous DCS. Colvin and colleagues reported a 32-year-old male tunnel worker who had DCS after oxygen decompression from only his third pressure exposure.[12] He worked in air at pressure equivalent to 355 kPa (25 msw) for 2.5 hours followed by one hour and 19 minutes of oxygen decompression using the Swanscombe Table. Approximately two hours after decompression he started to develop extensive cutaneous DCS with visual disturbance consistent with a migraine visual aura and pain in his left shoulder: the rash was present in the skin over the back of the left shoulder. Joint pain is not a feature of shunt-mediated DCS except when there is shoulder pain with a rash over the painful shoulder.[3] A transthoracic echocardiogram with bubble contrast showed a very large atrial right-to-left shunt at rest. He was found to have a 9 mm diameter atrial septal defect, which was closed.

Colvin and colleagues also reported that field testing with Doppler ultrasound showed that use of the Swanscombe Table liberates small numbers of venous bubbles in some workers.[12] Evidence supporting paradoxical gas embolism in the case described by Colvin and colleagues was that he had a visual aura consistent with migraine aura after his hyperbaric exposure at a time when the brain would not be supersaturated because it is a fast tissue.[12,15] Migraine visual aura can be precipitated by bubbles passing across a right-to-left shunt and it does not require supersaturation of neurological tissues, because it sometimes occurs after bubble contrast echocardiography when there is no supersaturation.[15]

The patient described in this report had two episodes of cutaneous DCS after acting as an inside attendant breathing air during hyperbaric treatments of patients at 243 and 284 kPa (14 and 18 msw). She breathed 100% oxygen during decompression on each occasion. Onset of symptoms was 2.5 hours and one hour after finishing oxygen decompression. Her bubble contrast echocardiography showed a large atrial right-to-left shunt that was found to be across an 11 mm diameter PFO. It was closed. She had a history of migraine with aura, which is associated with large right-to-left shunts.[15]

The pressure-time profiles of the two chamber dives that resulted in cutaneous DCS in the patient described in this case report were comparable to profiles demonstrated to liberate venous bubbles in some hyperbaric chamber attendants even when there was a longer period of oxygen breathing during decompression.[16,17] For example, Cooper and colleagues reported that after breathing air for 90 minutes at 243 kPa (14 msw) with 20 minutes decompressing whilst breathing oxygen, 32% of subjects had moderate to high numbers of venous bubbles on Doppler.[16] Walker and colleagues reported that 44% of exposures liberated venous bubbles after subjects breathed air at 203 kPa (10 msw) for 90 mins followed by 30 mins breathing oxygen during ascent to the surface.[17] Sixty-eight percent of exposures liberated venous bubbles after subjects breathed air at 283 kPa (18 msw) for 60 mins followed by 30 mins breathing oxygen during ascent to the surface.[17]

As far as we are aware, the patient described in this report and the patient in the paper by Colvin and colleagues are the only cases in which DCS occurred in individuals that had dry occupational hyperbaric exposure with oxygen decompression. The information available suggests that despite oxygen decompression, the profiles would liberate venous bubbles in some individuals.[12,16,17] Both individuals had large atrial defects (an 11 mm diameter PFO and a 9 mm diameter atrial septal defect). They each had cutaneous

DCS, which is commonly shunt-mediated, with onset times between one and 2.5 hours after surfacing. One had migraine visual aura at the time of their cutaneous DCS which is suggestive of paradoxical gas embolism.[15] However, in contrast to the individuals with atrial shunts who had DCS after dry hyperbaric exposures but who decompressed whilst breathing air, the two individuals who decompressed breathing oxygen did not suffer neurological DCS.

We cannot draw firm conclusions from small numbers of observations, but the data from these five case reports are consistent with the hypothesis that shunt-mediated DCS requires more than paradoxical gas embolism. It has been hypothesised that the additional requirement is amplification of embolic bubbles in supersaturated tissues as dissolved gas in the tissue diffuses into the bubbles.[6,7]

An alternative hypothesis is that shunt-mediated cutaneous DCS is not the result of amplification of bubble emboli in subcutaneous tissue, but is caused by paradoxical gas embolism to the brain that results in alterations in vasomotor control to produce the mottled skin rash of cutis marmorata, which has visual similarities to livido reticularis.[18,19]

Kemper and colleagues claimed that this hypothesis is supported by the incidental observation during experiments to investigate the effects of cerebral air embolism in which anaesthetised pigs developed a mottled skin rash, which bore a resemblance to the rash of cutaneous DCS in divers.[18,20] The development of the rash in the pigs was not reported in the original paper by Weenink and colleagues.[20] Later, Kemper and colleagues reported that in the experiments, each of the 22 pigs developed the rash within minutes of introducing air into the cerebral circulation.[18] However, the circumstances and findings in the pig experiments differed in many ways from those in divers with cutaneous DCS.[20–22]

The pigs (weights approximately 40 kg) were anaesthetised with ketamine and midazolam, paralysed with pancuronium and given atropine.[20] The experiment involved injection of 5.6 $\pm$ 1.3 ml of air directly into the ascending pharyngeal artery (equivalent to an internal carotid artery in humans) with the artery occluded by means of an inflated balloon.[20] The pigs had not been exposed to high ambient pressures before the air was injected. Therefore, their tissues were not supersaturated with gas. They were ventilated with a F_iO_2 of 0.4 during the experiments, including a stabilisation period of at least one hour.[20] Therefore the tissue partial pressures of nitrogen in the pigs in the experiments would have been lower than in a person or pig breathing air and lower than in a diver soon after a dive. The tissue partial pressure of nitrogen would also have been lower than the partial pressure of nitrogen in the air injected into the animals' cerebral vessels. As a result, the experimental model was more in keeping with cerebral arterial gas embolism in a non-diver occurring during medical interventions (for example, during cardiac surgery). In these clinical situations, a rash similar to cutaneous DCS is not a characteristic finding. Nor is the rash of cutaneous DCS a characteristic feature of cerebral arterial gas embolism in divers.

In the pigs, the rash had a wide distribution over the cheeks, neck, thorax, abdomen and thighs.[22] In divers, cutaneous DCS is usually localised to areas of the body with significant amounts of subcutaneous adipose tissue, such as over the trunk and/or thighs. In individual divers, who have recurrent episodes of cutaneous DCS, there is often a similar distribution of the rash on each occasion.

In the pigs, the rapid development of the widespread rash when air was injected coincided with large and rapid increases in intracranial pressure and a severe deterioration in cerebral metabolism.[18,20] Some pigs died immediately and the rest were euthanized. Kemper and colleagues have confirmed that if any of the animals had survived the experiments, they could have had severe neurological deficits.[22] We believe that the magnitude of the effects on intracranial pressure and metabolic derangement make it certain that if any pig survived they would have had severe neurological injury.

It is agreed that the associated rapid increases in heart rate and blood pressure in the pigs could have been the result of a catecholamine surge caused by the severe cerebral injury.[21,22] Livido reticularis is described in patients with phaeochromocytoma.[23,24] Therefore, we believe that the widespread rash observed in the pigs during the experiments reported by Weenink and colleagues was the result of the severity of the neurological injury they suffered.[20] In contrast, most divers who have cutaneous DCS do not have even mild neurological manifestations, not even when they have multiple episodes of cutaneous DCS.

There have been some attempts to demonstrate bubbles in skin rashes after diving.

Garcia and Mitchell reported ultrasound examination of the skin of four divers 4–5.5 hours after surfacing from relatively innocuous dives and 2–4.5 hours after the onset of cutis marmorata.[25] In each case, bubbles were detected passing through the microvasculature of the affected subcutaneous tissue, but not through adjacent normal skin. Each diver was later found to have a right-to-left shunt. These observations do not provide conclusive evidence about causation, because the rash was present before the bubbles were detected. Therefore, it is possible that the detection of the passage of bubbles through the affected subcutaneous tissue but not in unaffected skin could have been the result of differences in cutaneous blood flow in affected and unaffected tissues. In addition, each diver had neurological DCS at the same time as they had cutis marmorata. However, it is also possible that in affected subcutaneous tissues, bubbles were more easily detected because their size was increased by bubble amplification, but was not in unaffected skin.

It might appear that the failure of Qing and colleagues to detect bubbles in skin lesions of pigs after simulated dives in hyperbaric chambers is at variance with the report by Garcia and Mitchell, but the dive profiles were much more provocative.[26] Thirteen pigs were compressed to 507 kPa (40 msw) for 35 minutes followed by 11 minutes decompression. All animals developed widespread skin lesions and two died suddenly from what appears to have been cardiorespiratory DCS, which is consistent with a highly provocative dive profile. Transthoracic echocardiography was performed at various times from 30 minutes until six hours after surfacing. The bubble grade was greatest on the 30-minute images, when there was 'white-out' of right heart chambers in most pigs. That was also in keeping with a highly provocative dive profile. No bubbles were seen in the left heart chambers at any time. So it is unlikely that there was a right-to-left shunt, which makes it unlikely that the rashes in these pigs were the result of either paradoxical gas embolism to either the skin or the brain. As far as the authors could determine, the pigs that survived the experiment had no neurological injury. The rashes in the pigs may have had the same pathogenesis as cutaneous DCS after provocative dives in amateur divers who do not have a right-to-left shunt.

Additional observations support the hypothesis that paradoxical gas embolism with bubble amplification in subcutaneous adipose tissue can cause DCS and cannot be explained by a neurological mechanism secondary to cerebral gas embolism. Breast pain and a painful lipoma are described as manifestations of shunt-mediated DCS, but it is difficult to explain those as a result of cerebral gas embolism.[3,27]

Of 39 amateur divers that had lymphatic DCS, 30 had a significant right-to-left shunt and their dives were generally unprovocative.[28] In contrast, the remaining nine divers with lymphatic DCS either had no shunt or had only a small shunt but had performed deeper dives on trimix. Clearly lymphatic DCS cannot be explained by a cerebral insult.

The observations in individuals who had DCS after hyperbaric exposure in dry conditions may aid understanding of the role of tissue supersaturation in shunt-mediated DCS. A period of oxygen breathing during decompression allows tissues with rapid nitrogen elimination half-lives, specifically neurological tissues, to desaturate before venous bubble formation and paradoxical gas embolism occur. That means those tissues will not amplify bubble emboli. In contrast, tissues with a slow nitrogen elimination half-life, such as skin and subcutaneous tissue, remain supersaturated and able to amplify bubble emboli after decompression whilst breathing oxygen. In fact, prolonged oxygen breathing during decompression, as described by Colvin and colleagues, may actually slow elimination of dissolved nitrogen from some tissues, such as subcutaneous fat because of the vasoconstrictor effects of high partial pressures of oxygen.[29]

Although these are only a small number of cases, they add to the evidence refuting the hypothesis that cutaneous DCS is the result of a neurological mechanism caused by gas embolism to the brain.

It is difficult to draw conclusions from a small number of case reports, but these limited data suggest that individuals, who have a large atrial right-to-left shunt, either a PFO or an atrial septal defect, make up the majority of people who have DCS as a result of working in modern hyperbaric facilities. In each case, their manifestations of DCS were similar to manifestations of shunt-mediated DCS commonly observed in scuba divers.

Therefore, the guidance produced by SPUMS and UKDMC for assessment of divers who might have a PFO is also applicable to other hyperbaric workers such as inside chamber attendants and hyperbaric tunnel workers.[30]

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Five consecutive cases of sensorineural hearing loss associated with inner ear barotrauma due to diving, successfully treated with hyperbaric oxygen

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Abstract
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Introduction: This report describes the outcomes of sensorineural hearing loss (SNHL) due to cochlear inner ear barotrauma (IEBt) in five divers treated with hyperbaric oxygen (HBOT).
Methods: The case histories of five consecutive divers presenting with SNHL from IEBt due to diving, were reviewed. All divers provided written consent for their data to be included in the study. All had reference pre-injury audiograms. All noted ear problems during or post-dive. Independent audiologists confirmed SNHL in all divers prior to HBOT, then assessed outcomes after HBOT.
Results: Three divers breathed compressed air on low risk dives, and two were breath-hold. None had symptoms or signs other than hearing loss, and none had vestibular symptoms. All could equalise their middle ears. Inner ear decompression sickness was considered unlikely for all cases. All were treated with HBOT 24 hours to 12 days after diving. Two divers received no steroid treatment, one was treated with HBOT after an unsuccessful 10-day course of steroids, and two divers received steroids two days after commencing HBOT. All divers responded positively to HBOT with substantial improvements in hearing across multiple frequencies and PTA4 measurements. Median improvement across all frequencies (for all divers) was 28 dB, and for PTA4 it was 38 dB.
Conclusions: This is the first case series describing use of HBOT for IEBt-induced SNHL. The variable treatment latency and use/timing of steroids affects data quality, but also reflects pragmatic reality, where steroids have minimal evidence of benefit for IEBt. HBOT may benefit diving related SNHL from IEBt with no evidence of perilymph fistula, and provided the divers can clear their ears effectively. A plausible mechanism is via correction of ischaemia within the cochlear apparatus. More study is required including data collection via national or international datasets, due to the rarity of IEBt.

Introduction

Inner ear barotrauma (IEBt) affecting divers is rare, but can result in significant morbidity, potentially ending the diver's career.[1] The exact incidence is unknown, but even in specialised centres it can take years to accrue large numbers of cases.[2,3] The pathophysiology in divers is believed to be either explosive or implosive, or internal to the cochlea.[4]

For explosive injury, pressure during descent is transmitted to the tympanic membrane. In cadaveric studies, the tympanic membrane has been demonstrated to rupture at 97.7 kPa (nearly 1 atmosphere absolute [atm abs]) additional pressure.[5] If the tympanic membrane doesn't rupture, the pressure is transmitted through the auditory ossicles to the oval window, which transmits a hydraulic pressure wave via perilymph to the round window, which may rupture.[6] Explosive injury will be exacerbated (or precipitated) by forceful Valsalva against a locked Eustacian tube which raises perilymph fluid pressure, concurrently with negative pressure in the middle ear. For implosive injury, the mechanism is believed to be a sudden increase in middle ear pressure when a forceful Valsalva is successful in opening the Eustacian tube, and the tympanic membrane bulges outwards, distracting the auditory ossicles with it, leading to rapid lowering of perilymph pressure in association with a middle ear under positive pressure. This causes inward force on the round window breaching its integrity.

Despite emphasis in the literature about the round window as the injury point, the oval window can also be injured in a contra-coup way during pressure forces, however the ossicles may offer some protection. In addition, high energy pressure from blasts could cause injury to multiple structures in the

auditory chain. The round window has been observed to rupture in anaesthetised cats at mean pressures of 3.2 kPa (23.4 mmHg) above atmospheric, and in Norwegian cattle cadavers at greater than 202 kPa (2 atm).[7,8] It is difficult to quantify the pressure leading to injury in humans, but these animal studies produced values which are consistent with the range of pressures encountered in diving. In addition to implosive and explosive forces, barotrauma can lead to injury within the sensitive cochlear apparatus. One study reported additional pressures of only 4.8 kPa (0.047 atm) were required to rupture Reissner's (basilar) membrane in cattle.[8] Intracochlear injuries may occur simultaneously with window ruptures.[2] It is also possible that internal cochlear injuries occur in isolation without window injuries. Cochlear injuries may result from rupture of the basilar membrane (intracochlear membrane tear) which leads to admixture of peri- and endolymph (dissimilar fluids), inner ear haemorrhage within the cochlea or direct disruption of the organ of Corti. Mechanisms causing injury to the inner ear have been previously documented.[6,9]

Diagnosis of IEBt is challenging, with the main differential diagnoses being inner ear decompression sickness (IEDCS) (especially if vestibular symptoms are present), or middle ear barotrauma (MEBt) if symptoms are restricted to hearing loss and tinnitus.[6,9,10] Detailed clinical assessment including air conduction and bone conduction audiometry is required to differentiate cochlear IEBt from MEBt, the former demonstrating sensorineural hearing loss. Treatment options and recommendations for IEBt have been previously documented.[6,9,10] There is limited advice available for how to treat IEBt when hearing loss is the sole injury; steroids are unproven for this condition, and surgical exploration has very limited supporting evidence of benefit for hearing loss.[9–11] The logic for applying HBOT for IEBt originated from accumulating evidence demonstrating benefit from HBOT as a combination treatment for acute idiopathic sudden sensorineural hearing loss.[12–16] Despite this evidence, there is also a possibility that pressurisation and HBOT may worsen IEBt.

Outcome measurement using pure tone average across four frequencies: 500 Hz, 1,000 Hz, 2000 Hz and 4,000 Hz (PTA4), was used for consistency in a meta-analysis of HBOT for idiopathic sudden sensorineural hearing loss.[16] Although PTA4 forms only a small component of comprehensive hearing assessment, it does correlate with speech recognition.[17,18] This report details outcomes for five IEBt cases treated with HBOT.

Methods

All divers provided written consent for their cases to be reported.

Five consecutive divers were assessed at the Department of Diving and Hyperbaric Medicine for hearing loss after diving, from October 2019 to May 2023. All divers had pre-injury audiograms for reference, and post-injury, all satisfied the definition used for idiopathic sudden sensorineural hearing loss which is 30 dB hearing loss across three frequencies.[16,19] No diver had any other symptoms or signs. All divers had ear, nose and throat specialist consultation prior to referral to the hyperbaric facility. This specialist assessment included full history, clinical examination, and audiometry (with air conduction and bone conduction). The main criteria for exclusion of other possible diagnoses were clinical, taking into account dive type (freedives vs scuba dives), symptom onset relative to dive profile, dive profiles, lack of other risk factors for DCS, ear clearing problems, isolated (cochlear) symptoms, and absence of any other DCS symptoms. These criteria were identified as being useful to separate IEBt from IEDCS in a recent systematic review.[20]

In addition to confirming sensorineural hearing loss, the process of exclusing other diagnoses included neurological examination, and diving medicine specialist visual assessment of middle ear function (to ensure ease of ear clearing). The most likely diagnosis for all divers in this series was intracochlear IEBt (Appendix 1*).

Before proceeding to HBOT, all divers were provided with a detailed discussion about the experimental nature of using HBOT for their condition. This discussion was conducted with diving medicine specialists who had access to all of the diver's information. In particular, the divers were given the option of immediately aborting pressurisation if there were any ear clearing problems, or if they felt at any stage they did not want to continue with treatment. Divers also understood there was a possibility that HBOT could make their condition worse. The diving medicine specialists personally checked ear clearing capability for each diver (with observable movement of the tympanic membranes, by visual inspection during gentle active Valsalva manoeuvres. All divers provided written consent to receive HBOT.

All divers received courses of HBOT at 243 kPa (2.4 atm abs), after clinical assessment. Outcomes were assessed by comparing pre- and post-treatment audiometry using PTA4. In addition, the average loss across all nine standard audiogram frequencies 250–8,000 Hz compared to the non-injured ear was also assessed, and the number of frequencies with positive or negative change after intervention. Divers were followed up for a minimum of three months after receiving HBOT.

Results

Appendix 1* provides more detail about each diver's case history. All divers in this series were male: one scientific

* Appendix 1 can be found on the DHM Journal website: https://www.dhmjournal.com/index.php/journals?id=347

diver, two aquaculture divers, one hyperbaric professional and one recreational diver. Their ages ranged from 22–62 (median 51) years. All had pre-injury audiograms from either occupational diving medicals (≤ 12 months previously) or an occupational hearing assessment (the recreational diver). All divers had evidence of sensorineural hearing loss (across three or more frequencies) assessed by an independent audiologist prior to referral to the hyperbaric facility. Only one diver had imaging of their brain or internal auditory meati before receiving HBOT. For some divers, fistula tests were performed which were negative (see Appendix 1*). No diver had vestibular or neurological symptoms or signs, nor other symptoms except for their affected ear. No diver exhibited abnormal neurological signs when examined. All divers were able to clear their middle ears with gentle Valsalva manoeuvres, and were considered low risk of MEBt with chamber pressurisation. All divers successfully cleared their ears during HBOT and none sustained further symptoms or injury. The narratives for each case report are summarised in Appendix 1*, and their audiograms summarised in tabular form in Appendix 2*.

The details of the divers and outcomes of HBO treatment are summarised in Table 1. No diver had evidence of vestibular dysfunction. Audiometry in the last 12 months was normal for all divers with no significant difference between right and left ears except subject 3 in Table 1 who had pre-existing symmetrical significant (50 dB) hearing loss across 3–8 kHz. All divers had improved hearing following courses of HBOT ranging from 5–10 treatments at 243 kPa. Median improvement across all frequencies (for all divers) was 28 dB, and for PTA4 it was 38 dB. Three divers received steroids, two after two HBOT treatments and one for 10 days prior to HBOT (without benefit). Hearing improvements persisted to three months follow-up for all divers. Two divers had normal magnetic resonance imging scans, one had a normal computed tomography scan of the petrous temporal bones and two had no imaging of the brain or auditory meati.

Discussion

The author has been unable to locate previous reports of the use of HBOT for IEBt with isolated hearing loss and believes that the evidence for IEBt as the most likely diagnosis in all cases is robust, except perhaps subject 4. Subject 4 had sensorineural hearing loss following a single low risk dive, but with no clear history of ear injury. Symptoms were confined to the ear but had onset some hours after diving. This creates a degree of uncertainty when using published diagnositic criteria.[20] If case 4 was IEDCS, it may have been isolated cochlear DCS which is very rare.[20–22] All divers had symptoms localised to one ear including sensorineural hearing loss, confirmed by independent audiologists. Their injuries occurred during or were noted following diving. Divers 1 and 3 were solely shallow free diving, so IEDCS was not a possibility. Diver 2 had a 5-minute exposure to pressure in two short, controlled bounces, making IEDCS unlikely. Diver 5 reported definite barotrauma injury restricted to one ear with no other symptoms on dives that were relatively low risk.

All diver histories were checked in detail (face to face interview) by the author either at the time they presented or during their HBOT and at follow-up. As far as can be reasonably ascertained by direct questioning, ear clearing difficulties were infrequent. Two divers (2 and 4, both commercial divers) denied they had any ear clearing difficulties. Diver 5 described an actual event of right ear injury (associated with a mild upper respiratory infection). The breath-hold divers 1 and 3 noted ear problems during or after their dives (diver 3 acknowledged an actual injury at 5 m). Diver 1 may not have been snorkelling deep enough (2.4 m) to notice pain, but the fact that he continued an underwater hockey tournament for a week indicates any equalisation problems were minor. It is known that divers may under-report their injuries, but only one diver reported an upper respiratory tract infection, either active or recent, which was unexpected.[4] No diver in this series demonstrated signs of MEBt (acknowledging that three divers presented more than a week after injury). Absence of MEBt was noted in 38% of cases in one series,[4] and not found to be a useful discriminator in another,[20] mainly due to insufficient reporting in IEDCS series.

A reported series of IEDCS cases showed it is rare for IEDCS to be solely localised to the cochlea (6% of cases), hence isolated hearing loss is more likely to favour IEBt as a diagnosis.[21] An amalgamated review of four papers confirmed a low incidence of isolated cochlear DCS (5%), and a strong association of IEDCS with air divers from depths greater than 30 metres.[22] In another series of IEDCS 28 cases, 10 subjects had hearing loss, all had symptoms of vertigo, postural instability and 9/10 had nystagmus. None had isolated hearing loss.[23]

Using Rozycki et al's. HOOYAH criteria, all five cases presented in this report strongly favour cochlear IEBt and not IEDCS.[6] No diver had vestibular symptoms making it unlikely that any had a perilymph fistula affecting either round or oval windows. No divers went greater than 18 m and two were breath-hold. In addition, their ability to pressurise inside the hyperbaric chamber made perilymph fistula unlikely.

Lindfors et al. recently reported a systematic review to identify criteria which would help differentiate IEBt from IEDCS.[20] The most useful variables were dive type (free diving versus scuba diving), dive gas (compressed air vs mixed gas), dive profile (mean depth 13 vs 43 metres of seawater), symptom onset (when descending vs when

* Appendices 1 and 2 can be found on the DHM Journal website: https://www.dhmjournal.com/index.php/journals?id=347

Table 1

Diver characteristics and audiometry results post injury and post treatment; * hearing loss was the average loss in dB for all eight or nine measured frequencies compared to the non-injured ear for each diver; HBOT – hyperbaric oxygen treatment; SNHL – sensorineural hearing loss; SSBA – surface supply breathing apparatus

Diver	Diver profile	Ear clearing issues before problem	Initial symptoms	Audiogram post-injury description	Number of frequencies affected	Hearing loss* post-injury (dB)	PTA4 loss post-injury (dB)	HBOT day started, (*n* HBOT), When steroids started	Hearing loss* post-HBOT (dB)	PTA4 loss post-HBOT (dB)	Improvement PTA4 post-HBOT (dB)	Number of frequencies improved
1	Snorkelling under-water hockey 2.4 m max depth	No	Tinnitus reduced hearing right ear	Down-sloping SNHL	7 out of 9	43	46	Day 8 (9xHBOT) Steroids day 10	8	4	43	9
2	Compressed air SSBA to 18 m for 5 minutes	No	Tinnitus hearing loss left ear	Flat moderate to severe SNHL	8 out of 8	68	75	Day 0 (10xHBOT) Steroids day 2	21	23	53	8
3	Snorkelling < 5 m	Yes	Difficulty clearing and pain in right ear	Flat moderate to severe SNHL	5 out of 9 4 were pre-existing	29	65	Day 14 (10xHBOT) Steroids 10 days prior, no benefit	1	28	3	5
4	Compressed air SSBA to 15 m well inside table limits	No	Reduced hearing	Upsloping SNHL	8 out of 9	31	39	Day 7 (10xHBOT) No Steroids	6	10	29	8
5	Compressed air scuba to maximum 16 m well inside table limits	Yes	Pain right ear reduced hearing, tinnitus 24 h later	Down-sloping mild SNHL	7 out of 9	24	39	Day 12 (5xHBOT) No Steroids	8	25	14	6

ascending or surfacing), distribution of cochleovestibular symptoms (vestibular versus cochlear) and absence or presence of other DCS symptoms. Symptoms of difficult middle ear equalisation or MEBt were not reliable due to insufficient reporting in the IEDCS series.[20] Even with useful criteria, differentiation of IEBt vs IEDCS still frequently devolves to a balance of probabilities because there is considerable overlap in the symptom complexes of each condition.

On that basis, a question is raised: what is the pathophysiology of IEBt with sensorineural hearing loss, and how may HBOT have produced therapeutic benefit? It is acknowledged that recovery in all cases may have been spontaneous, and the temporal relationship of HBOT just a coincidence. An understanding of cochlear anatomy is useful to identify potential therapeutic mechanisms.

A basic depiction of ear anatomy is shown in Figure 1.[24] Figures 2 and 3 shows more detailed images of cochlea cross-sectional anatomy.[25,26] Figure 4 shows detail of the vascular supply to the vestibulocochlear apparatus.[27] The cochlear arteries and arterioles must travel inside the bone surrounding the cochlea (a relatively closed system). Supply of oxygen to the organ of corti is via the modiolar artery which provides arteriolar supply to the organ itself, the spiral ganglion, but mostly via the stria vascularis, allowing diffusion of oxygen to the organ of corti via the cochlear duct (scala media). There is some variability between species.[28] The cochlea is acutely sensitive to ischaemia, which may result from reductions in blood flow. It has been demonstrated in pigs that raised labyrinthine pressures cause reductions in blood flow which were reversed when the round window ruptured.[29]

It is conceivable that during IEBt, injuries less severe than the threshold for round window rupture could lead to localised swelling and raised perilymph hydrostatic pressures which in turn reduce the blood flow via the labyrinthine artery to the organ of corti, and induce hearing loss. This mechanism may precede basilar membrane rupture (in severity), and also precede rupture of the round window.

Given that all subjects in this report had demonstrable improvements in hearing after HBOT, it suggests that the IEBt was a reversible, non-structural injury. If any subject had physical injury to the round window, then it may have been minor, without perilymph extrusion or vestibular symptoms – a subclinical injury without fistula development. This has been suggested by Duplessis's group who investigated otoacoustic emissions testing in IEBt, and demonstrated abnormalities in divers undertaking multiple repetitive dives. Transient emission shifts were demonstrated more frequently with otoacoustic emissions testing than audiometry, suggesting potential for subclinical injury as a potential cause of sensorineural hearing loss.[30]

It is possible that IEBt actually spans a spectrum ranging from subclinical injury of the cochlear apparatus through to overt round or oval window ruptures. Less severe injury may precipitate local injury and oedema surrounding the window and/or focal intracochlear membrane injury. Isolated basilar membrane tears or intracochlear haemorrhage may cause hearing loss across multiple frequencies that is potentially less reversible.[9,10] A final unlikely mechanism for IEBt could be a small pneumolabyrinth, from middle ear gas entering the perilymph, rather than outward fluid leakage with a round window rupture.[31] Air could potentially enter the labyrinth with an implosive injury, rather than fluid extravasating. If this was proven to be the primary mechanism by which IEBt causes sensorineural hearing loss, then use of HBOT for the condition would not be regarded as controversial – it would be to shrink gas bubbles. Of the possible mechanisms causing reversible sensorineural hearing loss from IEBt, it is this author's belief that the injury/inflammation/oedema/vascular ischaemia pathophysiology is most plausible.

Figure 1
Cross section of ear anatomy including the inner ear and cochlea

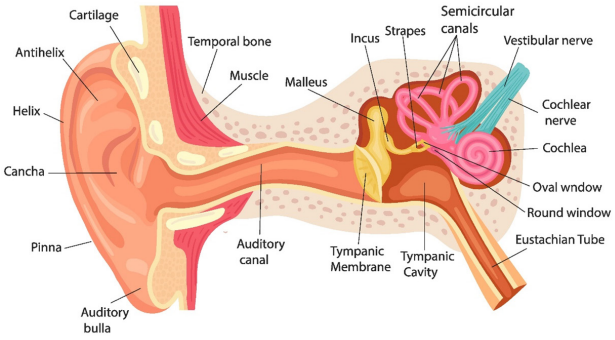


Figure 2
Cross section of one spiral of the cochlea

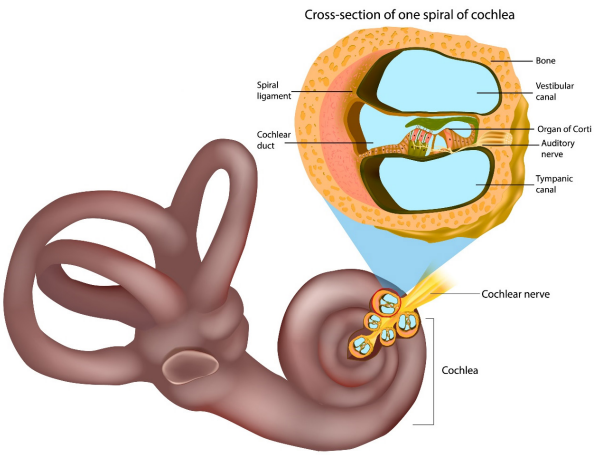


Figure 3
Detailed anatomy of the organ of Corti

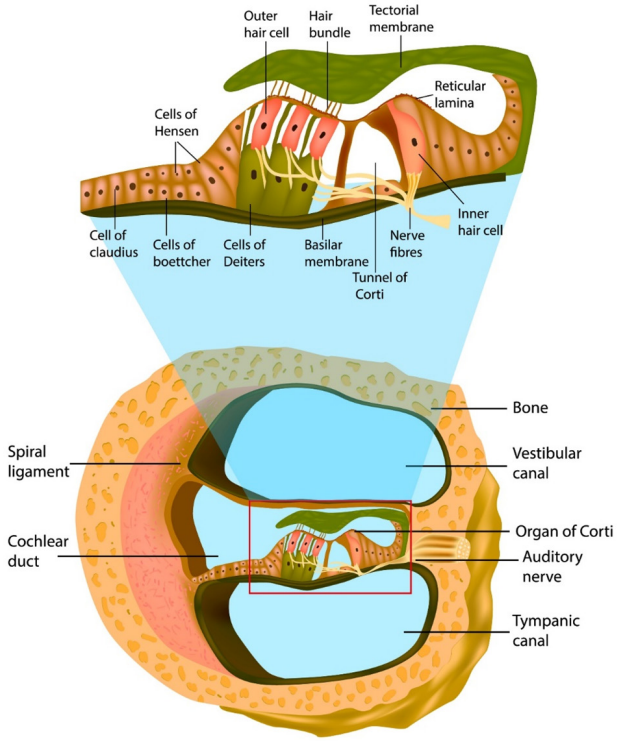
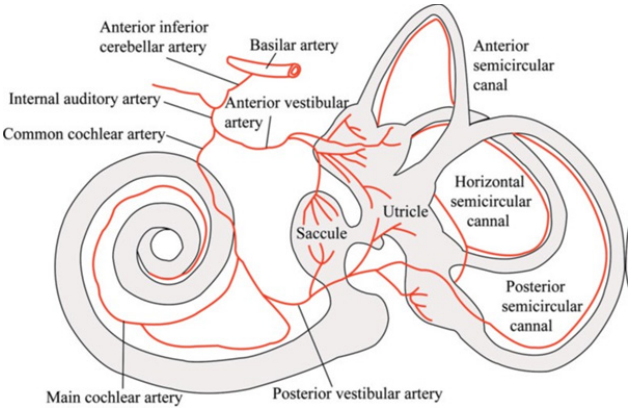


Figure 4
Diagram of the blood supply to the inner ear



The five divers in this series had flat or down sloping audiograms (highest frequencies worst), consistent with other reports.[32] The anatomical proximity of the round window to the cochlear vascular supply and organ of corti may be a factor in how IEBt affects hearing.[27] The base of the cochlea is located close to the round window, where the highest sound frequencies are detected. The arterial supply to both vestibule and cochlea is in close proximity.[27,33] There is a propensity for IEBt to have greater negative effect on higher frequencies.[6,32] The proximity of the structures (including venous drainage of the cochlea) provides some plausibility for a proposal of non-rupturing injury to the round window.[27,33] This could lead to oedema and raised perilymph/endolymph pressure causing ischaemia, as a pathophysiological mechanism of hearing loss.

There may be some potential parallels between idiopathic sudden sensorineural hearing loss and IEBt. There are multiple mechanistic theories for causation of idiopathic sudden sensorineural hearing loss. The vascular hypothesis proposes that ischaemia to the cochlear apparatus, cochlear nerve and other central auditory components is the cause of hearing loss.[34,35]

The use of HBOT for idiopathic sudden sensorineural hearing loss has been investigated extensively in recent years. A metanalysis concluded: HBOT as part of a combination treatment (with steroids) was significantly associated with improved hearing outcomes in patients with sensorineural hearing loss over control treatments.[16] Hyperbaric oxygen is now included as an option in ear nose and throat clinical practice guidelines.[36] In a recent retrospective series as a primary treatment, HBOT (without steroids) was effective in improving hearing in patients with idiopathic hearing loss.[37] The proposed mechanism of benefit of HBOT is via higher partial pressures of oxygen resulting in greater intracochlear oxygen tensions, in particular within the perilymph and endolymph, and reduction of inflammation and oedema.[34,35,37] It raises oxygen partial pressures and dissolved oxygen in plasma which correct cellular hypoxia through diffusion into ischaemic regions. In addition, HBOT has been demonstrated to reduce oedema via vasoconstriction and the osmotic effect of dissolved oxygen in plasma. It also reduces reperfusion injury.[38] These effects may explain why HBOT is effective for idiopathic sudden sensorineural hearing loss, and why the IEBt cases in this series responded. Reduction of oedema and restoration of oxygenation to an injured cochlear basilar membrane may also have beneficial effects. Further research is required to elucidate the pathophysiology of IEBt.

LIMITATIONS

This series had highly specific entry criteria, which are not frequently encountered: isolated sensorineural hearing loss from IEBt following diving. It is acknowledged that the diagnosis of IEBt (rather than IEDCS) has been made on the balance of probability for the cases, however low risk dives, breath hold dives and absence of any other symptoms makes IEDCS unlikely. It is also acknowledged there are potential confounders to the claim of efficacy of HBOT in these cases. The response to HBOT may have been coincidental, and the divers may have made spontaneous recoveries. The time-periods of unabated hearing loss (for divers 1, 3, 4 and 5), and the direct temporal relationship between HBOT and improved hearing lowers probability of such coincidence. Diver 2's response to HBOT the day after injury was particularly convincing. It is unlikely that steroids were a factor in recovery for these divers. Cases 1 and 2 had improvements in hearing before steroids were

administered (which followed their second HBOT). Case 3 was referred for HBOT after 10 days of steroid use had no effect on hearing, and cases 4 and 5 received no steroids. Hence steroids may have affected the outcomes in only 2/5 cases. The use of steroids for IEBt remains controversial, and has limited high-level supporting evidence.[9,11]

Conclusions

Hyperbaric oxygen may benefit sensorineural hearing loss from diving related IEBt which has no evidence of perilymph fistula, and provided the divers can clear their ears effectively for pressurisation. A plausible mechanism is via correction of ischaemia within the cochlear apparatus. More study is required in this field, including data collection via national or international datasets, due to the rarity of IEBt. The selection criteria used with these cases may provide guidance for future research.

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Presidents report

Jean-Eric Blatteau

As we approach the end of this year, I would like to take this opportunity to extend my warmest holiday greetings to all members of the European Underwater and Baromedical Society (EUBS), the South Pacific Underwater and Hyperbaric Medical Society (SPUMS), as well as the broader community of diving professionals and medical practitioners around the world. May this festive season bring you peace, joy, and inspiration for the year ahead.

In addition to celebrating the season, I am excited to announce the upcoming release of a major new reference book in diving medicine, particularly for the French-speaking members of our society. The book, titled "*Médecine de la Plongée*" ("*Diving Medicine*"), has been edited by myself (Jean-Eric Blatteau), along with Mathieu Coulange and Jean-Louis Méliet and is a comprehensive guide aimed at both medical and paramedical professionals, as well as diving instructors and guides.

Published by Elsevier Masson, the book offers a practical, hands-on approach to the prevention, diagnosis, and treatment of accidents related to recreational and professional diving activities, particularly in hyperbaric environments. It is structured in three main sections:

Fundamental concepts: We begin with a concise yet thorough overview of essential principles in physics and physiology, as they apply to diving. Topics such as gas exchange, decompression, and diving techniques are explored to provide readers with the foundational knowledge necessary to understand the risks and challenges of diving.

Accident prevention and management: The core of the book focuses on diving accidents and how to manage them effectively, both at the scene of the accident and in a hospital setting. One of the unique aspects of this book is its dual approach to clinical reasoning. In addition to traditional pathophysiological descriptions, we offer a **semiological approach** that begins with the symptoms, working back towards the underlying diagnoses. This contrasts with many texts that treat each pathology separately. By following this method, we guide the reader through the diagnostic process step by step, allowing for a more holistic and flexible understanding of diving-related medical conditions. The book also includes 'accident sheets' and 'management protocols', which are structured for ease of use, featuring diagnostic flowcharts and high-quality illustrations that facilitate quick, effective decision-making in emergency situations.

Systematic care levels: For each type of diving-related injury, we detail the different levels of care required, ensuring that readers can quickly identify the most appropriate actions and interventions at each stage of treatment.

This book is not only an essential resource for healthcare providers working in diving medicine but also for diving instructors and professionals who are responsible for the safety and well-being of their students and clients. Our goal is to make diving-specific medical knowledge accessible to a wide range of practitioners, ensuring that all those involved in the diving world are better equipped to handle the unique challenges posed by this sport and profession.

We are hopeful that an English-language version of "***Diving Medicine***" will soon be available to reach an even wider audience, furthering our shared mission to improve safety standards and enhance the medical support for diving communities worldwide.

As diving continues to grow in both recreational and professional spheres, it remains an activity that carries inherent risks. It is crucial that the practice of diving, whether for leisure or profession, be conducted within a framework of proper safety, training, and medical oversight. This book aims to support those efforts by providing the tools and knowledge necessary to manage those risks effectively.

I would like to take this opportunity to thank all of you for your continued commitment to diving safety and medicine, and to wish all the members of SPUMS, EUBS, and the readers of the *Diving and Hyperbaric Medicine Journal* a wonderful holiday season. I look forward to working with each of you to further advance our field in the coming year.

Wishing you all a peaceful and prosperous New Year.

Warm regards
Jean-Eric Blatteau
President, European Underwater and Baromedical Society (EUBS)

The EUBS website is at http://www.eubs.org/
Members are encouraged to log in and keep their personal details up to date.

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

EUBS Notices and news

EUBS 2025 Annual Scientific Meeting

After the great success of the 48th Annual Scientific Meeting in Brest, France, EUBS will move north, to Helsinki, Finland for its 49th meeting, which will take place from 2–6 September 2025. While this is somewhat earlier than usual, it will allow us to optimally benefit from the long days of summer which is ideal if you want to extend your stay either before or after the conference.

All details and information is available via the congress website www.eubs2025.com, as it becomes available. We urge you if you are considering attending to take advantage of the early-bird registration rates and book your spot early.

EUBS are delighted to welcome you to this meeting and contribute to its success.

EUBS Annual General Assembly

Our annual EUBS General Assembly took place on 20 September 2024, in Brest, on the last day of the EUBS Annual Scientific Meeting.

The report of the General Assembly, as well as the supporting documents (financial report, results of ExCom elections) is available for our members via the Member's area on the EUBS website.

The membership fees for the upcoming year will be increased, as they have remained unchanged for the past eight years. The new prices will take effect on 1 January 2025. The price for the 'print option' for the DHM Journal will remain at €50, which, considering the increased printing costs and a higher number of pages per issue, is still a bargain.

EUBS ExCom expresses their appreciation and thanks to our Corporate Members, as well as to our 10 'Affiliated Societies' – national scientific societies and organisations supporting and promoting EUBS among their members, who benefit from a 10% reduction in EUBS membership fee.

EUBS Executive committee

To replace Oscar Camacho from Porto (Portugal), after serving a four-year term, we have elected a new Member-at-Large. There were three highly qualified candidates: Pedro Coelho Barata (Portugal), Mihaela Ignatescu (UK) and Anders Kjellberg (Sweden). Anders Kjellberg has been elected as Member-at-Large 2024. Thanks to Oscar for his service to the Society.

The Executive Committee wish to express their thanks to all candidates and Members-at-Large for their willingness to help our Society move forward and their contributions to ExCom activities. A list of the new ExCom members can be found on the EUBS website, with contact information for each member.

EUBS social media

All EUBS members are reminded to bookmark and follow our social media channels:
Facebook: https://www.facebook.com/European-Underwater-and-Baromedical-Society-283981285037017/

X (formerlyTwitter): @eubsofficial
Instagram: @eubsofficial

While the 'EUBS website news' email messages are a way to communicate important information directly to our EUBS members, Facebook, X (formerlyTwitter) and Instagram will be used to keep non-members updated and interested in our Society. The EUBS social media is managed by Bengusu Mirasoglu (bengusu.mirasoglu@eubs.org).

EUBS membership

Don't forget to renew your EUBS membership. In case your membership has expired, you will see a message when trying to log in on the EUBS website. You can then immediately renew it online.

EUBS membership gives you significant advantages, such as immediate access to the most recent issues of the *Diving and Hyperbaric Medicine* journal, (if selected) a print copy of the ejournal for your convenience, reduced registration fee at our Annual Scientific Meeting (this alone already pays back your membership fee), reduced membership fees at selected Affiliate Societies, access to the GTUeM database of non-indexed scientific literature, searchable membership database, etc.

Members of Affiliate Societies benefit from a 10% discount on the EUBS membership fee. When applying for or renewing your membership, select your Affiliate Society from the drop-down list and the reduction in membership fee will be automatically applied.

In case you have difficulties renewing or accessing your membership area, please contact us at secretary@eubs.org. Please do note that payment by PayPal is by far the easiest and also cheapest way to pay your membership fee.

You can also pay by bank transfer, but please note, you will need to pay for the banking costs for international money transfers (EUBS is registered in the UK, which is now outside of Europe). Please ensure to select this ("*all banking costs carried by the sender*") when you make the transfer. If not, our bank will refuse your payment.

Also, the money transfer may take up to one week and may fail for some obscure reason.

Finally, please write your name (the EUBS member whose membership you are renewing) as ONLY information in the message attached to the payment, or we cannot identify the payment and your membership may not be renewed as expected.

Therefore, unless you are in the UK, we do not recommend this payment option. Using Wise (formerly 'Transferwise') is another option to reduce or avoid banking costs and have a faster and secure transfer of your membership fee.

EUBS website

Visit our EUBS website to be informed of News, Conferences and Meetings, Endorsed Documents and Courses. You can also find information on Travel and Research Grants, employment opportunities, research projects looking for multicentric collaboration, and much more.

The OXYNET database, previously managed by the European Committee for Hyperbaric Medicine (ECHM) is now an integral part of the EUBS website, and can be consulted through a Europe (and World) Map interface, through the Menu item 'OXYNET Map' (sounds logical) or directly at www.eubs.org/oxynet (or http://www.eubs.org/?page_id=1366).

Have a look at the 'EUBS History' section which has been added under the menu item 'The Society'. There is still some information missing in the list of EUBS Meetings, Presidents and Members-at-Large – please dig into your memories and help us complete this list.

Take a look at our Corporate Members – societies and companies who support the EUBS through membership. Their logos and contact information can be found at the Corporate Members page (http://www.eubs.org/?page_id=91).

If you have any suggestions for updating or correcting the information provided, please feel free to contact us at webmaster@eubs.org.

3Rd International Diving & Hyperbaric Medicine Conference - Muscat 2025

" EMPOWERING KNOWLEDGE AND PROFICIENCY "

Keynote Speaker
Professor Simon Mitchell - Consultant Anesthesiologist & Diving /Hyperbaric Physician

Themes
- Diving Medicine
- Hyperbaric Oxygen Therapy
- Marine Occptional Medicine

Visit Our Site Here
idhmc.mod.gov.om

REGISTER NOW
Early birds registration available

3 - 6 February 2025

Kempinski Hotel Muscat
Sultanate of Oman

- Health Care Professionals, Students
- Hyperbaric Physicians, Nurses & Technologist
- Diving Community
- Marine Scientists & Researchers

Organizers

South Pacific Underwater Medicine Society

Notices and news

SPUMS notices and news and all other society information can be found on:
https://spums.org.au/

President's report

Neil Banham

As we head towards Christmas, I would like to thank the work of all SPUMS members who have made 2024 another successful year, with special thanks to our hard-working ExCom and to Simon Mitchell and Nicky Telles and all others who contributed to our high-quality journal.

Our May Annual Scientific Meeting (ASM) was a great success with two Position Statements being workshopped and agreed upon which will soon be published. More details below.

The recently established Mike Bennett Scholarship, created to honour our great friend and colleague Professor Mike Bennett AM, has already received two applications. This Scholarship will fund the successful applicant to attend a Scientific Meeting of relevance to diving and hyperbaric medicine. The closing date is 31 December 2024. Further details regarding the Scholarship will follow my report in this issue as well as on the SPUMS website. South Pacific Underwater Medicine Society - SPUMS - Mike Bennett Scholarship.

The 2025 SPUMS ASM will be held in Bali, Indonesia. Diveplanit has again been contracted as our travel provider to assist Xavier Vrijdag and Hanna van Waart, our Bali ASM Convenors.

As of mid-November, there were already more than 50 registrants.

Dates: 18–23 May 2025.
Theme: "*Oxygen: Too little, too much or just right*"
Venue: Ramayana Candidasa, Bali, Indonesia
South Pacific Underwater Medicine Society - SPUMS-ASM.

Consideration for future venues for the 2026 and 2027 SPUMS conferences were canvassed at the 2024 Fiji ASM. Popular options were Palau and the Maldives, with Palau being agreed to as the preferred destination at our November virtual ExCom meeting.

Thank you to Doug Falconer and Ian Gawthrope who have volunteered to convene. Tentative dates are the week commencing Sunday 3 May 2026. Qantas are currently selling flights to Palau from 2025, departing Brisbane Saturday mornings and returning Sunday morning. Further details will hopefully be available on the SPUMS website in early 2025.

The Paediatric Diving Position Statement, which was workshopped at our 51st ASM in Cairns, will be published in the December issue of *Diving and Hyperbaric Medicine,* as will be the SPUMS and United Kingdom Diving Medical Committee (UKDMC) Joint Position Statement (JPS) on return for diving following an episode of Immersion Pulmonary Oedema (IPO). The updated SPUMS and UKDMC JPS on persistent (patent) foramen ovale (PFO) and diving originally published in 2015 will be published in the March issue of *Diving and Hyperbaric Medicine.* This JPS will also include an Appendix with photographs highlighting important quality control issues for bubble contrast echocardiography.

I am privileged to be invited to speak at the 3rd International Conference on Diving and Hyperbaric Medicine in Muscat, Oman from 3–6 February 2025. Our Editor, Simon Mitchell is also invited, along with EUBS President Jean-Eric Blatteau and UHMS President Peter Witucki. This will be a wonderful opportunity to network with colleagues and to learn about diving and hyperbaric medicine from a Middle Eastern perspective. My thanks to the organising team for this fantastic opportunity. Home | 3rd ID&HMC Muscat 2025 (mod.gov.om).

Nicky Telles and I have just finished updating the contact details for Australasian hyperbaric facilities on the SPUMS website. South Pacific Underwater Medicine Society - SPUMS-Hyperbaric Medicine Units.

The ANZHMG Introductory Course in Diving and Hyperbaric Medicine will be next held 17–28 February 2025, again in Fremantle. The 2025 course is now fully subscribed, with a wait list. I strongly suggest that you register your interest if you are considering attending the course in 2026 and dates again will be from mid to late February 2026 for two weeks. https://spums.au/index.php/education/spums-approved-courses-for-doctors.

Scholarships for trainees to attend this course are available thanks to the generosity of the Australasian Diving Safety Foundation (ADSF). For more information contact John Lippmann at johnl@adsf.org.au. ADSF also kindly sponsored SPUMS membership for a year for course participants.

I am pleased to be able to announce the commencement of data entry into the Australasian Decompression Illness Registry from 1 July 2024. Almost all Australasian hyperbaric facilities are currently participating, with the remainder hopefully completing the bureaucracy to participate soon. The Registry is hosted by Monash University and generously funded by ADSF and collects data on all divers treated for decompression illness. In the near future, data will be available for research purposes. This data set will be a useful resource for those seeking to complete their SPUMS Diploma thesis.

A reminder that just prior to the 2025 ASM, nominations for the position of SPUMS President-Elect will be sought, with the position being decided at the Bali AGM. The incoming President-Elect will have a year to 'learn the ropes' prior to the completion of my second 3-year term as President at the 2026 AGM. Please consider yourself for this.

On behalf of SPUMS ExCom, I wish everyone happy diving over the festive season and a safe and prosperous 2025.

Dr Neil Banham
President, SPUMS

Mike Bennett Scholarship

Dr Sue Pugh, the wife of the late Professor Mike Bennett AM (a past SPUMS President and mentor to many), has bequeathed funds to create a Scholarship ('The Mike Bennett Scholarship') to fund the successful applicant to attend a Scientific Meeting of relevance to diving and hyperbaric medicine.

Suitable meetings may include (but are not limited to) the Annual Scientific Meeting (ASM) of South Pacific Underwater Medicine Society (SPUMS), Undersea and Hyperbaric Medical Society (UHMS), European Underwater and Baromedical Society (EUBS), Hyperbaric Technicians and Nurses Association (HTNA), British Hyperbaric Association (BHA).

The Mike Bennett Scholarship will be offered annually with one successful applicant chosen if they are considered to meet the selection criteria. The Scholarship may not be awarded in any given year if the applications received are not deemed suitable by the Selection Panel.

The Mike Bennett Scholarship is open to anyone working in the field of diving and hyperbaric medicine, including doctors, technical staff, nurses and those performing research in the field. Applications from those from Pacific nations who might not otherwise have the opportunity to attend an international scientific meeting are also encouraged.

Selection of the successful applicant will be overseen by a SPUMS Selection Panel comprising:

Dr Sue Pugh
SPUMS President (currently Dr Neil Banham)
SPUMS Immediate Past President (currently Prof David Smart)
SPUMS Education Officer (currently Dr David Cooper)
Diving and Hyperbaric Medicine Journal Editor (currently Professor Simon Mitchell)

The successful applicant for The Mike Bennett Scholarship will have the actual costs of ASM Registration, travel and accommodation funded to a maximum of AUD $10,000. However, the applicant will be responsible for all other expenses incurred.

There are no rigidly defined Selection Criteria, however, preference will be given to the following:

- SPUMS members
- Presenting at the ASM:
  (1) A diving or hyperbaric medicine presentation
  (2) An evidence-based medicine presentation
- Those who have previously made a significant contribution to SPUMS.

Applications should include a brief synopsis (1–2 pages) of the project and be submitted to president@spums.org.au.

Closing date: 31 December 2024

Dr Neil Banham MBBS, FACEM, DipDHM, ANZCA DipAdvDHM
SPUMS President

SPUMS
South Pacific Underwater Medicine Society

SPUMS Facebook page
Find us at:
SPUMS on Facebook

The Australian and New Zealand Hyperbaric Medicine Group

Introductory Course in Diving and Hyperbaric Medicine

Please note: This course is fully subscribed with a waiting list. If you are considering attending the course in 2026, dates will again be from mid to late February 2026 for two weeks.

Dates: 17–28 February 2025
Venue: Hougoumont Hotel, Fremantle, Western Australia
Cost: AUD$3,200.00 (inclusive of GST) for two weeks

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

The course content includes:

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

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

The

SPUMS
South Pacific Underwater Medicine Society

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

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

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

Royal Australian Navy Medical Officers' Underwater Medicine Course

Dates: 10–21 March 2025

Venue: HMAS Penguin, Sydney

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

Cost: The course cost remains at AUD$2,332 (excl GST)

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

For information and application forms contact:
Rajeev Karekar, for Officer in Charge
Submarine and Underwater Medicine Unit
HMAS Penguin
Middle Head Rd, Mosman
NSW 2088, Australia
Phone: *+61 (0)2-9647-5572*
Fax: *+61 (0)2-9647-511*
Email: *rajeev.karekar@defence.gov.au*

HBOE
HBOEvidence

HBOEvidence

HBOEvidence is seeking an interested person/group to continue the HBOEvidence site. The database of randomised controlled trials in diving and hyperbaric medicine: hboevidence_wikis.unsw.edu.au. The HBOEvidence site is planned to be integrated into the SPUMS website in the near future.

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

SPUMS Diploma in Diving and Hyperbaric Medicine

Requirements for candidates (May 2014)

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

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

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

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

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

Additional information – prospective approval of projects is required

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

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

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

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

Projects will be deemed to have lapsed if:

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

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

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

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

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

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

SPUMS 53rd Scientific Meeting

Have you registered?

Courses and meetings

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

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

Scott Haldane Foundation

As an institute dedicated to education in diving medicine, the Scott Haldane Foundation has organized more than 320 courses all over the world, over the past 33 years. SHF is targeting on an international audience with courses worldwide.
Below the schedule of upcoming SHF-courses in 2025.

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

2025

March/ April	Level 1 course Diving Medicine part 1 The Netherlands
April/May	Level 1 course Diving Medicine part 2 to be decided
6–7 June	31st in-depth course diving Medicine "*Hear, smell, feel*", (level 2d) The Netherlands
8–15 November	32nd SHF in-depth course diving Medicine (level 2d) Bali, Indonesia
15–22 November	32nd SHF in-depth course diving Medicine (level 2d) Bali, Indonesia
On request	Internship HBOt (level 2d) NL/Belgium

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

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

(Short version – updated June 2024)

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

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

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

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

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 may be considered at the editor's discretion. These articles should be subdivided into the following sections: an **Abstract** (subdivided into Introduction, Methods, Results and Conclusions) of no more than 250 words (excluded from word count), **Introduction, Methods, Results, Discussion, Conclusions, References, Acknowledgements**, **Funding** sources and any **Conflicts of interest**. **Legends/captions** for illustrations, figures and tables should be placed at the end of the text file.

Review articles: up to 5,000 words is preferred and a maximum of 50 references (excluded from the word count); include an informative **Abstract** of no more than 300 words (excluded from the total word count); structure of the article and abstract is at the author(s)' discretion.

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

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

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

The journal occasionally runs **'World as it is'** articles; a category into which articles of general interest, perhaps to divers rather than (or in addition to) physicians or scientists, may fall. This is particularly so if the article reports an investigation that is semi-scientific; that is, based on methodology that would not necessarily justify publication as an original study. Such articles should follow the length and reference count recommendations for an original article. The structure of such articles is flexible. The submission of an abstract is encouraged.

Supplements to a particular issue are occasionally published for purposes deemed appropriate by the editor. These may accommodate articles / treatises that are too long for the main journal or collections of articles on thematic areas. There is no open portal for submission of such material and any plans or suggestions for supplements should be discussed with the Editor before writing.

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 in the full version of these instructions.

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

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

Instructions for authors (full version 2024 – this document)
DHM Keywords 2023
DHM Mandatory submission form 2024
Trial design analysis and presentation
Conflict of interest statement
English as a second language
Guideline to authorship in DHM 2015
Samples of formatted references for authors of journal articles (last reviewed 2024)
Recommendations for the conduct, reporting, editing, and publication of scholarly work in medical journals 2024
Helsinki Declaration revised 2013
Is ethics approval needed?

DIVER EMERGENCY SERVICES PHONE NUMBERS

AUSTRALIA – DAN
1800-088200 (in Australia toll free)
+61-8-8212-9242 User pays
(outside Australia)

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

ASIA, PACIFIC ISLANDS – DAN World
+618-8212-9242

EUROPE – DAN
+39-06-4211-8685 (24-hour hotline)

SOUTHERN AFRICA – DAN
+27-10-209-8112 (International call collect)

USA – DAN
+1-919-684-9111

JAPAN – DAN
+81-3-3812-4999 (Japan)

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.
