Diving and Hyperbaric Medicine

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Nutrition in patients receiving hyperbaric oxygen

Differentiating inner ear barotrauma and DCS

Tissue oxygenation and blood flow during HBO treatment

Scuba fatalities in New Zealand

Scuba interventions in neurological disability

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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

Many of us will be glad to see the end of another year in which SARS-CoV-2 has significantly impacted our lives, but the pandemic has not deterred the submission of high-quality work to DHM as evidenced by the content of this, our final issue for 2021. There are seven original articles and three case reports all addressing topics of high interest.

Rutger Lalieu and colleagues remind us that many patients receiving hyperbaric oxygen for wounds and radiation tissue injury may not be receiving the nutrition required for optimising wound and tissue healing. It is a timely reminder that the best outcomes for these patients are likely achieved when all risk factors for non-healing (including poor nutrition) are simultaneously corrected.

Oskari Lindfors and colleagues complete an excellent series of papers on ear and sinus barotrauma with a review aimed at informing that most difficult of differentials; inner ear barotrauma versus inner ear decompression sickness. Some distinguishing features were relatively obvious and some less so. The diagnostic guidance arising from their review will form a very useful resource going forward.

Naoki Yamamoto and colleagues provide arguably the most careful evaluation yet of the effect of tissue vasoconstriction on tissue oxygenation during hyperbaric oxygen administration in human subjects. This study is an extremely useful counter to the argument sometimes made that the potentially beneficial effect of hyperbaric oxygen is countered or offset by tissue vasoconstriction.

John Lippmann and colleagues provide the latest in a long series of studies investigation fatalities in Antipodean divers. As has always been the case with John's studies, this latest offering identified opportunities for targeted education for improving safety among divers in future.

Karlie Naumann and colleagues review the potential effects of scuba diving as positive intervention in sufferers of autism or neurological/intellectual disability. Scuba diving for disabled people is attracting wide interest presently. The review revealed little in the way of published evidence, but that in itself opens up the opportunity for motivated researchers to work alongside providers of scuba experiences for disabled divers, to quantify positive (or negative) outcomes and safety.

Marguerite St Leger Dowse and the DDRC group performed a very interesting survey to characterise the behaviour of travelling divers with respect to pre-flight surface intervals and outcomes. The study reminds us that decompression sickness certainly can occur when flying after diving. Even though adherence to current recommendations most often results in safe outcomes, occasional cases do occur despite such adherence.

Thijs Wingelaar and the Royal Netherlands Navy/ Amsterdam University Medical Centre group present the latest of their thematic studies that critically examine practices around assessing fitness to dive. In this paper they propose a change to interpretation of the FEV_1/FVC ratio that reduces investigations without compromising safety.

Yaling Liu presents a case of incipient cerebral herniation following clipping of a cerebral aneurysm which improved in temporal association with hyperbaric oxygen treatment. Based around a case of immersion pulmonary oedema, Jacques Regnard and colleagues present an interesting discussion of the potential respiratory and cardiovascular effects of a close-fitting neoprene vest.

In marking the last issue of the journal for 2021 I would like to take this opportunity to thank our peer reviewers for their high-quality work over the year. We try very hard to match the subject matter of papers to reviewer expertise, which means reviewers in some thematic areas receive multiple requests from me. Thorough, constructive review is crucial to quality scientific publishing and I am deeply grateful for the contributions our reviewers make. We will publish a list of 2021 reviewers in the first issue in 2022.

I would also like to thank outgoing EUBS President Ole Hyldegaard for his famous pragmatism and support of the journal over his tenure, and to welcome incoming president Jean-Eric Blatteau. Jean-Eric has an immense reputation in the field of diving medicine and I look forward to working with him in continuing to publish a high quality journal.

Finally, to end the year on a high note, and having recently reported national honours for two other members of our editorial board (David Doolette and Mike Bennett), I am delighted to recognise that on 08 December Prof Rob van Hulst was appointed Knight of the Order of the Lion of the Netherlands for his services to his country, to medicine and to our field (cover photo). Rob has had an extremely distinguished career involving military service, years of clinical medicine, ground-breaking research (particularly in relation to arterial gas embolism), and education. The energy Rob has put into mentoring young clinicians and researchers in diving and hyperbaric medicine, and the success he has enjoyed in doing so, has certainly been an inspiration to me. This extremely prestigious and rare award is richly deserved, and I have no doubt members of the two societies will join in congratulating Rob.

Simon Mitchell
Editor

Cover photo caption:
Prof Rob van Hulst and Foekje van Hulst after Rob's appointment as Knight of the Order of the Lion of the Netherlands. Photo: Hille Hillinga.

Original articles

Nutritional status of patients referred for hyperbaric oxygen treatment; a retrospective and descriptive cross-sectional study

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Key words
Hyperbaric medicine; Hyperbaric research; Irradiation; Nutrition; Obesity; Osteoradionecrosis; Wounds

Abstract
(Lalieu RC, Akkerman I, van Ooij PJAM, Boersma-Voogd AA, van Hulst RA. Nutritional status of patients referred for hyperbaric oxygen treatment; a retrospective and descriptive cross-sectional study. Diving and Hyperbaric Medicine. 2021 December 20;51(4):322–327. doi: 10.28920/dhm51.4.322-327. PMID: 34897596.)
Introduction: Due to the global rise of obesity, the role of nutrition has gathered more attention. Paradoxically, even overweight persons may be malnourished. This may delay wound healing or recovery of late radiation tissue injury (LRTI). Hyperbaric oxygen treatment (HBOT) is used to improve wound healing or LRTI complaints. The aim of this study was to assess the dietary intake levels of nutrients important for recovery in patients referred for HBOT.
Methods: This was a retrospective, cross-sectional study of patients referred for HBOT to a single centre between 2014 and 2019. Patients were offered a consultation with a dietitian as standard care. Information on nutrients was calculated from questionnaires, and compared to recommended daily allowances.
Results: One hundred and forty-six patients were included (80 female). Eighteen patients were treated for diabetic ulcers, 25 for non-diabetic ulcers and 103 for LRTI. Most were overweight or obese (64.4%), but did not consume the recommended quantities of calories, protein, or micronutrients. Vitamin C consumption was higher than recommended. Male patients had a higher intake of calories and protein than female patients but not other nutrients. No differences in intake existed between age or body mass index categories.
Conclusions: The nutritional status of patients referred for HBOT may be inadequate for healing wounds or LRTI, despite anthropomorphic data indicating a positive energy balance. Daily attendance for HBOT provides a unique opportunity to monitor and correct these deficiencies. Routine screening for malnutrition and supplement deficiencies is recommended for patients referred for HBOT.

Introduction

The important role of adequate nutrition in health has long been known. Over the last few years it has garnered increased attention due of the global rise in obesity.[1] Paradoxically, the presence of increased body mass index (BMI) does not translate to being well-fed. In fact, overweight people can still suffer from nutrient deficiencies, or malnutrition.[2] The worldwide prevalence of malnutrition is, therefore, high but often unrecognised, and is associated with adverse clinical outcomes and costs.[3]

Wound healing is one example of health that is negatively impacted by malnutrition.[4] Energy demand may be up to 50% higher than usual.[5] Nutritional deficiencies can prolong the inflammatory phase, decrease fibroblast proliferation and alter collagen synthesis, which may lead to a non-healing wound.[6] Elderly persons are especially at risk for this due to underlying comorbidities.[7] When ulcers become chronic, they can cause pain and other physical discomforts,[8] while quality of life is also negatively impacted.[9] They are also an economic burden, influencing health care budgets[10] and causing loss of work.[9] Non-healing ulcers, especially diabetic ulcers, may result in amputation of digits or limbs.[11] After amputation, overall mortality rate is significantly increased.[12] Due to increased life expectancy, the number of chronic wounds and associated costs is expected to rise in the coming years.[13]

Another example is late radiation tissue injury (LRTI). Radiation injury after head and neck cancer can present as osteoradionecrosis of the jaw,[14] which leads to dental pain, reduced oral intake and as a consequence, weight loss and low BMI.[15] Pelvic radiation may cause radiation enteritis and thus malabsorption of nutrients, leading to the same outcome.[16]

In both groups of patients, malnutrition may contribute to failure in wound healing as well as increased risk of postoperative complications, including infections, delayed recovery, and increased mortality.[17]

Hyperbaric oxygen treatment (HBOT) may be used to facilitate healing of both chronic wounds and LRTI.[18] The treatment involves inhalation of 100% oxygen under increased atmospheric pressure, and usually takes place daily for several weeks. The daily patient contact provides a unique opportunity to identify and monitor nutritional status in this at-risk population. Indeed, a recent study found that one-third of a population treated with HBOT were moderately or severely malnourished.[17] The aim of the current study was to assess the dietary intake levels of nutrients important for wound healing and recovery from LRTI in patients referred for HBOT to a single centre over a period of six years.

Methods

This study was approved by the local Medical Ethical Commission.

This study uses a retrospective and descriptive cross-sectional design to assess the dietary intake levels of nutrients for wound healing and general health. All patients treated in the hyperbaric centre of the Antonius hospital in Sneek, the Netherlands, are offered a consultation with a clinical dietitian (AB) as part of their standard care. All patients treated between 2014 and 2019 were eligible for inclusion in the current study. Patients were solely treated for chronic (diabetic) wounds and LRTI; no acute indications were treated. Patients who had been treated earlier at this centre, and thus already received advise on improving nutrition, were excluded. Additional exclusion criteria were the standard safety exclusion criteria for HBOT. No other exclusion criteria were applied. The data from the dietary consultation was generated as part of regular care and anonymously and retrospectively used in the current study.

The dietitian took a standard recall questionnaire of a single 24-hour midweek food intake period and entered this into a software system (Nevo-Online, version 5.0, 2016). The Dutch Food Composition Database (NEVO) contains data on the composition of foods. NEVO is owned by the Dutch Ministry of Health, Welfare and Sports, and is maintained at the Dutch National Institute for Public Health and the Environment (RIVM). Data generated included a nutrient analysis of the patient-reported diet on a per-day basis. The software programme extracts information on 26 nutrients from the entered data, including calories, proteins, fats and several vitamins and trace elements. Based on age, gender and body composition, a personal recommended daily allowance (RDA) was calculated. For vitamins and minerals, a standard RDA was provided based on European guidelines.[19] The increased nutritional requirements for wound healing were also taken into account when calculating the RDA. When deficiencies were found, supplements were prescribed. Vitamin C was always supplemented, as recommended for complex wound healing.[6] See Appendix 1* for an example calculation.

For this study, information on calories, proteins, iron, zinc and vitamins A, C, D and E was collected. Age and gender were recorded, and height and weight were measured to calculate BMI. BMI was categorised as underweight (< 18.5 $kg \cdot m^{-2}$), normal weight (18.5–24.9), overweight (25–29.9) and obese (> 30). The different treatment indications were categorised as LRTI and diabetic or non-diabetic wounds; LRTI was further split into different body regions (e.g., breast, head and neck, colorectal, etc.).

STATISTICAL ANALYSIS

Descriptive statistics for continuous variables were reported as medians with interquartile ranges. Discrete variables were reported as numbers and percentages. Comparisons of intake of all nutrients between gender, age (continuous) and BMI categories and different indications were made with one-way ANOVA. Daily intake of all nutrients was compared against the RDA for female and male patients separately. Since the data were not normally distributed after a Shapiro-Wilk test, a Wilcoxon signed-rank test was performed for the comparison. The null hypothesis for each test was that there is no difference between groups and that daily intake does not differ from the recommended intake. A P-value < 0.05 was considered statistically significant for all tests. All statistical analyses were performed with SPPS version 26 (IBM, New York, USA).

Results

During the study period, 146 patients were included, of which 80 were female (54.8%). Eighteen patients were treated for a diabetic ulcer, 25 patients for a non-diabetic ulcer and 103 patients for LRTI. The median age was 66 years (interquartile range [IQR] 53 to 74) and the median BMI was 26.4 $kg \cdot m^{-2}$ (IQR 23.8 to 29.5). Most patients (41.8%) were overweight (Table 1).

Footnote: * Appendix 1 is available on DHM Journal's website: https://www.dhmjournal.com/index.php/journals?id=287

Table 1
General description of population; data are median (interquartile range) or *n* (%); BMI – body mass index; LRTI – late radiation tissue injury

Parameter	Total *n* = 146	Female *n* = 80	Male *n* = 66
Age (years)	66 (53–74)	59 (51–72)	70 (61–77)
BMI ($kg \cdot m^{-2}$)	26.4 (23.8–29.5)	27.1 (23.3–31.8)	25.9 (24.5–28.7)
Underweight (BMI < 18.5)	3 (2.1)	2 (2.5)	1 (1.5)
Normal weight (BMI 18.5–24.9)	49 (33.6)	26 (32.5)	23 (34.8)
Overweight (BMI 25–29.9)	61 (41.8)	30 (37.5)	31 (47.0)
Obese (BMI > 30)	33 (22.6)	22 (27.5)	11 (16.7)
Diabetic wound	18 (12.3)	8 (10.0)	10 (15.2)
Non-diabetic wound	25 (17.1)	10 (12.5)	15 (22.7)
LRTI	103 (70.5)	62 (77.5)	41 (62.1)
Breast	36 (35.0)	36 (58.1)	–
Head and Neck	10 (9.7)	3 (4.8)	7 (17.1)
Urologic	27 (26.2)	5 (8.1)	22 (53.7)
Gynaecologic	8 (7.8)	8 (12.9)	–
Colorectal	10 (9.7)	4 (6.5)	6 (14.6)
Other	12 (11.7)	6 (9.7)	6 (14.6)

Median daily intake and RDA of all nutrients are reported in Tables 2 and 3 for females and males respectively. Also included is the number of patients that managed to reach their RDA. Most patients did not consume the amounts of calories, protein, vitamins A and D and zinc that were recommended for them. Conversely, around 70% of patients consumed vitamin C above the recommended level. More female patients achieved their RDA of calories and protein than male patients. For iron, more male patients achieved their RDA than female patients. There was no gender difference in intake of other nutrients. Furthermore, no differences in intake existed between age or BMI categories (findings not shown). Although there were statistically significant differences in vitamin A intake between the breast, head and neck and other LRTI categories, this was not considered clinically relevant. No other differences were found between LRTI subgroups.

Discussion

These data show that nutrition status is suboptimal in most patients referred for HBOT with wound healing problems or LRTI, despite the fact that more than 60% were overweight or obese based on BMI. This may not immediately impact general health but may prolong already delayed healing of wounds or LRTI. The results confirm those reported previously,[17] in which 30% of patients referred for HBOT were at risk for malnourishment.

Calories and protein are well-known factors that influence healing processes in the human body. The number of calories consumed per day is a measure of energy, which may be produced from different sources, such as carbohydrates, protein or fats.[6,20] Other than a source of energy, proteins are used for tissue growth, can be broken down to amino acids, and be used for synthesis of different proteins.[4,20] Some of these amino acids are essential, meaning they cannot be synthesised by the body but need to be derived from intake of food. The uptake and utilisation of proteins is partly dependent on certain vitamins and trace elements, such as zinc.[20] Vitamin A is derived from carotenoids in vegetables that enhances the early inflammatory phase and plays a role in the proliferation and re-epithelization phases.[4,20] Vitamin C is a cofactor for collagen synthesis, reduces reactive oxygen species (ROS) and facilitates uptake of iron, among many other roles.[20] Vitamin D plays an important role in the regulation of cell proliferation and differentiation, and in the modulation of immune system responses. There is growing evidence it might protect against cancer, prediabetes and metabolic syndrome.[21] The role of vitamin E is mostly that of antioxidant, reducing ROS and thereby limiting their deleterious effects on cell membranes, and facilitating wound healing.[4,20] Zinc is a cofactor for major enzyme systems, facilitates carbohydrate metabolism and plays a role in uptake of certain vitamins, such as vitamin A.[4,20] Finally, iron is used in heme in red blood cells to facilitate oxygen transport, and iron deficiency may lead to anaemia. It also plays a role in mitochondrial respiration and immune function.[22]

There is ample research on the role of nutrients in wound healing in the human body.[4] While LRTI may lead to non-healing wounds, the underlying mechanism differs from other chronic wounds such as diabetic ulcers. Exposure to ionising radiation produces ROS,[23] causing direct damage to DNA and proteins, which in turn leads to cell death.[24] This

Table 2

Female patients; intake and recommended amounts of nutrients, and numbers of patients achieving recommended daily allowance (RDA); † – RDA based on gender, age, and body composition; * RDA for age > 50 years = 10 µg·day^{-1}, > 70 years = 20 µg·day^{-1}

Parameter	Calories (kcal·day^{-1})		Protein (g·day^{-1})		Vitamin A (µg·day^{-1})		Vitamin C (mg·day^{-1})		Vitamin D (µg·day^{-1})		Vitamin E (mg·day^{-1})		Zinc (mg·day^{-1})		Iron (mg·day^{-1})	
	Intake	RDA†	Intake	RDA†	Intake	RDA	Intake	RDA	Intake	RDA*	Intake	RDA	Intake	RDA	Intake	RDA
25th percentile	1,350	1,978	64.0	80.0	335.0	800.0	67.5	75.0	1.7	10.0	6.0	8.7	7.9	10.0	7.1	10.8
Median	1,517	2,178	71.0	86.5	456.0		90.0		2.7		8.6		9.0		8.4	
75th percentile	1,808	2,350	80.0	95.8	558.8		115.5		3.7		11.2		10.0		10.2	
RDA achieved, *n* (%)	8 (10.0)		19 (23.8)		10 (12.8)		57 (71.3)		3 (3.8)		43 (53.8)		22 (27.5)		31 (38.8)	
Intake versus RDA	$P < 0.01$		$P < 0.01$		$P < 0.01$		$P < 0.01$		$P < 0.01$		$P = 0.339$		$P < 0.01$		$P = 0.005$	

Table 3

Male patients; intake and recommended amounts of nutrients, and numbers of patients achieving recommended daily allowance (RDA); † – RDA based on gender, age, and body composition; * RDA for age > 50 years = 10 µg·day^{-1}, > 70 years = 20 µg·day^{-1}

Parameter	Calories (kcal·day^{-1})		Protein (g·day^{-1})		Vitamin A (µg·day^{-1})		Vitamin C (mg·day^{-1})		Vitamin D (µg·day^{-1})		Vitamin E (mg·day^{-1})		Zinc (mg·day^{-1})		Iron (mg·day^{-1})	
	Intake	RDA†	Intake	RDA†	Intake	RDA	Intake	RDA	Intake	RDA*	Intake	RDA	Intake	RDA	Intake	RDA
25th percentile	1,491	2,300	65.5	93.0	365.0	900.0	72.5	75.0	2.3	10.5	6.9	10.0	7.8	10.0	7.6	9.0
Median	1,757	2,616	75.5	98.0	500.0		99.0		3.1		9.6		9.0		9.1	
75th percentile	1,992	2,814	90.0	105.3	674.0		123.8		4.0		10.9		10.8		10.6	
RDA achieved, *n* (%)	4 (6.1)		11 (16.7)		9 (13.6)		47 (71.2)		2 (3.0)		30 (45.5)		25 (37.9)		35 (53.0)	
Intake versus RDA	$P < 0.01$		$P < 0.01$		$P < 0.01$		$P < 0.01$		$P < 0.01$		$P = 0.129$		$P = 0.042$		$P = 0.776$	

also leads to loss of vascular tissue, causing hypoxia, and an overproduction of proinflammatory markers in the radiated tissue.[25] This hypoxic and proinflammatory state persists even after radiotherapy has ended,[26] and this continued inflammation is one of the most important factors causing LRTI.[27] HBOT may be used to improve tissue oxygenation and decrease circulating levels of proinflammatory cytokines.[28] However, it also leads to increased production of ROS. Antioxidants counter the production of ROS, and since exposure to hyperoxia during HBOT is usually limited, normal levels of antioxidants are usually adequate to counter the extra production caused by HBOT.[28] In the human body, certain enzymes act as antioxidants, but there are also non-enzymatic antioxidants, such as vitamins C and E, which can be acquired from food.[28] Eating food rich in these vitamins is recommended to maintain an adequate supply.[29] Since most people are aware of the beneficial properties of vitamin C, this is probably the reason why this is consumed more than average. While not an antioxidant itself, zinc can help maintain enzymatic antioxidants.[29]

STRENGTHS AND LIMITATIONS

The above list of nutrients is not exhaustive when it comes to wound healing or other regenerative processes. The vitamin B complex, magnesium and lipids are examples of other nutrients that are necessary in wound healing.[4,20] Therefore, this study gives an incomplete picture. However, it is argued that since most of the nutrients reported are already deficient in this population, this will not be different for other nutrients. Another limitation of this study is the method of data gathering. A single 24-hour period was used to measure average intake, and intake on other days and weekends may very well differ from this measurement. However, this short time-period was deliberately chosen, as keeping a week-long food diary was considered too impactful next to HBOT. Furthermore, questionnaires are inherently biased since patients may forget or choose to omit certain foods on it. For future studies specifically looking at nutrients, a food diary spanning multiple days may improve accuracy. Although biomarkers in urine or blood samples might also provide more information regarding a person's nutritional status, these biomarkers are not specific enough to detect malnutrition.[30] However, they can be used as an early signal of certain deficiencies.[30] A last limitation is the population itself, which is mostly composed of patients from a northern province of the Netherlands which is not as densely populated or urbanised as other parts. Although there are no specific data on this topic, dietary habits (and thereby nutrition) may differ from persons living in other parts of the Netherlands.

Aside from these limitations, the strength of the current study is the inclusion of a relatively large group of patients with different morbidities. This is likely a representative sample of the population that is usually referred for HBOT in the Netherlands. Furthermore, data on intake and personal RDA of a large number of nutrients are included, instead of only dietary habits or certain food groups. This provides an opportunity for direct supplementation and a more targeted approach.

Conclusions

The current study adds to the literature by highlighting that energy, protein and micronutrient intake may be suboptimal in patients attending HBOT. This may be because they cannot meet the increased metabolic demand presented by their affliction. The daily attendance for HBOT provides a unique opportunity to monitor and correct these deficiencies. However, a robust, prospective study with a large population is warranted to further examine the specific deficiencies that may be present in this population. These data should be further contextualised with treatment results and quality of life parameters. In the meantime, it is recommended to routinely screen patients referred for HBOT for nutritional intake and to supplement deficiencies when necessary.

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Inner ear barotrauma and inner ear decompression sickness: a systematic review on differential diagnostics

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Abstract
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Introduction: Inner ear barotrauma (IEBt) and inner ear decompression sickness (IEDCS) are the two dysbaric inner ear injuries associated with diving. Both conditions manifest as cochleovestibular symptoms, causing difficulties in differential diagnosis and possibly delaying (or leading to inappropriate) treatment.
Methods: This was a systematic review of IEBt and IEDCS cases aiming to define diving and clinical variables that help differentiate these conditions. The search strategy consisted of a preliminary search, followed by a systematic search covering three databases (PubMed, Medline, Scopus). Studies were included when published in English and adequately reporting one or more IEBt or IEDCS patients in diving. Concerns regarding missing and duplicate data were minimised by contacting original authors when necessary.
Results: In total, 25 studies with IEBt patients (n = 183) and 18 studies with IEDCS patients (n = 397) were included. Variables most useful in differentiating between IEBt and IEDCS were dive type (free diving versus scuba diving), dive gas (compressed air versus mixed gas), dive profile (mean depth 13 versus 43 metres of seawater), symptom onset (when descending versus when 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 findings consistent with middle ear barotrauma could not be reliably assessed in this context, being insufficiently reported in the IEDCS literature.
Conclusions: There are multiple useful variables to help distinguish IEBt from IEDCS. Symptoms of difficult middle ear equalisation or findings consistent with middle ear barotrauma require further study as means of distinguishing IEBt and IEDCS.

Introduction

Inner ear barotrauma (IEBt) and inner ear decompression sickness (IEDCS) are the two dysbaric inner ear injuries associated with diving. Whereas IEBt ultimately results from mechanical damage due to a pressure gradient between the middle and the inner ear, IEDCS results from bubble formation from dissolved gas either within the venous blood with subsequent arterialisation of bubbles and distribution to the labyrinthine artery, or within the membranous labyrinth itself.[1–5] Although the physiology and pathophysiology of IEBt and IEDCS are distinctly different, both conditions may manifest similarly, presenting as symptoms of cochlear (hearing loss, tinnitus) and/or vestibular (vertigo, nausea and vomiting) involvement.[1,2] These similarities can cause difficulties in differentiating between IEBt and IEDCS, possibly delaying (or leading to inappropriate) treatment.

These difficulties in differential diagnosis have been repeatedly discussed in previous literature,[1,2] and progress in differentiating between the two conditions has been made. Recently, based on a review of the relevant literature, the '*HOOYAH tool*' has been created to assist in the differentiating between IEBt and IEDCS. The tool consists of: 1) H – hard to clear; 2) O – onset of symptoms; 3) O – otoscopic exam; 4) Y – your dive profile; 5) A – additional symptoms and 6) H – hearing.[6] Although the tool is convenient, there are some limitations in the literature review on which it is based, including the inclusion of non-original studies (e.g., review articles), the inclusion of studies with neither IEBt nor IEDCS patients (e.g., studies examining otoacoustic emission testing or studies examining diving-related injuries in general), and the inclusion of patients with inner ear injuries resulting from non-diving related activities (e.g., inner ear injuries after head trauma). In addition, the review of the literature primarily focused

on IEBt, with significantly less attention given to IEDCS characteristics.[6]

Taking this into account, a systematic review with differently refined inclusion and exclusion criteria might provide additional information on the subject. Therefore, we carried out a systematic literature review to both elucidate and elaborate the differentiation between IEBt and IEDCS.

Methods

SEARCH STRATEGY

A preliminary literature search was carried out (search date 10 December 2020) in the PubMed database to identify all appropriate index terms and keywords for the final systematic literature search. This consisted of carrying out the preliminary search (index terms "*Diving*" AND "*Inner Ear*"), obtaining the preliminary search results (n = 228), and scanning these results (including the titles, abstracts, index terms, and key words of the 'similar articles' and 'cited articles') for all appropriate index terms and key words. Details of the preliminary search are presented in Appendix 1*.

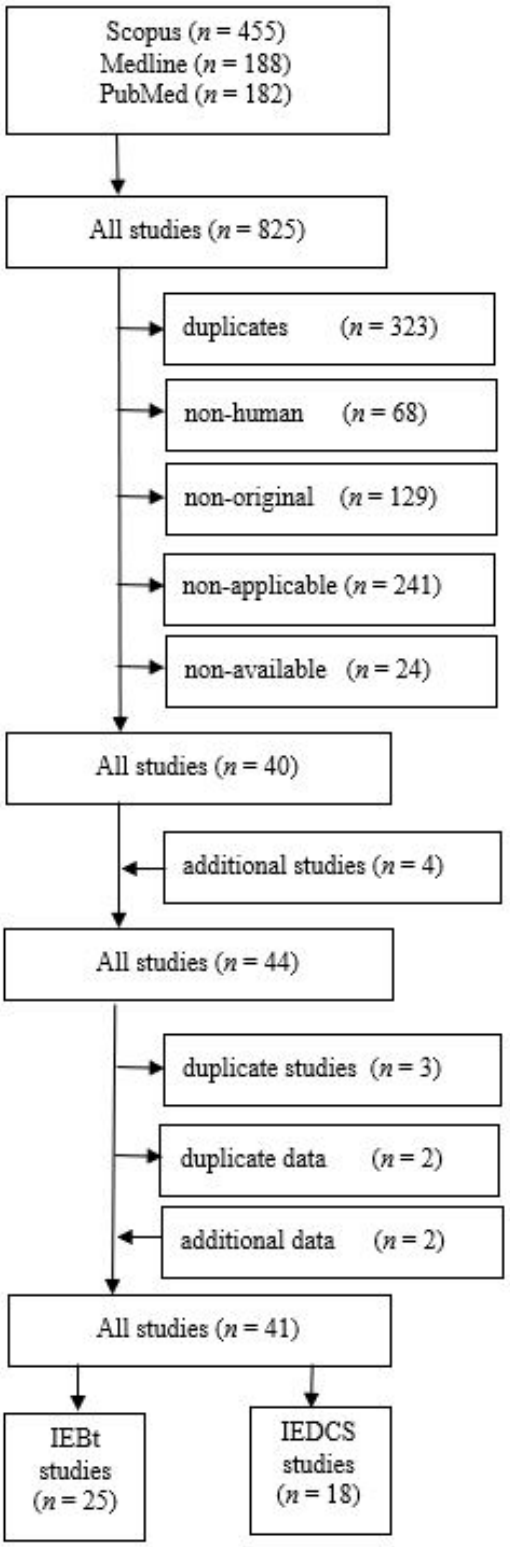
A systematic literature search was carried out (search date 10 December 2020, confirmatory search date 26 April 2021) in the PubMed, Medline, and Scopus databases, utilising all the index terms and key words identified in the preliminary search, and limiting the search to studies published in English. Details of the systematic search are presented in Appendix 2*.

STUDY SELECTION STRATEGY

A flow chart of the study selection process is presented in Figure 1. The studies were included when adequately reporting at least one or more IEBt or IEDCS patients in connection to diving activity, resulting in 40 included studies after the exclusion of all duplicate, non-human (i.e., animal or laboratory studies), non-original (i.e., review articles, commentaries, letters, and editorials), non-applicable (i.e., no applicable patients) and non-available studies (i.e., no full text available). Furthermore, four more additional studies, extracted from the references or the references of references, were included, resulting in 44 included studies.

Missing data were minimised by sending out data requests to all corresponding authors (n = 8) of studies with large ($n \geq 30$) sample sizes, resulting in additional information on two studies. Conversely, duplicate data were minimised by sending out data requests to the corresponding authors when necessary, resulting in the exclusion of three studies and the deletion of some of the patients in two studies. A list of the final 41 studies that were included is presented in Appendix 3*.

Figure 1
A flow chart of the study selection process; the total number of IEBt (n = 25) and IEDCS (n = 18) studies exceeds the total number of all studies (n = 41) as two studies included both IEBt and IEDCS patients. IEBt – inner ear barotrauma; IEDCS – inner ear decompression sickness



Footnote: * Appendix 1–3 are available on DHM Journal's website: https://www.dhmjournal.com/index.php/journals?id=288

DATA EXTRACTION STRATEGY

The data extracted from the final 41 studies included the study design, the study setting, and the number and general characteristics of all applicable patients. In addition, data on the relevant dive details were extracted, including the depth and duration of the incident dives, the breathing gases used, and the predisposing factors reported in connection to the incident dives; defined as middle ear equalisation difficulties in IEBt patients and any of the generally established DCS risk factors (i.e., consecutive days of diving, multiple dives per day, altitude exposure after the incident dive, uncontrolled ascent from the dive, dehydration or feeling cold during the dive, physical exertion during or after the dive, obesity) in IEDCS patients.

Data on the development, distribution and laterality of cochleovestibular symptoms were extracted, as well as data regarding symptoms attributable to other DCS manifestations. Data on the relevant findings, the treatment delay, the treatment(s) received and the outcomes at discharge and at follow-up were also extracted, when available. Finally, data regarding the continuation of diving activity were also extracted.

STATISTICAL ANALYSIS

All statistical analyses were performed using SPSS Statistics for Windows, version 27.0, released 2020 (IBM Corp, Armonk, NY, USA). A two-tailed *P*-value of < 0.05 was interpreted to indicate statistical significance.

The data are presented as numbers and percentages in the case of categorical variables (analysed using Fisher's exact test) and as means and ranges in the case of continuous variables (analysed using independent samples *t*-test). The Bonferroni correction was applied to adjust for multiple comparisons.

Results

OVERVIEW OF STUDIES

The 25 IEBt studies included 183 patients and were published between 1970 and 2016.[7–31] Approximately half (44.3%) of the patients were from studies published between 1970 and 2000, the other half (55.7%) from studies published between 2001 and 2016. All studies were case reports, case series or retrospective chart reviews; with the diagnosis in most cases verified via pure tone audiometry,

Table 1

Characteristics of incident dives; categorical data are presented as *n* (%) and continuous data are presented as mean (range). Triplets of superscripted numbers denote the numbers of observations: total, IEBt, IEDCS (e.g., data on dive type reported in 570 patients, 173 IEBt patients, 397 IEDCS patients). Each subscript letter denotes a subset of categories whose column proportions do not differ significantly from each other at the 0.05 level. IEBt – inner ear barotrauma; IEDCS – inner ear decompression sickness; min – minutes; msw – metres of sea water

Variable	All (*n* = 580)	IEBt (*n* = 183)	IEDCS (*n* = 397)	*P*
Dive type[570, 173, 397]				
Scuba diving	536 (94.0)	140 (80.9)$_a$	396 (99.7)$_b$	< 0.001
Free diving	32 (5.6)	31 (17.9)$_a$	1 (0.3)$_b$	
Other	2 (0.4)	2 (1.2)$_a$	0$_b$	
Dive gas[450, 59, 391]				
Air	362 (80.4)	59 (100.0)$_a$	303 (77.5)$_b$	< 0.001
Nitrox	23 (5.1)	0	23 (5.9)	
Heliox	22 (4.9)	0	22 (5.6)	
Trimix	43 (9.6)	0$_a$	43 (11.0)$_b$	
Dive depth (msw)[332, 51, 281]				
Mean	38.0	13.0	42.5	< 0.001
Range	1–200	1–49	9–200	
Dive duration (min)[179, 6, 173]				
Mean	38.7	25.2	39.2	0.012
Range	5–180	5–40	5–180	
Predisposing factors				
for IEBt[303, 137, 164]	124 (40.9)	118 (86.1)$_a$	6 (3.7)$_b$	< 0.001
for IEDCS[534, 137, 397]	211 (39.5)	0$_a$	211 (53.1)$_b$	

Table 2

Symptom and findings after incident dives; categorical data are presented as *n* (%) and continuous data are presented as mean (range). Triplets of superscripted numbers denote numbers of observations: total, IEBt, IEDCS (e.g., data on symptom onset reported in 489 patients, 92 IEBt patients, 397 IEDCS patients). Each subscript letter denotes a subset of categories whose column proportions do not differ significantly from each other at the 0.05 level. HL – hearing loss; IEBt – inner ear barotrauma; IEDCS – inner ear decompression sickness; MEBt – middle ear barotrauma

Variable	All (*n* = 580)	IEBt (*n* = 183)	IEDCS (*n* = 397)	*P*
Onset[489, 92, 397]				
When descending	17 (3.5)	17 (18.5)$_a$	0$_b$	< 0.001
When ascending	33 (6.7)	3 (3.3)	30 (7.6)	
When surfacing	57 (11.7)	30 (32.6)$_a$	27 (6.8)$_b$	
After surfacing	382 (78.1)	42 (45.7)$_a$	340 (85.6)$_b$	
Onset delay (hours)[347, 7, 340]				
Mean	0.9	25	0.4	0.010
Range	0–72	1.5–72	0–16	
Inner ear symptoms[580, 183, 397]				
Cochlear	303 (52.2)	172 (94.0)	131 (33.0)	< 0.001
Vestibular	446 (76.9)	82 (44.8)	364 (91.7)	< 0.001
Cochlear symptoms[465, 183, 282]				
Hearing loss	232 (49.9)	152 (83.1)	80 (28.4)	< 0.001
Tinnitus	169 (36.3)	123 (67.6)	46 (16.3)	< 0.001
Vestibular symptoms				
Vertigo[582, 183, 397]	446 (76.9)	82 (44.8)	364 (91.7)	< 0.001
Nausea and vomiting[313, 98, 215]	177 (56.5)	13 (13.3)	164 (76.3)	< 0.001
Other DCS symptoms[582, 183, 397]				
No	478 (82.1)	181 (98.9)	297 (74.8)	< 0.001
Yes	102 (17.9)	2 (1.1)	100 (25.2)	
Laterality of symptoms[382, 86, 296]				
Right-sided	230 (60.2)	35 (40.7)$_a$	195 (65.9)$_b$	< 0.001
Left-sided	147 (38.5)	48 (55.8)$_a$	99 (33.4)$_b$	
Both-sided	5 (1.3)	3 (3.5)$_a$	2 (0.7)$_b$	
Otological findings				
Sensorineural HL[505, 179, 326]	230 (45.5)	155 (86.6)	75 (23.0)	< 0.001
Nystagmus[322, 84, 238]	182 (56.5)	24 (28.6)	158 (66.4)	< 0.001
MEBt[267, 118, 149]	65 (24.3)	57 (48.3)	8 (5.4)	< 0.001
Other findings				
Right-to-left shunt[255, 3, 252]	176 (69.0)	0	176 (69.8)	0.029

electronystagmography and/or surgical exploration of the tympanic cavity (i.e., exploratory tympanotomy).

The 18 IEDCS studies included 397 patients and were published between 1976 and 2019.[12,18,32–47] A minority of the patients (9.1%) were from studies published between 1976 and 2000, and the majority (90.9%) from studies published between 2001 and 2019. All studies were case reports, case series or retrospective chart reviews; the diagnosis in many cases verified via pure tone audiometry and/or electronystagmography. Quantitative synthesis of the studies is presented in Tables 1, 2 and 3, and described below.

COMPARISON OF INCIDENT DIVES

Characteristics of incident dives are presented in Table 1. A minority of IEBt cases appeared after free diving (17.9%), while the majority of both IEBt (80.9%) and IEDCS (99.7%) cases appeared in connection to scuba diving. The breathing gas used during the scuba dives was compressed air in all (100.0%) dives preceding IEBt and in three quarters (77.5%) of the dives preceding IEDCS ($P < 0.001$). The remaining quarter of IEDCS cases appeared after the use of nitrox (5.9%), heliox (5.6%), or trimix (11.0%). The mean depth and duration of the dives were 13 metres of

Table 3

Treatment protocols and outcomes after incident dives; categorical data are presented as *n* (%) and continuous data are presented as mean (range). Triplets of superscripted numbers denote numbers of observations: total, IEBt, IEDCS (e.g., data on treatment delay reported in 402 patients, 72 IEBt patients, 331 IEDCS patients). Each subscript letter denotes a subset of categories whose column proportions do not differ significantly from each other at the 0.05 level. HBOT – hyperbaric oxygen treatment; IEBt – inner ear barotrauma; IEDCS – inner ear decompression sickness

Variable	All (*n* = 580)	IEBt (*n* = 183)	IEDCS (*n* = 397)	*P*
Delay to treatment (hours)[403, 72, 331]				
Mean	39.1	189	6.6	< 0.001
Range	0–1176	0–1176	0–336	
Modality of treatment[558, 161, 397]				
Conservative	127 (22.8)	108 (67.1)$_a$	19 (4.8)$_b$	< 0.001
Surgical	53 (9.5)	53 (32.9)$_a$	0 (0.0)$_b$	
HBOT	385 (69.0)	7 (4.4)$_a$	378 (95.2)$_b$	
Number of HBOT[342, 7, 335]				
Mean	2.9	1.0	2.9	< 0.001
Range	1–26	1–1	1–26	
Full recovery				
At discharge[387, 80, 307]	125 (32.3)	13 (16.2)	112 (36.5)	< 0.001
At follow-up[323, 83, 240]	174 (53.9)	27 (32.5)	147 (61.3)	< 0.001
Continuation of diving[157, 31, 126]				
Yes	106 (67.5)	27 (87.1)	79 (62.7)	0.010
No	51 (32.5)	4 (12.9)	47 (37.3)	

seawater (msw) and 25-min preceding IEBt and 43 msw and 39-min preceding IEDCS ($P < 0.001$ for depth and $P = 0.012$ for duration, respectively).

While predisposing factors for DCS were documented in approximately half (53.1%) of the IEDCS cases, a predisposing factor for IEBt (i.e., middle ear equalisation difficulties) was documented in the vast majority (86.1%) of IEBt patients.

COMPARISON OF SYMPTOMS AND FINDINGS

Symptoms and findings after the incident dives are presented in Table 2. In IEBt patients, the symptoms appeared in all stages of the dive; either when descending (18.5%), when ascending (3.3%), when surfacing (32.6%) or after surfacing (45.7%). Conversely, the symptoms of IEDCS appeared when ascending (7.6%) or when surfacing (6.8%) in only a minority of cases, developing in most cases shortly after reaching the surface (85.6%). The mean delay to onset of symptoms was 0.4 h in IEDCS patients. Among 42 IEBt patients with onset after surfacing, latency was only reported for seven and the mean 25 hour latency may be anomalous.

The symptoms of IEBt patients were cochlear in almost all cases: a total of 83.1% reported hearing loss and 67.6% reported tinnitus, whereas only 44.8% reported vertigo. In contrast, the symptoms of IEDCS patients were predominantly vestibular, with 91.7% reporting vertigo while only 28.4% reported hearing loss and only 16.3% reported tinnitus. Symptoms of other DCI manifestations affected 25.2% of IEDCS and 1.1% of IEBt patients ($P < 0.001$).

The symptoms of IEBt patients had a slight tendency for left-sided lateralisation (55.8% versus 40.7%, $P = 0.047$) while the symptoms of IEDCS patients were predominantly right-sided (65.9% versus 33.4%, $P < 0.001$). Whereas approximately half (48.3%) of the IEBt patients presented with middle ear barotrauma, this was the case in only a few (5.4%) IEDCS patients ($P < 0.001$).

COMPARISON OF TREATMENT PROTOCOLS AND OUTCOMES

Treatment protocols and outcomes are presented in Table 3. The mean delay to treatment was 189 h in IEBt and 7 h in IEDCS patients ($P < 0.001$). Approximately two thirds (67.1%) of IEBt patients were treated conservatively (bed rest with the head elevated, pharmacological management, daily audiometric monitoring) and the remaining one third (32.9%) underwent surgery (exploratory tympanotomy or an injected intratympanic blood patch). Seven IEBt patients (4.4%) were recompressed before the appropriate treatment was instituted. This resulted in no worsening of symptoms in six patients (no data in one patient). Almost all IEDCS patients (95.2%) were recompressed (mean number of recompressions 2.9) but a small minority (4.8%) were not; the reason for this was unspecified in most (14 of 19) cases.

Although infrequently reported, recovery from the inner ear insult seemed less frequent in IEBt than in IEDCS patients; complete recovery was less frequent both at discharge (16.2% versus 36.5%, $P < 0.001$) and at follow up (32.5% versus 61.3%, $P < 0.001$) after IEBt. In contrast, a return to diving was reported more often after IEBt than after IEDCS (87.1% versus 62.7%, $P = 0.010$).

Discussion

AGREEMENT WITH PREVIOUS LITERATURE

This is the first systematic literature review specifically examining the differential diagnosis between IEBt and IEDCS in the context of diving, and the results largely aligned with previous literature. The IEBt cases appeared after both free and scuba diving, while conversely, the IEDCS cases appeared almost exclusively (99.7%) after scuba diving. Furthermore, while all scuba dives (100%) preceding IEBt were carried out using compressed air as the breathing gas, some IEDCS cases appeared after the use of mixed breathing gases (22.5%). This may be utilised in differentiating between IEBt and IEDCS in the future.

The depth and duration of the dives were indeed markedly different between IEBt and IEDCS patients: the mean depth and duration of the incident dive was 13 msw and 25 min preceding IEBt and 42 msw and 39 min preceding IEDCS. This being said, these data were not reported in a large proportion of patients (data on dive depth missing in 132 IEBt and 116 IEDCS patients; data on dive duration missing in 177 IEBt and 224 IEDCS patients), rendering the findings less reliable. Overall, the data suggest that knowledge of the dive profile can be utilised to guide differential diagnosis between IEBt and IEDCS (as proposed by the HOOYAH tool).[6]

Concerning the symptoms, those of the IEBt patients potentially appeared in all stages of the dive. In contrast, the symptoms of IEDCS patients never appeared when descending and appeared in only a minority of cases (i.e., in connection to technical diving with mixed breathing gases) when ascending or immediately when reaching the surface. These findings suggest that in some cases, the onset of symptoms can be a determining factor in differentiating IEBt from IEDCS.

The symptoms were predominantly cochlear in IEBt patients (94.0% with cochlear and 44.8% with vestibular symptoms) and predominantly vestibular in IEDCS patients (91.7% with vestibular and 33.0% with cochlear symptoms). These findings are at best suggestive of the underlying condition when differentiating between IEBt and IEDCS, whereas the presence of other DCI manifestations seem to strongly point towards IEDCS. Overall, the findings suggest that both the onset and distribution (cochlear versus vestibular, isolated versus non-isolated) of the symptoms can contribute to differentiating between IEBt and IEDCS (as proposed by the HOOYAH tool).[6] A summary of the differential diagnostic process between IEBt and IEDCS is presented in Figure 2.

It is worth noting that despite the difficulties in differential diagnosis, the great majority of patients appeared to be diagnosed correctly: only seven IEBt patients (4.4%) were misdiagnosed and recompressed before receiving the appropriate treatment, and no worsening of symptoms was reported in six of these seven patients (no data in one patient). Correspondingly, a total of three IEDCS patients (0.8%) were not recompressed due to diagnostic difficulties, while another two (0.5%) were not recompressed due to the long treatment delay (no data on the reason in 14 patients). This suggests that although most patients are diagnosed correctly, there is a chance of a misdiagnosis in both directions.

DISAGREEMENTS WITH PREVIOUS LITERATURE

Although most of the findings aligned with the guidelines proposed by the HOOYAH tool, this was not the case regarding middle ear equalisation difficulties or middle ear barotrauma. While our results certainly indicate that middle ear equalisation difficulties are a predisposing factor for IEBt but not for IEDCS (86.1% versus 3.7%), a look at the relevant literature suggests that the matter is more complicated.

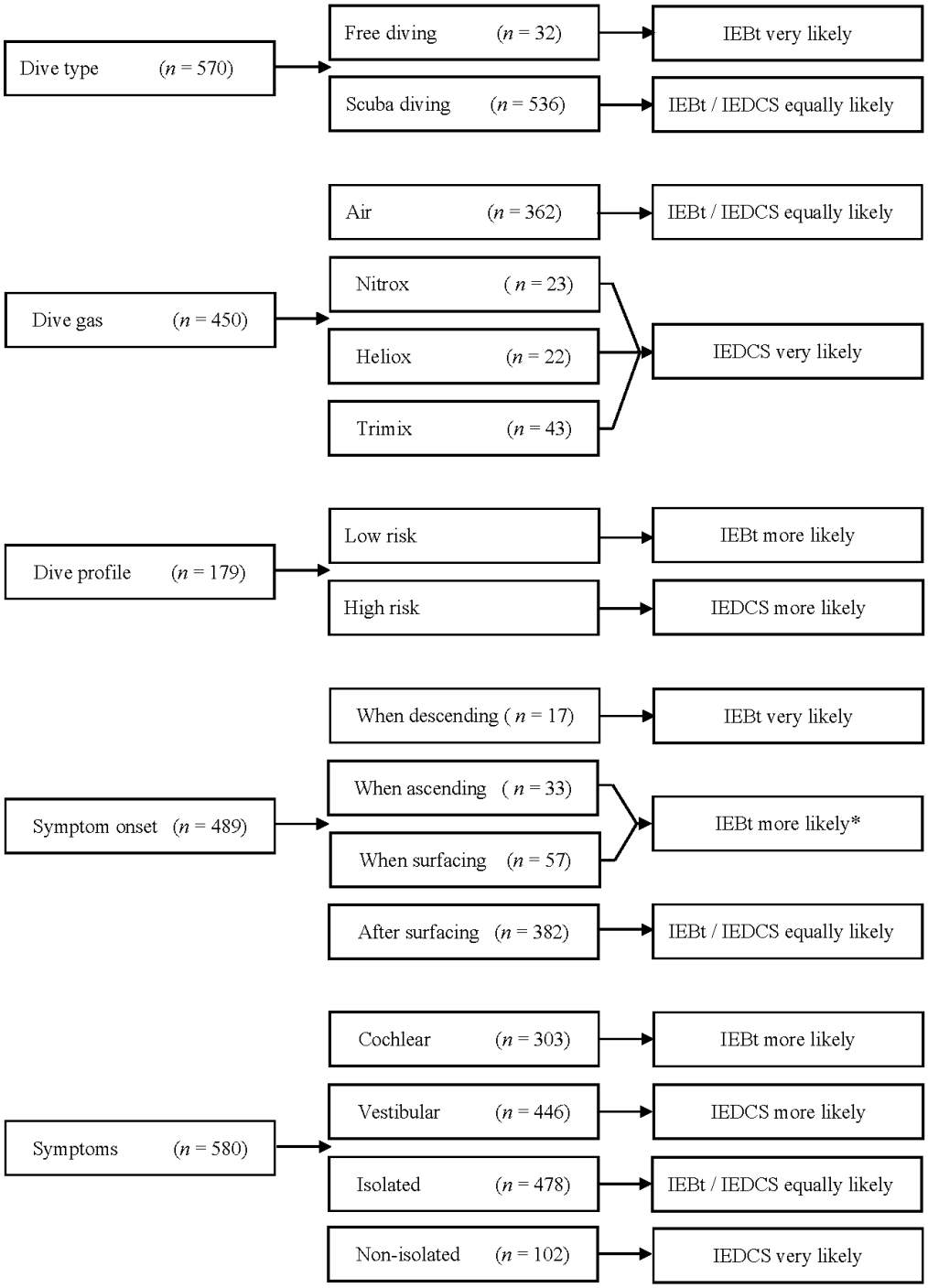
While the connection from middle ear equalisation difficulties and forceful Valsalva manoeuvres to IEBt has been thoroughly discussed and documented in the relevant literature,[1,2] such a connection to IEDCS has never been examined or established. However, forceful Valsalva manoeuvres provoke intrathoracic pressure changes that could promote the passage of venous inert gas bubbles through a (moderate to large) right-to-left shunt.[48,49] Moreover, the connection between right-to-left shunts and IEDCS has been thoroughly established in several publications.[41,43,50–52] Therefore it should be appreciated that if there are inert gas bubbles present in the venous blood (e.g., at the start of a repetitive dive) middle ear equalisation difficulties are not just a predisposing factor for IEBt but could, under such circumstances, predispose to IEDCS as well.

Taking this into account, there is a shortage of data in the relevant literature connecting middle ear equalisation difficulties with IEDCS. This can be explained by a multitude of factors.

Firstly, patients with middle ear equalisation difficulties (or findings consistent with middle ear barotrauma) are routinely excluded from IEDCS studies. Although the practice is in itself certainly justified (so that no IEBt patients are inadvertently included in the studies), it does lead to a slight distortion of the literature, and IEDCS patients with middle ear equalisation difficulties end up being insufficiently acknowledged in the literature. Importantly, considering that patients with middle ear equalisation difficulties (or findings consistent with middle ear barotrauma) were excluded from

Figure 2

A summary of the differential diagnostic process between IEBt and IEDCS; the phrase '*more likely*' is adopted in the case of a substantial polarisation between IEBt and IEDCS patients. The phrase '*very likely*' is adopted in the case of a ≈ 99% polarisation between IEBt and IEDCS patients. * IEBt more likely but IEDCS more likely when scuba diving with mixed breathing gases. IEBt – inner ear barotrauma; IEDCS – inner ear decompression sickness



the IEDCS studies from which the HOOYAH tool originates from, such findings cannot be the basis for differentiating between IEBt and IEDCS in the algorithm.

Secondly, even when patients with middle ear equalisation difficulties are not excluded from the IEDCS studies, the presence or absence of middle ear equalisation difficulties in IEDCS patients is rarely reported in the original publications (for example, all the patients with middle ear equalisation difficulties in this study were identified by contacting the authors and asking directly about any such difficulties). This would suggest that the true number of IEDCS patients

with middle ear equalisation difficulties is greater than that described in the relevant literature.

Thirdly, even when the patients are not excluded, and even when the middle ear equalisation difficulties are documented, the patients themselves tend to insufficiently report their possible difficulties in middle ear equalisation.[25] That middle ear equalisation difficulties will often be missed without a careful and complete interrogation of patients has been documented previously.[25] This means that even when the patients are not excluded, and even when the middle ear equalisation difficulties are systematically recorded, the patients themselves have to be elaborately questioned to reveal any difficulties with middle ear equalisation during the incident dive.

Overall, this means that contrary to the current guidelines provided by the HOOYAH tool, symptoms of poor middle ear equalisation or findings consistent with middle ear barotrauma may not be reliable in the differentiating between IEBt and IEDCS in all circumstances, for example, when there are venous inert gas bubbles present at the start of a repetitive dive. Although it is possible that these could be useful tools in differentiating between the conditions, such inferences cannot be made from the current literature: therefore these variables should be interpreted with caution when trying to differentiate between IEBt and IEDCS.

STRENGTHS AND LIMITATIONS

The main strength of the study is its overall scope. The results can be considered fairly representative of both conditions as the systematic literature review included all original publications with any IEBt or IEDCS patients, including both small case reports and case series as well as retrospective chart reviews with larger sample sizes (with additional data requests sent to the authors of large studies). Although this can be argued to make the study the most comprehensive review of inner ear disorders in diving published to date, it still remains subject to several limitations.

Firstly, the missing data resulting from the exclusion of studies based on language (studies not published in English) and availability (studies with no full text available) limits the generalisability of our findings. Secondly, the missing patient data resulting from unsystematic reporting (e.g., data on dive depth and duration in IEBt patients, see above) limits the reliability of some of the findings. This could (and should) be mitigated by a more systematic approach to data collection and reporting in the future, whenever possible.

Conclusion

This is the first systematic literature review specifically examining the differential diagnostics between IEBt and IEDCS in the context of diving. The data suggest that the variables most useful in differentiating between IEBt and IEDCS are dive type (freediving versus scuba diving), dive gas (compressed air versus mixed breathing gases), dive profile (mean depth 13 msw versus 43 msw), and the onset (when descending versus when ascending or surfacing) and distribution of cochleovestibular symptoms (vestibular versus cochlear, isolated versus non-isolated). Symptoms of poor middle ear equalisation or findings consistent with middle ear barotrauma could not be reliably assessed as a means of differentiating between IEBt and IEDCS, being insufficiently reported in the relevant literature. These variables should be interpreted with caution when differentiating between IEBt and IEDCS, and future research should focus on examining them in both IEBt and IEDCS patients before a guideline regarding their utilisation can be formulated.

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Appendix 1
Details of the preliminary literature search
Appendix 2
Details of the systematic literature search
Appendix 3
Publications included in the final systematic review

Microcirculation and tissue oxygenation in the head and limbs during hyperbaric oxygen treatment

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Key words
Cardiovascular; Laser Doppler; Hyperoxia; Patient monitoring; Peripheral blood flow; Transcutaneous oximetry

Abstract
Yamamoto N, Takada R, Maeda T, Yoshii T, Okawa A, Yagishita K. Microcirculation and tissue oxygenation in the head and limbs during hyperbaric oxygen treatment. Diving and Hyperbaric Medicine. 2021 December 20;51(4):338–344. doi: 10.28920/dhm51.4.338-344. PMID: 34897598.)

Introduction: Hyperbaric oxygen (HBO) exposure for 10–15 min has been shown to reduce peripheral blood flow due to vasoconstriction. However, the relationship between decreased peripheral blood flow and the therapeutic effects of HBO treatment on peripheral circulatory disorders remain unknown. Longer exposures have been reported to have vasodilatory effects and increase peripheral blood flow. This study investigated the effect of HBO treatment on blood flow and transcutaneous oxygen pressure ($TcPO_2$).

Methods: Twenty healthy volunteers aged 20–65 years (nine males) participated in this study. All participants breathed oxygen for 60 min at 253.3 kPa. Peripheral blood flow using laser Doppler flowmetry and $TcPO_2$ on the ear, hand, and foot were continuously measured from pre-HBO exposure to 10 min post-exposure.

Results: Peripheral blood flow in each body part decreased by 7–23% at the beginning of the HBO exposure, followed by a slow increase. Post-exposure, peripheral blood flow increased 4–76% in each body part. $TcPO_2$ increased by 840–1,513% during the exposure period, and remained elevated for at least 10 min after the exposure.

Conclusions: The findings of the current study suggest vasoconstriction during HBO treatment is transient, and even when present does not inhibit the development of increased tissue oxygen partial pressure. These findings are relevant to studies investigating changes in peripheral blood flow during HBO treatment in patients with circulatory disorders.

Introduction

Hyperbaric oxygen (HBO) treatment is an effective treatment for diseases such as decompression sickness, radiation tissue injury, and selected non-healing wounds.[1–4] Standard HBO treatment is generally performed for 90–120 min, and involves oxygen (O_2) inhalation at 202.6–253.3 kPa,[5] often with intermittent air breaks. While the therapeutic effect of HBO is thought to be associated with an increase in dissolved O_2 in the blood and changes in blood flow,[1–6] the exact nature of the relationship between oxygen partial pressure (PO_2) and flow remains unknown. Therefore, it is important to clarify the effects of HBO treatment on blood O_2 level and peripheral blood flow.

Peripheral blood flow decreased and transcutaneous partial pressure of oxygen ($TcPO_2$) increased when healthy humans were exposed to HBO for a short duration.[7–10] Increased dissolved O_2 concentrations in the plasma during HBO treatment contributes to the therapeutic effects of HBO treatment.[11] Increased TcO_2 has been reported at the beginning of treatment, followed by an increase in the levels of nitric oxide (NO) and superoxide (O_2^-) that are produced by endothelial cells.[6,10] Subsequently, O_2^- reacts with NO to generate peroxynitrite ($ONOO^-$) between the endothelium and vascular smooth muscle cells, leading to vasoconstriction as the vasodilatory effects of NO were antagonised.[10] This reaction has been considered the reason for decreased peripheral blood flow during single HBO exposures of short duration.

During HBO exposure, extracellular superoxide dismutase (SOD) is gradually activated between the endothelium and vascular smooth muscle cells. This scavenges O_2^- thus ameliorating antagonism of NO.[6,10] Thus, an increase in peripheral blood flow after an initial reduction may occurs due to restoration of the vasodilatory effect of NO during a longer HBO exposure. However, previous reports have only measured peripheral blood flow during short HBO exposures (10–15 min).[8,9] Therefore, it was hypothesised that a longer HBO exposure (e.g., 60 min), as in typical HBO treatments, would increase peripheral blood flow. This

study aimed to investigate the changes in peripheral blood flow and $TcPO_2$ associated with a longer HBO exposure in healthy participants.

Methods

PARTICIPANTS

Ethical approval for this study was granted by the Medical Research Ethics Committee of Tokyo Medical and Dental University (M 2,000–1,814-01). Participants between the ages of 20–65 years were recruited from healthy volunteers at our institution. Informed consent was obtained from all participants who met the inclusion criteria. All participants were lifetime non-smokers and were able to equalise their ears. Exclusion criteria included a history of peripheral circulatory disorders, pneumothorax, convulsions, claustrophobia and current pregnancy. The study was undertaken in accordance with the ethical standards of the Declaration of Helsinki.

HBO PROTOCOL

Each participant received a single HBO exposure session in a multi-place HBO chamber (NHC-412-A, Nakamura Iron Works, Tokyo, Japan). The HBO protocol involved 60 min of O_2 breathing at 253.3 kPa with two 5-min air breaks (Figure 1).

During HBO exposure the participants sat on a chair with their hands and feet on the arm- and foot-rests, respectively. They were instructed to stay relaxed, breathe normally, and notify the staff if they faced any problems during the exposure. Oxygen breathing was via a non-rebreather oxygen mask supplied at 20 $L \cdot min^{-1}$ flow.

MEASUREMENTS

Peripheral blood flow ($mL \cdot min^{-1}$) was measured using laser Doppler flowmetry (LDF) (MBF-11A, Pioneer Corp., Kawasaki, Japan). LDF is a non-invasive method used for real-time assessment of skin perfusion by a fibre optic probe.[12,13] The principle of LDF measurement is that when the tissue is irradiated by a laser, peripheral blood flow can be estimated using the spread of the Doppler-shifted frequency generated by the interference between light backscattered from static tissue and light backscattered from red blood cells flowing in the capillaries.[13–15] The LDF device used in this study was small (105 mm × 62 mm × 25 mm), lightweight (144 g), cordless, and included a wireless transfer function.[14] Additionally, this device is designed to generate minimal artefact in a dynamic environment, including postural changes.[14] The LDF system was confirmed to work normally under HBO exposure by using it with an infusion pump system filled with milk instead of blood.[15]

$TcPO_2$ (mmHg) was measured using a transcutaneous oximeter (TCM400, Radiometer Pacific TCM400, Radiometer Pacific, Copenhagen, Denmark). Transcutaneous oximetry measurement is non-invasive; it is performed through a heated sensor on the skin.[16,17] It is widely used to assess tissue hypoxia and demonstrate responsiveness of the peri-wound tissue to O_2 breathing.[16–18]

Although it is still controversial and not proven, it has been reported that HBO treatment may be effective for diseases such as cerebral infarction as it causes an increase in $TcPO_2$ and cerebral blood flow.[19,20] Several studies have also reported that the measurement of blood flow using LDF in the ear may reflect the values for cerebral blood flow.[13,14] Thus, blood flow and $TcPO_2$ on the ear were measured based on these reports.

Figure 1

HBO protocol; a single HBO exposure for 60 min with oxygen (O_2) inhalation at 253.3 kPa and two air breaks was performed. Pre – pre-phase; UP – under pressure; O_2-1 – first phase of 253.3 kPa O_2 inhalation; Air-1 – first phase of 253.3 kPa air inhalation; O_2-2 – second phase of 253.3 kPa O_2 inhalation; Air-2 – second phase of 253.3 kPa air inhalation; O_2-3 – third phase of 253.3 kPa O_2 inhalation; D – decompression; post – post-phase. ∗ 1–7 – denote points of skin and ambient temperature measurements

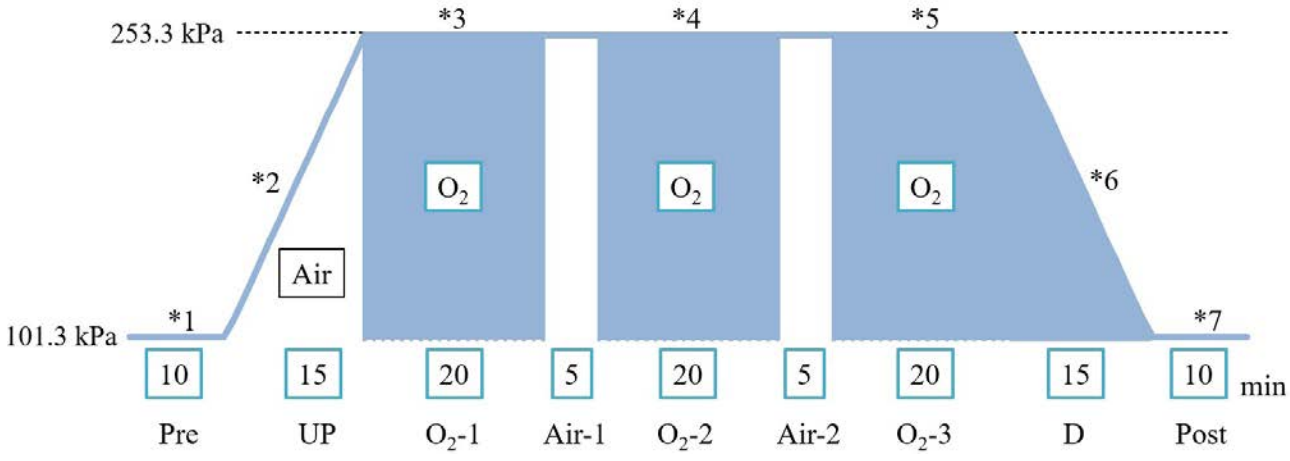


Figure 2
Position of sensors on the earlobe, hand and foot; laser Doppler flowmetry sensor placement: ear sensor – right earlobe; finger sensor – palmar aspect of right index finger; toe sensor – palmar aspect of right first toe. Transcutaneous oximetry sensor placement: ear sensor – front of right ear; hand sensor – dorsum of the right first interdigital space; foot sensor – dorsum of the right foot between the first and second metatarsal heads. Skin temperature sensor placement: ear sensor – front of ear; finger sensor – palmar aspect of right middle finger; toe sensor – palmar aspect of right first toe

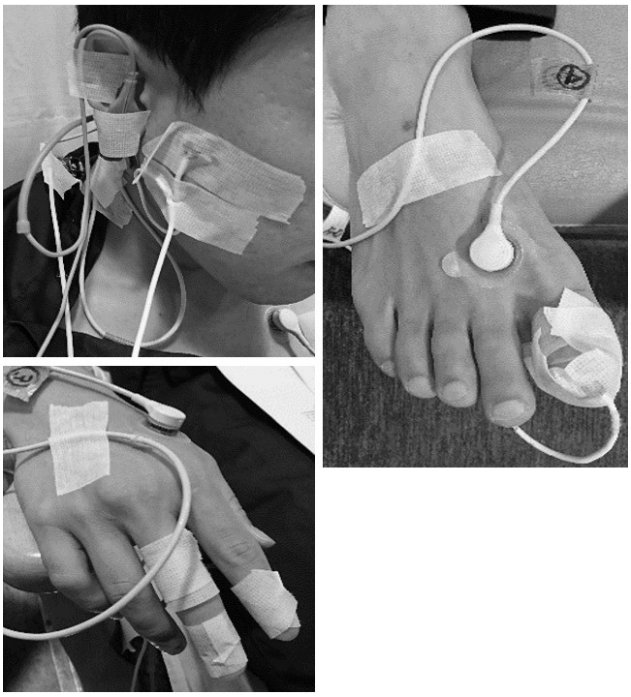
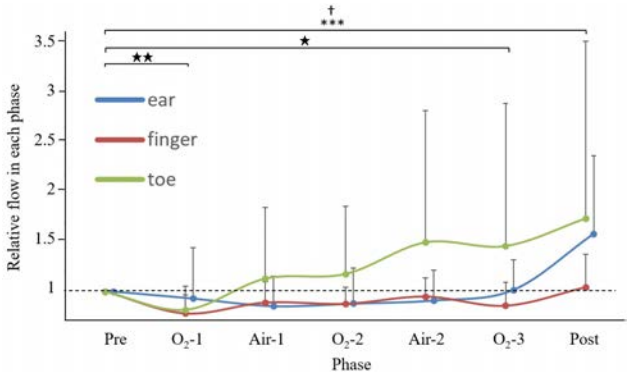


Figure 3
Changes in peripheral blood flow relative to the Pre phase in the ear, finger, and toe (n = 20 subjects); *** difference in the peripheral blood flow in the ear, $P < 0.001$; difference in the peripheral blood flow in the finger, ★★ $P < 0.01$, ★ $P < 0.05$; † difference in the peripheral blood flow in the toe, $P < 0.05$



It was possible that the change in peripheral blood flow and $TcPO_2$ was just a consequence of using the instrument itself rather than an effect of HBO treatment. To assess this, the pulse rate was measured at the palmar aspect of the right index finger simultaneously, and ambient and skin temperatures were measured at the ear, hand, and foot.

The LDF, $TcPO_2$ and temperature sensors were placed on the ear, hand, and feet of the participants (Figure 2). A co-author (TM) attached all the sensors to all participants. Each measurement was continuously recorded between pre-HBO exposure and 10 min post-exposure. Peripheral blood flow, $TcPO_2$, and pulse rate were recorded continuously during each phase (designated in Figure 1) except at one min before and after each phase to exclude the influence of attachment or detachment of participants' masks for air breaks. These measurements were averaged to obtain a single value for each phase. The ambient and skin temperatures were recorded once for each phase (time points indicated in Figure 1). In addition, the changes in peripheral blood flow and $TcPO_2$ were assessed using relative-value-based comparisons (the value of pre-phase was set as the baseline).

ANALYSIS

Sample size was calculated based on an effect size (f) of 0.25, which was calculated as a 25% decrease in peripheral blood flow during HBO exposure compared to baseline, which was statistically significant, and correlation among levels was 0.5 of the repeated one-way analysis of variance (ANOVA) based on the previous reports.[7,8] The power (1-β) was 80%, type I error rate (α) was 5%, and dropout rate was 10%. The results of this calculation indicated that 22 participants were required for this study. All data were analysed using EZR (Saitama Medical Center, Saitama, Japan).[21] Data were analysed for normality using the Shapiro-Wilk test and for homogeneity using the Levene's test. Statistical analyses were performed using repeated one-way ANOVA followed by Dunnett's post-hoc test. The correlation between skin temperature and peripheral blood flow was evaluated using the Spearman's rank correlation coefficient. The significance level for statistical analysis was set at $P = 0.05$.

Table 1
Subject demographic and baseline details; data are mean (SD). BMI – body mass index

Group	Age (year)	Height (cm)	Weight (kg)	BMI (kg·m^{-2})
All subjects	30.7 (5.7)	165.9 (7.2)	60.0 (11.0)	21.7 (2.5)
Male (n = 9)	31.3 (3.5)	170.2 (4.0)	65.5 (9.5)	22.6 (2.4)
Female (n = 11)	29.9 (7.5)	158.6 (2.3)	50.5 (4.4)	20.1 (2.1)

Table 2
Peripheral blood flow (ml·min^{-1}), mean (SD), in the ear, finger, and toe during HBO exposure (n = 20 subjects); * $P < 0.05$, ** $P < 0.01$

Site	Pre	O_2-1	Air-1	O_2-2	Air-2	O_2-3	Post
Ear	23.0 (15.5)	21.0 (10.8)	19.2 (15.9)	18.0 (12.5)	19.0 (12.5)	21.7 (12.5)	32.1 (20.2) *
Finger	67.8 (20.0)	54.5 (19.9) **	61.9 (23.1)	60.3 (19.0)	64.4 (18.0)	59.7 (22.6)	70.3 (22.0)
Toe	48.2 (30.2)	36.6 (19.4)	46.0 (22.8)	45.4 (19.3)	54.0 (25.1)	49.3 (22.6)	56.3 (28.7)

Table 3
$TcPO_2$ (mmHg), mean (SD), in the ear, finger, and toe during HBO exposure (n = 20 subjects); * $P < 0.001$

Site	Pre	O_2-1	Air-1	O_2-2	Air-2	O_2-3	Post
Ear	62.5 (26.2)	814.8 (159.1) *	252.4 (79.4) *	840.8 (157.8) *	293.9 (98.0) *	857.0 (199.1) *	232.1 (182.7) *
Finger	80.0 (12.0)	824.0 (219.1) *	325.1 (64.0) *	852.3 (224.5) *	336.6 (37.2) *	886.5 (202.2) *	164.0 (67.3) *
Toe	82.0 (10.3)	759.3 (147.2) *	306.4 (85.3) *	794.9 (157.9) *	312.9 (45.1) *	813.1 (172.2) *	162.3 (58.0) *

Results

Twenty-two participants (10 males and 12 females) were recruited for the study. Two participants were excluded due to missing data; therefore, 20 sets of data were analysed (nine males and 11 females). The participants' demographic data are shown in Table 1.

Peripheral blood flow measurements are shown in Table 2 and Figure 3. For the ear, the average peripheral blood flow decreased during the O_2-1 and O_2-2 phases relative to pre-phase values, and increased again during the O_2-3 and post-phase. For the finger, the relative value decreased during the initial oxygen exposure, increased during the O_2-2 phase, and returned to the baseline in the post-phase. For the toe, the relative value decreased during the O_2-1 phase and increased during the O_2-2, O_2-3, and post-phase.

$TcPO_2$ measurements are shown in Table 3 and Figure 4. The average value of $TcPO_2$ relative to pre-phase measurements significantly increased in all sites during all oxygen breathing periods ($P < 0.001$).

Measurement of ambient and skin temperatures are shown in Figure 5. Although there were significant differences of skin temperature on the ear at the O_2-1 phase and on the toe at the O_2-2 and O_2-3 phases compared to the pre-phase, there were no significant differences in skin temperature between the pre -and post-phases for each body part. Moreover, there was no significant correlation between skin temperature and peripheral blood flow for each body part (ear: 0.028, $P = 0.741$; hand: 0.077, $P = 0.369$; feet: 0.066, $P = 0.447$).

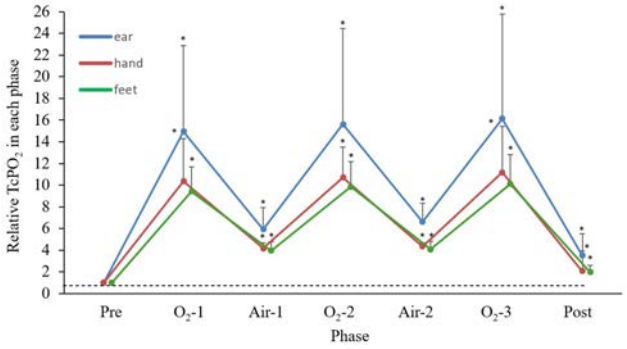
There were minimal and non-significant changes in pulse rate over the exposure phases: pre, mean 68.9 (SD 11.6); O_2-1, 64 (10.8); O_2-2, 63.3 (10.3); O_2-3, 62.5 (11.5); Post, 66.8 (10.6).

Discussion

The increase in dissolved O_2 in blood during HBO treatment contributes to the therapeutic effect of HBO;[1–6] however, any related changes in tissue blood flow have to date been under-researched. Thus, this study was designed to evaluate changes in blood flow and dissolved O_2 during HBO treatment. $TcPO_2$ levels significantly increased at all sites during all oxygen exposure phases. At two of our three sites (ear and finger) tissue perfusion exhibited a sustained reduction followed by a late increase back to baseline, and in the toe there was an early reduction followed by a steady rise to supra-baseline levels (Figure 3).

Previous research has shown that peripheral blood flow using LDF decreased and $TcPO_2$ increased in the hand and foot, respectively, during a period of 10 min HBO exposure at 253.3 kPa.[8] Several studies have reported a decrease in middle cerebral arterial blood flow velocity by transcranial Doppler during a short duration of HBO exposure in healthy volunteers.[7,10] A decreased peripheral blood flow early in the HBO exposure was also seen in this current study; however, as the exposure continued, an increase in the peripheral blood flow was observed. These results are consistent with the proposed hypothesis that following an initial decrease in peripheral blood flow there would be a subsequent increase in flow as a result of a relative increase in the vasodilatory effect of NO compared to the vasoconstrictive effect of

Figure 4
Changes in $TcPO_2$ relative to the Pre phase in the ear, hand, and foot (toe) (n = 20 subjects); *P < 0.001

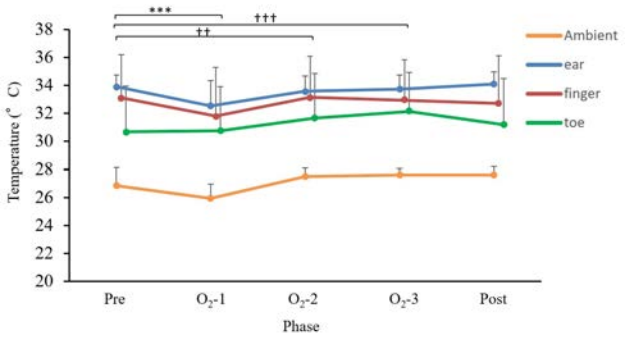


O_2 during a longer HBO exposure. It has been unclear how early effects of HBO treatment on peripheral blood flow would evolve over a longer exposure. Thus, this study is the first to examine peripheral blood flow and $TcPO_2$ during a longer HBO exposure and to show that peripheral blood flow increases.

There have been several reports that the peripheral blood flow in the earlobe might reflect the cerebral blood flow as the branches from the external carotid artery supply the earlobe.[13,14,22,23] Research in mice and rats has revealed an increase in the cerebral blood flow after a certain duration of HBO exposure.[23,24] Clinical research in healthy humans has indicated an increase in cerebral blood flow within one hour following the end of HBO treatment at 253.3 kPa.[19,25] Previous studies had assessed the peripheral blood flow in the limbs rather than in the ear.[8,9,19,20] In this study, it was observed that the changes in the blood flow in the earlobe (an initial decrease followed by an increase) were similar to those in the limbs. Thus, the changes in the blood flow in the earlobe observed in this study were consistent with the changes in the cerebral blood flow reported in other studies.[23–25]

The fluctuation measured in peripheral blood flow in the limbs (the acral dermis) was large and that in the ear (the non-acral dermis) was small. The vasoconstriction and vasodilation in the non-acral dermis are controlled by the sympathetic nervous system, and those in the acral dermis are controlled mainly by the opening and closing of arteriovenous anastomoses.[26] Thus, it has been reported that the vasoconstriction and vasodilation in the acral dermis may be influenced by the surrounding environment, such as temperature and O_2 levels, to a larger extent than those in other regions.[26,27] These findings are congruent with the findings in the current study, as fluctuation measured in the values of the peripheral blood flow was greater in the limbs than in the ear. Further studies should be conducted to provide details on the direct relationship between cerebral blood flow and peripheral blood flow in the earlobe.

Figure 5
Changes in ambient room temperature and skin temperature in the ear, finger, and toe (n = 20 subjects); comparisons are made with the Pre measure. *** difference in the temperature in the ear, P < 0.001; difference in the temperature in the toe, ††† P < 0.001, †† P < 0.01



In this study, there was a sustained increase in peripheral blood flow in the ear and feet after HBO exposure; however, the peripheral blood flow in the hand after HBO exposure returned to the level observed in the pre-phase. Although the exact reason for this difference is unclear, the hand may be more likely to undergo hyperoxia-induced vasoconstriction as it is a highly perfused area with systemic nervous regulation.[8] Thus, the peripheral blood flow in the hand may not increase more than the baseline level after HBO treatment. Further studies should be conducted to explore this issue.

LIMITATIONS

This study has several limitations. First, there is a possibility of errors in the measurement occurring due to differences in the positioning of sensors and body movements. Body movements and changes in sensor positioning may cause measurement errors as the LDF is a sensitive device.[20] However, a new wireless LDF device developed to reduce the influence of artefacts and noise was used in this study.[14] Thus, the results of this study may be considered more accurate than those measured by the conventional LDF devices in previous studies.[20]

Second, changes in skin temperature may have influenced the peripheral blood flow measurements.[8] Although significant differences in skin temperature were observed during several phases, the skin temperature in the post-phase was close to that in the pre-phase for each body part (Figure 5). Moreover, there was no significant correlation between the skin temperature and the peripheral blood flow. Thus, it was inferred that the influence of changes in skin temperature was too small to affect the outcome.

Third, $TcPO_2$ levels have been reported to return to pre-exposure levels gradually after HBO treatment;[28] however, we are unable to comment on the duration of increased peripheral blood flow as it was only measured for 10 min post exposure.

Fourth, only the effects of 60 min of HBO exposure at 253.3 kPa were evaluated in this study. This is a common treatment duration in Japan, although it is acknowledged that 90–120 min durations are standard in many places.[13,14] Thus, similar studies of HBO treatment with 90–120 min of exposure should be conducted in the future.

Fifth, it is still unclear whether the vasculature will respond in a similar manner after several HBO treatments as this study only evaluated a single HBO treatment in healthy volunteers. Changes in the peripheral blood flow following multiple HBO treatments should be investigated in the future.

Sixth, it is unclear why the values of peripheral blood flow measured in the finger and toe were higher than that in the ear lobe (Table 2). Furthermore, it has been reported that the value of peripheral blood flow measured in the ear was lower than those in the limbs in normal room environments.[13] Further studies exploring this issue should be conducted in the future.

Seventh, because of chamber configuration limitations it was necessary to use non-rebreather masks supplied by oxygen at 20 $L \cdot min^{-1}$ for oxygen administration. It is possible that this did not result in a 100% inspired fraction of oxygen. Nevertheless, the method was consistent between patients, and would have resulted in delivery of HBO at close to the ambient pressure of 253 kPa.

Finally, patients with circulation disorders, diabetic wounds, and radiation necrosis may not respond in the same way as the healthy volunteers included in this study. Based on the current study, future studies to investigate changes in the peripheral blood flow during HBO treatment in patients with circulatory disorders should be performed.

Conclusion

This study continuously examined the peripheral blood flow and $TcPO_2$ throughout the entire duration of a 60 min HBO exposure in healthy participants. Peripheral blood flow decreased at the beginning of the exposure, followed by a gradual increase, maintaining this level or increasing for at least 10 min after exposure compared to the baseline. $TcPO_2$ levels also increased throughout the treatment profiles. These findings show that peripheral blood flow increases. Thus, the study findings are meaningful for understanding that a longer HBO exposure causes an increase in peripheral blood flow after a decrease seen at 10–15 min of HBO exposure.

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Scuba diving-related fatalities in New Zealand, 2007 to 2016

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Key words
Cardiovascular; Coroner's findings; Diving deaths; Diving incidents; Drowning; Epidemiology; Obesity

Abstract
(Lippmann J, Lawrence C, Davis M. Scuba diving-related fatalities in New Zealand, 2007 to 2016. Diving and Hyperbaric Medicine. 2021 December 20;51(4):345–354. doi: 10.28920/dhm51.4.345-354. PMID: 34897599.)
Introduction: New Zealand (NZ) diving-related fatalities have been reported since the 1960s. The aim is to identify contributing risk factors, including medical, and to inform appropriate preventative strategies.
Methods: NZ scuba diving fatalities from 2007 to 2016 were searched from multiple sources – the National Coronial Information System (NCIS); the NZ Chief Coroner's office; Water Safety NZ *Drownbase*™ and the NZ Police National Dive Squad records. For inclusion, a victim must have been wearing a scuba set (which may include a rebreather). A key word search of the NCIS was made and the results matched to the other databases. An Excel® database was created and a chain of events analysis (CEA) conducted.
Results: Forty-eight scuba diving fatalities were identified, 40 men and eight women, average age 47 years (range 17–68), 20 of Māori ethnicity. Thirty-five were classified as overweight (14) or obese (21). Pre-existing medical risk factors were identified, either pre dive or at autopsy, in 37 divers, the commonest being ischaemic heart disease (IHD, 20), left ventricular hypertrophy (LVH, 18) and hypertension (seven). IHD, LVH and obesity were variously associated with each other. The likely commonest disabling conditions, identified in 32 cases, were asphyxia (15), cardiac (nine) and pulmonary barotrauma/cerebral arterial gas embolism (five). Multiple environmental and diving practice breaches and poor planning were identified in the CEA, similar to those seen in other studies. Thirty-eight divers had not released their weight belt. Information on resuscitation was limited.
Conclusions: Obesity and cardiovascular disease were common and Māori appear to be over-represented, both as previously reported.

Introduction

With its long and accessible coastline and its diverse underwater environment rich in marine life, scuba diving in New Zealand (NZ) is an attractive recreation. A 2018 online Water Safety NZ (WSNZ) survey of the NZ public reported that 11% of 1,094 respondents had dived or snorkelled in the previous year, although it did not differentiate between these activities.[1] There are few reliable data on the total number of active divers at any given time in NZ,[2,3] although over the study period there were an average of slightly over 5,000 entry-level divers certified annually by the Professional Association of Diving Instructors (PADI; Richardson D, personal communication, June 2021), which likely represents at least 80% of the diver training market in NZ. This has declined from about 6,000 in 2000.[2] This decline would partly reflect the withdrawal of generous government subsidies previously offered to encourage career-related training in the recreational diving industry, as well as a general softening of the industry. Also, it has been reported anecdotally that a relatively high proportion of scuba divers in NZ are uncertified.

Earlier reports have reviewed scuba- and snorkelling-related deaths in NZ from 1961 through 1973,[4,5] 1981–1986,[6–8] 1980–2000[2] and 2000–2006.[9] A recent report by the present authors detailed snorkelling-related deaths from 2007 to 2016.[10] This companion study examines scuba diving-related deaths in NZ waters for the same period with the aim of identifying underlying risk factors, including medical, and of informing appropriate preventative strategies.

Methods

This was a case series of scuba diving fatalities that occurred in NZ waters from 01 January 2007 to 31 December 2016.

For inclusion in this series, a victim must have been reported to have been wearing a scuba set (which may include a rebreather).

ETHICS APPROVAL

Ethics approvals for the collection and reporting of these data were received from the Victorian Department of Justice Human Research Ethics Committee (CF/18/12735) and the NZ Chief Coroner, NZ Department of Justice, to access additional coronial records, WSNZ to access *DrownBase*™ (https://watersafety.org.nz/drowning%20statistics), the NZ Police Research Review and Access Committee (EV-12-531) and the NZ Police Maori Pacific and Ethnic Services to access NZ Police National Dive Squad (NZPNDS) records. The benefits of reviewing multiple data sources have been described previously.[11]

SEARCH

All fatalities reported to NZ coroners since July 2007 have been added to the Australian-based National Coronial Information System (NCIS).[12] A comprehensive key-word search was made of the NCIS for scuba diving-related deaths from 01 July 2007 to 31 December 2016. Key words included scuba, compressed air, compressed gas and div* and underwater fishing. Data obtained from the NCIS was matched with that listed on the Australasian Diving Safety Foundation (ADSF) diving fatality database, the WSNZ *Drownbase*™ (https://watersafety.org.nz/drowning%20statistics), and the NZPNDS diving fatality database to minimise the risk of over- or under-reporting. Coronial data not included on the NCIS (i.e., 1 January to 30 June 2007 and for 'open' cases) were provided by the NZ Ministry of Justice.

REVIEW PROCEDURE

The principal investigator (JL) reviewed all datasets to resolve any discrepancies between the various sources, and then prepared initial incident summaries for each case and created an anonymised, protected Microsoft Excel® spreadsheet. The coronial reports and these summaries were then independently reviewed by each of the co-investigators (CL and MD), any differences in interpretation debated and consensus reached; CL focusing, in particular, on the reported autopsy findings. The NZPNDS records were then hand-searched (MD) to help fill any gaps in the data, especially related to the equipment used. Based on these reviews, the Excel database was finalised.

A chain of events analysis (CEA) was performed for each case using a validated template.[13] Each CEA is based on the evidence in the coronial and autopsy reports. However, in some cases the authors disagreed with the interpretation of the findings, so the disabling agents and disabling injuries reported in the CEAs are based on our consensus interpretations, but the cause of death given is that of the pathologist conducting the autopsy or by the coroner where no autopsy was performed.

OUTCOME MEASURES

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

Results

From 01 January 2007 to 31 December of 2016 there was 48 identified scuba diving-related deaths in NZ territorial waters, 40 men and eight women. Forty-seven of the victims used open-circuit scuba and one used a closed-circuit rebreather (CCR).

DEMOGRAPHICS

Ethnicity is documented in NZ and 17 of the 48 divers were NZ residents of European origin, 20 were of Māori ethnicity, whilst ten were tourists (seven of European origin and three Asians) and there was one diver from another Pacific Island. Mean (SD) age was 47 (12) years, and there were no differences in age between the sexes ($P = 0.60$) or between ethnicities ($P = 0.20$). Body mass index (BMI) was available for 43 victims (mean [SD] 29.3 [5.3] $kg{\cdot}m^{-2}$) and was similar between the sexes (29.6 [5.1] $kg{\cdot}m^{-2}$ for 36 men, 28.0 [6.4] $kg.m^{-2}$ for seven women). Thirty-five victims were classified as overweight (14, 12 men and two women; BMI 25–29.9 $kg{\cdot}m^{-2}$) or obese (21, 18 men and three women; BMI ≥ 30 $kg{\cdot}m^{-2}$) (Table 1). Seventeen of the 35 NZ residents for which the BMI was known were classified as obese. There were no statistically significant differences in the actual BMIs between NZ residents of European extraction and Māori victims ($P = 0.60$) or in the BMI classification between these two groups ($P = 0.93$; Table 1).

PRE-EXISTING MEDICAL CONDITIONS AND MEDICATIONS

Eighty-four pre-existing medical or pharmaceutical risk factors were identified as either known pre-dive (55), or found postmortem (29), in 37 of the 48 divers; some divers having more than one risk factor present. Apart from obesity, other known pre-dive health factors were present in over one third of victims. These included ischaemic heart disease (IHD, 10); hypertension (seven); asthma or chronic obstructive pulmonary disease (five); epilepsy (two); use of amphetamines/alcohol (five), depression on antidepressants

Table 1
Body mass index (BMI) classification of 43 scuba fatality victims according to their ethnicity; no data for five divers; $P = 0.93$ for the difference in BMI classification between Māori and New Zealand residents of European extraction

BMI ($kg \cdot m^{-2}$)	Māori ($n = 18$)	NZ European ($n = 16$)	Others ($n = 10$)
Normal (18.5–24.9)	2	2	4
Overweight (25–29.9)	6	6	2
Obese (≥ 30)	9	8	4
Mean (SD)	30.4 (6.4)	29.1 (3.6)	27.7 (5.3)

(three) and one diver with non-insulin-dependent diabetes mellitus. At autopsy, a further 10 cases of IHD and 18 divers with left ventricular hypertrophy or cardiomegaly (including the seven with known hypertension) were identified. One diver had a history of shortness of breath whilst diving, suggestive of previous immersion pulmonary oedema (IPO).

Whether or not victims were taking medications was documented in the coronial reports for only 17 divers. Generally poorly documented in the coronial reports, the recording of such data appeared to improve in the final two years of the study. Anti-hypertensives (seven); anti-depressants (three); bronchodilator inhalers (three), anti-epileptics (two) and allopurinol and analgesics were noted. Methamphetamine, cannabis metabolites and/or alcohol were identified in the blood of five divers, four of whom had other medical risk factors present as well.

CERTIFICATIONS AND EXPERIENCE

Thirty (21 non-Māori and nine Māori) of the victims were recorded as having received some form of diver certification, usually Open Water Diver (OWD). At least six divers had been certified as Advanced Open Water Diver (AOWD or equivalent), and there were two instructors. At least eight of the victims were uncertified, five of them Māori. No information about certification was available in 10 cases, six of them Māori. Most reports included very little pertinent information regarding 'experience' other than comments from family, buddies or investigating officers. Based on these, 22 divers were classified (often subjectively) as 'experienced'; four were defined as 'novices', three divers had no experience at all and there was insufficient information to make any determination in one case.

LOCATION AND SETTING

Thirty-nine of the incidents occurred in the North Island and the remaining nine in the South Island. Nine incidents occurred in a commercial setting, either on dive charters and/or during training. The other 39 incidents occurred during private diving activities.

BUDDY AND SUPERVISION CIRCUMSTANCES

In the 46 incidents where the buddy circumstances were clear, 17 divers had set out solo, 17 had separated from their buddy or group before the incident, three separated during the incident and the remaining nine were still with a buddy or group. Māori victims were five times more likely to have been solo diving than their non-Māori counterparts (OR = 4.9 [95% CI 1.24, 19.46], $P = 0.02$). There was no information about supervision in one incident; however, 34 divers were supposedly under some supervision and 13 were unsupervised.

DIVE PURPOSE

The activity was unknown for one incident. Twenty-nine victims were harvesting seafood; other activities included sightseeing (eight), tasks associated with boats (three), wreck diving (two) and training (five). Three of the training-related deaths occurred during AOWD training or equivalent, including a double fatality in a freshwater lake with a depth of 54 msw. These two victims separated from the group in poor visibility. The other AOWD incident was due to a medical issue not directly related to training or skills. Another death occurred during the initial check-out dive of a commercial training course, when the diver became separated from the group in low visibility. This diver who was from the tropics had failed to complete the prior swim tests due to the cold. There was also one death during a Discover Scuba Diving (DSD) experience. The instructor had requested that the student make her way to shore while he continued to dive with another diver. The victim was later found deceased, with her BCD inflated and an empty tank.

DEPTH OF DIVE AND INCIDENT

The maximum dive depth recorded in 43 cases ranged from 2 to 67 metres (median [IQR] 11 [7, 19] metres). Only six of the fatal dives were to 30 metres or deeper. Twenty-seven incidents likely occurred underwater, with at least six of these during ascent, and 13 incidents were reported to have occurred on the surface before or after the dive. One diver collapsed after returning home from a dive on which he had felt unwell; this death was included as being diving-related. There were insufficient data to determine incident depth in seven cases.

DIVE SUITS, WEIGHTING AND BUOYANCY COMPENSATORS

Forty-four divers had worn wetsuits and one (the CCR diver) a drysuit. One diver only wore shorts and t-shirt despite carrying 9 kg of weights. There were no data in two cases. At least 38 divers were found still wearing their weights,

five had ditched their belts, and one diver had ditched only one of two pockets of integrated weights. The amount of weight carried was recorded in 29 cases, and ranged from 7 to 19.2 kg, with mean (SD) of 12.6 (2.8) kg. The NZPNDS investigations suggested that at least 15 of the victims were overweighted. At least three of the divers were not wearing a buoyancy compensator device (BCD) during the dive, instead wearing cylinders with backpacks. All these carried weight belts (with weights ranging from around 8 to 19 kg). Of the 44 divers whose incident occurred at sea and who were wearing a BCD, eight were found with an inflated BCD; 24 divers' BCDs were not inflated and in 12 cases the state of BCD inflation was not reported.

BREATHING GAS SUPPLY

One diver was using a CCR and the remainder used open circuit scuba. The CCR diver used trimix and at least 46 of the others were breathing air. At least 17 of the victims had exhausted or near-exhausted their breathing gas supply, whilst 25 had sufficient remaining air to surface safely. In six cases, the air supply circumstances were unstated, and in one not applicable (a diver who died at home). Analysis of the remaining gas was available in 22 cases. One cylinder was reported to be slightly contaminated with methane and carbon monoxide, although these were not considered to be contributory by the police. Another contained 900 ppm of carbon dioxide (acceptable level 480 ppm).[14] A higher than recommended water vapour content was found in 16 cylinders.

EQUIPMENT FAULTS

Equipment was tested by the NZNPDS in at least 41 cases and faults, sometimes minor, were identified in 25 of these. In 11 cases, faults were assessed as being contributory to the incident, and possibly contributory to another eight. The main problems identified were with demand valves (e.g., faulty/ill-fitting mouthpieces or diaphragms causing 'wet breathing', high breathing resistance or free flowing), BCDs (poor fit, leaks, faulty inflators), inaccurate gauges, overweighting and weight belts that could not be released easily in an emergency. Of note, the bodies of six divers were found with their catch bag still attached to their BCD or weight belt.

RESCUE AND FIRST AID

A rescue attempt (i.e., the victim was accessed and landed relatively quickly, with an arguable possibility of survival) was made in 25 of the incidents. It was reported that in-water rescue breathing was performed in at least two of these. At least 22 of 38 divers still wearing their weight belt had to be searched for and recovered from underwater, introducing substantial delays and reducing the likelihood of survival irrespective of any resuscitation attempts. There was generally very little information about the first aid provided, other than whether basic life support (BLS) was performed. BLS was attempted in at least 27 incidents but withheld in two when possibly reasonable to have been performed. Resuscitation was not appropriate in 16 cases due to extended delays. There was no information in three cases. Only five reports included a mention of airway complications, which included frothy sputum (three) and regurgitated stomach contents (two). The use of a defibrillator was only mentioned in two reports. Both were used by medical responders, rather than anyone involved with the dive, and after considerable delays. The presenting rhythm, or if any shock was delivered, was not indicated in either report. Information about whether oxygen was available at the site was sparse, and oxygen was only mentioned in three reports, all of these in a commercial setting. In two cases, it was used during resuscitation. In the third, the report mentioned that the distressed diver had asked for oxygen and a crew member looked for a '*bottle*'.

AUTOPSIES

Where there was a delay in recovery of the body, particularly in the South Island with significant sea lice activity, extensive soft tissue loss in three cases severely limited the contribution that the autopsy made to the determination of cause of death. No body was recovered in one case.

As in the companion report on snorkellers,[10] IHD (20), left ventricular hypertrophy (18), and obesity (21) were present at autopsy and represented health risks to diving. In all 18 divers with LVH or cardiomegaly, this was undiagnosed pre dive, whilst known hypertension was present in seven. Undiagnosed IHD was present in 10 cases.

Methamphetamine was detected in blood from four divers. These divers all had other medical risk factors: one had IHD; one had cannabis and IHD; one had cannabis and LVH and one had cannabis and epilepsy.

CHAIN OF EVENTS ANALYSIS

Predisposing factor (PF)

There were 113 possible or likely PFs identified in the 48 incidents (Table 2). Medical conditions are discussed above. Approximately one half of the PFs could be classified as deviations from accepted safe diving practice (e.g., diving under the influence of recreational drugs, diving solo, diving with inadequate or faulty equipment among others). Equipment faults included poorly adjusted or leaking demand valves (seven), faulty contents gauges (three) and faulty BCDs (three). Inappropriate equipment included ill-fitting fins (three), over-tight wetsuits (two), an overly large BCD and a weight belt secured by a harness which made it impossible to release in an emergency. Absent equipment which might have prevented the incident or changed the outcome included a BCD (two), knife (two), torch (two), fin (one), wetsuit (one) and secondary demand valve.

Table 2
Predisposing factors (n = 113) associated with 48 scuba fatalities; some deaths involved multiple predisposing factors. * Some of these 24 equipment incidents involved multiple faults or omissions; CCR – closed-circuit rebreather

Predisposing factors (n divers)	**Subgroup**	**Number of factors**
Health (n = 37)	Significant medical condition(s)	29
	Drug/alcohol intake	5
	Obesity only	3
Absence of appropriate equipment or use of faulty equipment* (n = 24)	Overweighted	15
	Faults	10
	Absence	5
	Other	9
Planning (n = 24)	Solo diving	15
	Poor choice of dive site	3
	Adverse conditions	3
	Poor buddy system	2
	Poor air planning	1
Training/experience/skills (n = 12)	Lack of skills and/or experience for dive	7
	No recent experience	3
	Untrained and inexperienced	2
Activity (n = 5)	Deep diving in zero visibility and beyond experience	2
	Deep, working dive on CCR	1
	Deeper dive hunting crayfish	1
	Removal of scuba in strong surge	1
Organisational (n = 4)	Poor choice of site in conditions	3
	Failure to check certification	1
Poor supervision by: (n = 3)	Divemaster	1
	Instructor	1
	Boat operator	1
Other (n = 4)	Poor communication	2
	Poor attitude	2

The main planning factor was a decision to dive solo or intentionally separating during the dive. In two incidents, the separation, though not anticipated, occurred directly because of an intentionally loose buddy system. A poor choice of dive site (a lake with a depth of 54 metres and low visibility at depth) contributed to the deaths of two students during an AOWD course; they were easily able to disappear and exceed the planned depth of 39 metres. In at least three cases, the victims did not recognise that the prevailing conditions were beyond their capabilities, especially given they were diving solo. One diver started a solo dive with only 50 bar of air in his tank and ran out of air.

Training, experience and/or skills-related PFs were identified in 12 incidents, which included the training-related deaths described earlier. Four of the five activity-related deaths were associated with deeper diving. These included the double fatality, another diver possibly ran out of air while hunting crayfish at 40 metres' depth, whilst the CCR diver was working hard at depth. The fifth victim did an out-of-air ascent after removing his scuba unit to enter a cave in a strong surge.

Organisational factors included poor matching of the dive site to the skills and/or experience of the divers. These included the double training fatality and the diver participating in commercial diver training. Another diver was highly inexperienced and likely uncertified and the dive operator failed to take this into account. Incidents involving poor supervision included a divemaster who allowed an inexperienced diver to dive in poor conditions and lost sight of him, an instructor who requested the DSD participant to make their way to shore without supervision and a boat operator who failed to disengage the boat's propeller when a diver entered the water. Poor communication is likely present in many diving mishaps but was obvious in two cases; one involving three CCR divers working together underwater to raise a heavy object and the other the boat propeller incident. The incident in which a poor attitude was evident involved the two student divers who reportedly tried to race each other to the bottom of the deep lake.

Triggers

Gas supply triggers, running out of or very low on air, were implicated in at least 17 incidents (Table 3). In 14 of these, the victim was either solo (six), or had separated from their buddy prior to the incident (eight). There was no statistical association between harvesting seafood and running out of air (OR = 2.2. [95% CI 0.60, 8.13], P = 0.23).

Table 3
Triggers associated with 48 scuba fatalities; some deaths were associated with multiple triggers

Triggers	Subgroup	*n*
Gas supply (*n* = 17)	Out of gas	13
	Low gas	3
	Loss of regulator	1
Environmental (*n* = 17)	Immersion effects	10
	Conditions	4
	Narcosis	2
	Entrapment	1
Exertion (*n* = 8)	During dive	6
	Pre dive	1
	Post dive	1
Equipment (*n* = 2)	Various	2
Buoyancy (*n* = 1)	Excessively overweighted	1
Primary diver error (*n* = 1)	Entered with propellor engaged	1
Unknown (*n* = 8)	–	8

Ten of the environmental triggers were believed to have arisen from the direct effects of immersion, which can impact cardiac function and lead to cardiac arrhythmias in susceptible persons. Conditions such as swell, surge, current, poor visibility and cold were also implicated in incidents, in some cases compounding the effects of immersion. Of the eight cases identified with likely exertion triggers, six of the divers were obese, four were carrying heavy catch bags, two were taking beta blockers (known to reduce exercise tolerance), at least three had stiff demand valves and at least one was substantially overweighted for the dive. Cardiac conditions were identified as the disabling agents in at least four, possibly six of these incidents.

Although equipment deficiencies were apparent and likely contributory to some incidents, they were only identified as direct triggers in two. One perforated demand valve diaphragm caused aspiration and a likely subsequent asthma event. The collapse of a surface marker buoy being used to lift a heavy object caused the loss of the ascent shot line and subsequent complications during ascent from a deep dive (the CCR diver).

Disabling agent (DA)

Medical factors, predominantly cardiac-related (11), but also epilepsy (two), asthma (two), IPO (one) and methamphetamine toxicity (one) were identified as the main likely DA (Figure 1). The gas-related DAs all involved exhaustion of breathing air supply. The buoyancy problems were related to being negatively buoyant from overweighting, the absence of or lack of a properly functioning BCD and/or

Figure 1
Pareto chart of disabling agents associated with 39 of 47 scuba fatalities; one occurred on land post dive and in eight cases, no disabling agent could be identified. In some fatalities more than one possible disabling agent was present. The blue line represents cumulative percentage

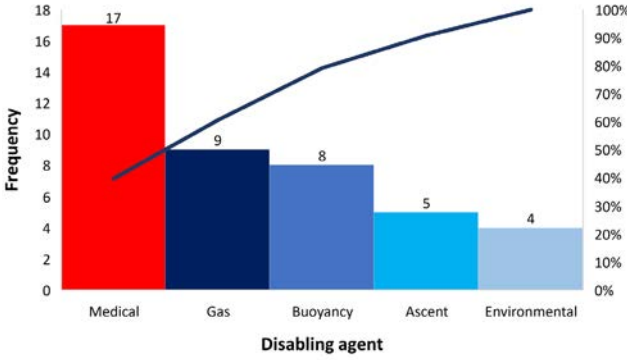
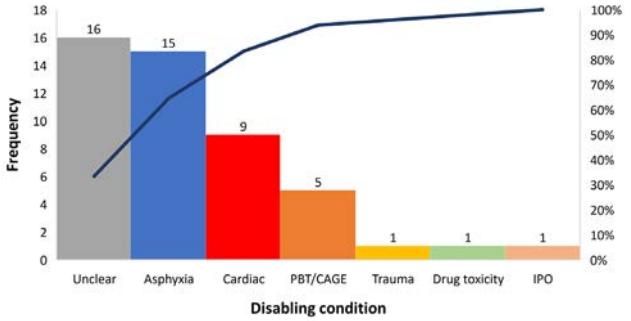


Figure 2
Pareto chart of disabling conditions in 48 scuba diving fatalities; the blue line represents cumulative percentage. IPO – Immersion pulmonary oedema; PBT/CAGE – pulmonary barotrauma/cerebral arterial gas embolism. The blue line represents cumulative percentage



carrying a heavy catch bag. The ascent-related issues arose from out-of-gas ascents leading to pulmonary barotrauma (PBT) and cerebral arterial gas embolism (CAGE). The environment-related DAs included adverse conditions, entrapment and contact with a boat propeller.

Disabling condition (DC)

The predominant DCs identified were asphyxia, cardiac causes and CAGE with or without evidence of PBT (Figure 2). Others included IPO (one), trauma (one) and likely methamphetamine toxicity (one). In 16 cases, no clear DC could be identified. In six of these, there were indicators of a possible cardiac-related incident, although other factors, such as signs of drowning or CAGE, hampered a clear determination. PBT with CAGE was a possible disabling condition in another six incidents although the evidence was unclear and there were also indications of possible drowning.

Discussion

DEMOGRAPHICS AND MEDICAL CONDITIONS

PADI, which is the largest recreational diving instruction agency in the world, reported that, over much of the period of this study, the ratio of male to female recreational entry-level divers certified worldwide by PADI was roughly two-thirds to one-third.[15] However, certification data may not reflect the actual proportions of the sexes diving in NZ as a substantial proportion of NZ divers appear to be uncertified, and there is anecdotal evidence that uncertified divers are predominantly male. Nevertheless, the proportion of female divers in this (8/48) and previous NZ fatality series (23 of 174, 13%)[2,9] would seem to be less than expected on the basis of their level of diving activity.

The mean age of NZ diving fatalities has increased from 34 years in the 1980–2000 study[2] by over a decade, to 47 years. Whilst the average age in the present series is similar to that of contemporaneous fatalities amongst DAN America members,[16] other reports suggest the average age of divers at certification is in the mid-30s, with more active divers aged in their mid-40s to 50s.[15,17] These data are consistent with the apparent increase in deaths of older divers with important medical co-morbidities.

In those victims where BMI was known, one half were obese, irrespective of ethnicity, with the prevalence of obesity in the general adult NZ population being reported as 48% and 29% in Māori and non-Māori respectively.[18] Although the numbers in this study are small and need to be interpreted cautiously, the proportion of obese non-Māori fatality victims was higher than the general population. Obesity was reported as a possible risk factor in cardiac-related diving deaths in Australia,[19] and in one third of the NZ divers, both obesity and cardiac disease were present.

Examining *DrownBase*™ reveals that between 2015 and 2019, almost one-fifth (18%) of all drownings were associated with alcohol and/or drug consumption. In the present series, alcohol or drugs were likely risk factors or a direct cause of death in five of 47 deaths (one body missing). Numbers are too small, and the study relates to an earlier time period, to determine whether this plays less of a role in scuba-related drownings than overall in drowning deaths in NZ. However, the prevalence appears not to have changed from that of the 1980–2000 NZ series (10/123 scuba divers).[2]

AUTOPSIES

Previously unrecognised LVH was present in 18 divers at autopsy, likely related to obesity and hypertension,[20] and previously unrecognised IHD in ten. There is an extensive literature studying the links between LVH, sustained arrhythmias and sudden cardiac death.[21] A recent review summarising the basic scientific and clinical data for this concluded that "*overall there appears to be a dearth of trials confirming association between hypertensive LVH and progression to sustained* [ventricular arrhythmias] *and sudden cardiac death*".[22] The prevalence of LVH in a Scandinavian population study was 14.9% in males on echocardiography, with BMI and hypertension being the most pronounced independent risk factors.[20]

Within a diving context, a comparative study reported that LVH was significantly greater in 100 scuba diving fatalities than in 178 reasonably matched traffic fatalities taken from a USA database.[23] In an Australian series from 2001 to 2013, LVH was documented at autopsy in 24 (20%) of 126 victims, especially in association with hypertension.[24] In a recent open-water study, five of 60 divers undergoing echocardiography had evidence of LVH.[25] Therefore, the presence of LVH in 17 of 43 autopsies (four unreliable autopsies and one body missing) in the present series is of serious concern. It remains difficult to assess the relative contribution of cardiovascular events in drowning due to the lack of a definitive postmortem test to confirm whether an arrhythmia has occurred. However, the presence of cardiovascular disease increases the risks of sudden arrhythmias and is likely to reduce the chances of the victim's survival.

Despite the increasing availability of computed tomography (CT) scans pre autopsy, it is often not possible to perform a post-mortem CT scan within the first three hours after death. As the pre-scan interval extends beyond this, the distinction between gas embolism and postmortem decompression artefact at autopsy becomes increasingly difficult, if not impossible.[26] Thus, the diagnosis of CAGE at autopsy remains problematic in this series despite five cases having histories suggestive of CAGE. However, in the 49 autopsies in the 1980–2000 series considered to have been performed to the Royal Australasian College of Physicians guidelines,[2,27] CAGE was given as the cause of death in 27 divers – a much higher proportion than in the present study.

The presence of methamphetamine in four cases is disturbing, particularly in combination with cannabis, IHD, LVH and epilepsy. Methamphetamine is highly arrhythmogenic and in a person with ischaemic heart disease, cardiomegaly and LVH would be regarded by a forensic pathologist as a significant contributor to death from a cardiac event.

NZ has both forensic pathologists and regional anatomical pathologists who perform coronial autopsies. Whilst the standard of autopsy reports has improved compared with the 1980–2000 NZ study when only half the autopsies were performed to the guidelines for scuba diving autopsy procedures,[2,27] there were two autopsies by regional pathologists which were considered not to have met the recommended guidelines. Whenever possible, autopsies

of scuba diving deaths should be performed by a trained forensic pathologist in order to extract the best information on the possible cause(s) of and contributor(s) to death.

DIVING CERTIFICATION AND EXPERIENCE

It is difficult to effectively define 'experience' as this depends on many factors, including dives done, time of accumulation and currency, among others. As a result, our classification of 22 divers as 'experienced' was often subjective. Of note, most deaths in the training/experience/skills category were likely a result of primary drowning or CAGE, unsurprising in inexperienced divers. In the 1980–2000 NZ series,[2] lack of diving qualifications and/or experience were common. In the present series, whilst three-quarters of the non-Māori divers had evidence of some form of dive training certification, this was the case in only half the Māori divers. Again, the numbers are small, but this does suggest that increasing formal training in scuba diving amongst Māori men, in particular, may be worthwhile to improve diving safety. It is debated as to whether legislation to prevent unqualified diving would help in this regard (e.g., dive centres and filling stations legally required to demand evidence of a diving certificate before selling or hiring diving equipment or filling cylinders).

Twenty of the 48 divers were of Māori ethnicity, whereas in 2012 (the middle of this study) 16.5% of the general NZ population were recorded as Māori. As was noted for breath-hold/snorkelling deaths over the same period,[10] Māori appear to be over-represented in these fatality data. This is in slight contrast to the 1980–2000 study in which only 23 of 123 divers (19%) were Māori.[2] At that time, fatalities in Māori tended to be more amongst snorkellers (21 of 61).[2] Poorer health, greater uptake but less formal scuba training and deviation from accepted scuba diving practices seem to be specific risk factors for this community. The latter was particularly highlighted by the relative frequency of solo diving in this cohort.

Deviation from accepted scuba diving practices has been highlighted as a problem elsewhere.[28] In that study of 122 divers, "*divers who died from something other than a medical cause were seven times as likely to have one or more violations associated with the fatality*".[28]

SETTING AND PURPOSE

Unlike in Australia where almost half of scuba deaths occurred in a commercial setting,[19] a far higher proportion (39/48) of the deaths in this series occurred in a private setting. This suggests that more NZ divers undertake private diving but there appear to be no published data to confirm this. In theory, diving in a commercial setting should be safer due to the availability of professional supervision. However, deaths can and do occur despite this, as evidenced by Australian data.[19] The activities of the NZ victims also differed from the Australian victims, in that a much higher proportion (29/48) were harvesting seafood compared to Australia (18%) at the time of their demise. Divers can easily become distracted when harvesting seafood and fail to closely monitor their air supply and surroundings. Although there was no association found between harvesting seafood and running out of air, this is based on small numbers. Another marked difference in diving practice was the more than threefold higher proportion of NZ divers who set out solo compared to their Australian counterparts.[19] Although the prevalence of solo diving in NZ victims has fallen substantially over the years (half compared to 78% in 1980–2000)[2] it remains higher than reported in the United States (USA 9%)[29] and the United Kingdom (UK 18.5%).[30]

WEIGHTS, BCD AND GAS SUPPLY MANAGEMENT

Carrying excess weights affects buoyancy management and leads to additional exertion and air usage. Almost one third of divers were considered by the NZPNDS to have been overweighted. Correct weighting and buoyancy control skills are important requirements for safe diving, and divers should strive to achieve these. Overweighting was assessed as the primary causal factor in 5.7% of a series of 140 diving deaths in the UK.[30] In addition, it is important for divers to try to reach the surface in an emergency, rather than become unconscious and need to be found and recovered from underwater.[31] To this end, it is usually necessary to inflate the BCD and/or ditch weights. In NZ fatalities, 159 of 173 divers (92%) between 1980 and 2016 had not released their weights,[2,9] whilst in Australian fatalities, around one half had uninflated BCDs and 82% still had their weights in situ.[19]

Of the 41 cases where details of the remaining breathing gas supply were available, 17 divers had exhausted or near-exhausted their supply. This is consistent with Australian data where more than 40% of victims were completely or almost out of gas;[19] albeit higher than earlier reports from the USA (21%)[29] and the UK (8.6%).[30] These data highlight an ongoing problem of divers failing to adequately monitor their breathing gas and leave sufficient to surface safely. Despite being a fundamental and obvious requirement for diving with scuba, it appears to require continual reinforcement. The high number of cylinders recorded with elevated water content raises concerns about compressor maintenance and the regularity of replacing or correctly repacking the water filters. In NZ, the NZ Underwater Association (NZUA) is contacted by Worksafe to monitor commercial air filling stations but there is no check on privately-owned compressors.

FIRST AID

Little or no information is included in the police or coronial reports regarding first aid, including oxygen provision, defibrillator usage and ambulance management. A similar paucity of information on rescue and resuscitation was reported in a 13-year Australian series.[32] Improved data

collection and recording by official on-site investigators, preferably with knowledge of diving, would better inform potential or necessary improvements.

POLICE INVESTIGATIONS

The NZ National Police Diving Squad investigates and reports on almost all scuba fatalities in New Zealand. This is possible because of New Zealand's comparatively small population and enables consistency in such reports. The reports, which follow a fixed format developed in the early 2000s, are generally of a high standard and provide valuable information, including a review of the circumstances of the incident, extensive equipment reports and gas analyses. Summaries of these appear in many of the published coroner's findings.

LIMITATIONS

Even using multiple sources, it is possible that some fatalities were not recorded due to limitations in recording and NCIS searches. In previous studies,[2,4–9] a few cases in which the cause of death was not recorded as 'drowning', and not documented in WSNZ's *Drownbase*™, may have been missed, but the current search was wider than for those studies. Information from immersion incidents is notoriously patchy and incomplete; especially when unwitnessed. However, in this series, the majority of the coronial and autopsy reports were quite detailed and provided good insight into what likely happened. Health records were often deficient, so there is a strong subjective element to determining what personal factors contributing to a death were important. The CEA attempts to identify the predominant features of each case, but there always remains an element of uncertainty. Nevertheless, some clear lessons can be learned, such as the high frequencies of pre-existing deleterious medical conditions, the contribution of environmental conditions and/or poor diving practices and the apparent disproportionate number of Māori.

Conclusions

Forty-eight scuba fatalities occurred in NZ between 2007 and 2016 in victims of an average age of 47 years. Numerous pre-dive risk factors, both medical and non-medical were present in these incidents. Multiple environmental factors, poor planning, and diving practice breeches were identified in the CEA, similar to those seen in some other studies. Solo diving or separation from dive buddies was a common feature. Thirty-eight divers had not released their weight belt. Information on resuscitation was limited. Obesity and cardiovascular disease were common, and Māori appear to be over-represented, both as reported in previous NZ studies.

Recommendations

- Dive training agencies need to improve training for emergencies, particularly the release of the weight belt. This remains a highly unsatisfactory part of recreational dive training.
- More emphasis during training needs to be placed on breathing gas management and the importance of a good buddy system.
- Middle-aged and elderly, overweight males, especially those with hypertension, are at greatest risk from scuba diving and need to undergo thorough, regular medical assessment, preferably by doctors with knowledge of diving medicine.
- More emphasis on diving safety (both snorkelling and scuba) is needed for Māori in the current water safety programmes of WSNZ and its partners.
- Legislation requiring evidence of diving certification before selling or hiring dive equipment or filling diving cylinders should be considered.
- There is still room for improvement in the documentation of diving fatalities, including obtaining autopsies in as timely a manner as possible; all diving autopsies should be performed by a forensic pathologist.
- The NZUA and worksafe need to review why the air in many cylinders, including from registered filling stations that they monitor, had water vapour levels that exceeded the recommended standard.

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What are the effects of scuba diving-based interventions for clients with neurological disability, autism or intellectual disability? A systematic review

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Adapted physical activity; Disabled diver; Evidence; Physiology; Psychology; Review article

Abstract
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Introduction: Recreational scuba diving has existed for over 70 years with organisations emerging that teach individuals with disabilities to dive. It is unclear what the physical and psychosocial effects of scuba interventions might be. This systematic review explores evidence for the effects of scuba diving in individuals with neurological disability, intellectual disability and autism.
Methods: The databases Medline, EMBASE, Ovid Emcare, and SportDiscus were searched. Included papers described a scuba-based intervention for clients with a neurological disability, intellectual disability and autism, with physical or psychosocial outcomes explored in the paper. Quality of the included papers was assessed using the McMaster Appraisal Tools, with descriptive data synthesis completed to explore the physical and psychosocial effects of the interventions.
Results: Four papers met the inclusion criteria: a cross-sectional investigation, a phenomenological study, a case-control study and a multiple case study. The quality of the papers was low to moderate. Papers addressed the psychosocial effects of scuba diving, including motivation to participate, participant experiences, the effect on cognition and physical self-concept. One study reported an increase in self-concept for the majority of participants. An increase in understanding instructions and in visual attention was reported in another. Enjoyment of the activity was reported and motivators to be involved in scuba diving for people with disabilities included fun and excitement. No papers addressed functional outcomes.
Conclusions: Whilst scuba diving interventions appear to enhance physical self-concept and are enjoyable, conclusive evidence regarding effectiveness could not be determined. Research in this area is extremely limited.

Introduction

The World Health Organisation (WHO) estimates that over 15% of the world's population is living with a disability.[1] Therefore, it might be assumed that some scuba divers live with a disability. There are several organisations providing training and scuba experiences for people living with a disability, such as the International Association for Handicapped Divers (https://www.iahd.org/), the Handicapped Scuba Association (https://www.hsascuba.com/) and Disabled Divers International (https://www.ddivers.org/). Additionally, there are services offering pool-based scuba diving experiences in several countries.[2,3]

A recent scoping review[4] revealed that there is little research looking at scuba interventions compared to other water-based interventions for people with neurological disabilities, intellectual disabilities, or autism; however, this is an emerging area. The quality of the sources identified in this review was limited (18 web sites and non-scientific articles met inclusion criteria), and unlike other forms of water-based activity, no review of therapeutic outcomes from scuba diving has been done.

The literature on hydrotherapy suggests that the water environment can be beneficial for people both physically and psychosocially, owing to four properties of water:[5–7]

- Freedom from the effects of gravity on the body

(buoyancy), allowing movement that may be impaired on land;[6]
- the hydrostatic pressure of water can aid in the reduction of oedema;[5]
- as water is more viscous than air, it adds resistance to a moving limb which can help increase strength;[7]
- a typical therapy pool is usually between 32–33°C, and this warm environment allows for greater muscle relaxation and a decrease in tone.[8]

It is unknown to what extent these qualities may apply to scuba diving, particularly considering potential differences such as decreased water temperature and increased hydrostatic pressure. However, even at the lower temperatures typical of diving environments, there is evidence of physical benefits such as a decrease in plasma renin activity, plasma cortisol concentrations, heart rate and blood pressure.[9] In addition to the physical effects of water environments, there is a potential for psychosocial benefits, with one theory, 'blue mind',[10] positing that being in or around water improves brain chemistry and decreases stress levels, ultimately improving mental health. Such factors as cold, increased pressure, hyperoxia and the formation of intravascular bubbles all potentially contribute to increase oxidative stress.[11,12]

The aim of this review was to explore all available research on the physical and psychosocial effects of scuba diving for individuals with neurological disability, autism and intellectual disabilities.

Method

The review protocol was registered with PROSPERO (ID number CRD42019131724) and followed the preferred reporting items for systematic reviews and meta-analyses (PRISMA) statement 27-item checklist.[13]

INCLUSION AND EXCLUSION CRITERIA

The modified PICO (population, intervention, comparator and outcome), PIO (population, intervention and outcome) format was used in the formation of the question and the inclusion/exclusion criterion, as there was no comparator in this review.

The population group included in this review was participants of any age or gender with neurological disabilities, intellectual disabilities or autism. As such, musculoskeletal conditions and other disabilities (not defined as those above) were excluded. Scuba diving-based interventions were considered for inclusion in this review. The intervention must have utilised scuba equipment in any water setting (pool, ocean). Both recreational and therapeutic focused scuba interventions were considered. Other water-based interventions that did not use scuba equipment were excluded. All types of physical and psychosocial outcomes were explored. Papers were excluded if they did not explore intervention outcomes. Only papers written in English (full text) were included. No publication date or publication status restrictions were imposed. Papers could include other disability groups or participants without a disability as long as data for the disabilities of interest (neurological disability, intellectual disability and autism) could be extracted separately.

SEARCH STRATEGY

The initial search was completed on 14 June 2018 and was updated on 05 May 2020. MEDLINE, EMBASE, Ovid Emcare, and SportDiscus online databases were searched, with the following search terms from MEDLINE: 'Disabled Persons/', 'disabil*', 'handicapped', 'Hydrotherapy/', 'hydrotherap* or hydro-therap* or aqua therap* or water therap* or water based or water exercise*', 'immersion* or submersion*', and 'diving or swimming or scuba or snorkel*'. The broader terms were used to ensure no papers were missed owing to alternative descriptions (e.g., swimming or hydrotherapy), and all non-scuba interventions were excluded. The key search terms from above were altered to suit each database, ensuring that as many papers were captured as possible. The above search was completed on the same day for all four databases, ensuring that the information gathered were within the same date restriction.

STUDY SELECTION AND SCREENING

Peer-reviewed literature was imported into EndNote X9.2 (Clarivate, Philadelphia, USA) and then screened through Covidence (Veritas Health Innovation, Melbourne, Australia), with two reviewers completing the abstract, full text and extraction phases (principal investigator and colleague). Any disagreements between the two reviewers were resolved through face-to-face discussion.

QUALITY ASSESSMENT

The McMaster Critical Appraisal tools (quantitative and qualitative) were used for quality assessment.[14] The appraisal tools had extended responses and yes/no questions (yes receiving 1 point, N/A or No receiving 0 points) helping to determine the quality of the papers. Quantitative papers received a total score out of 14, while qualitative papers were scored out of 22. The quality assessment tool was completed by both reviewers and compared for discrepancies with any disagreement resolved through face-to-face discussion. Finally, included papers were categorised into an evidence hierarchy as determined by study design, degree of bias and subsequent level of evidence.[15]

DATA EXTRACTION

Data were extracted into a purpose-designed Excel® spreadsheet (Microsoft Office 365, Redmond, USA) by both reviewers. From this, a comparison between the information extracted was conducted to ensure consistency. There was

a 100% agreement rate between reviews. If there had been any disagreements, these would have been resolved by face-to-face discussion with a third reviewer being involved if needed. The information extracted included author, year, country of origin, participant details (including, age, gender and condition), intervention details (description and length of time participants were involved) and study design.

DATA SYNTHESIS

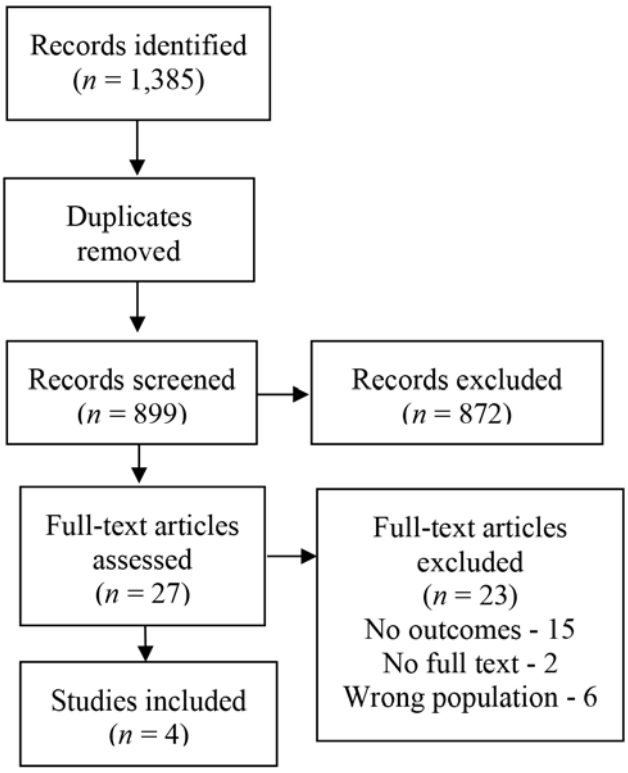
It was predicted from preliminary literature searches, that there would be a low number of included papers; thus, there was no minimum number required for this review. The included papers were diverse in design and quality. Therefore, a narrative synthesis was conducted to summarise findings. An exploration of the similarities and differences of the interventions, outcome measures and the results was carried out. The narrative synthesis follows the four elements outlined by the Centre for Reviews and Dissemination:[16] theory development; preliminary synthesis of included papers; exploring relationships between papers and assessing the robustness of the synthesis.

Results

INCLUDED PAPERS

A total of 1,562 papers were screened in this review of which only four met the inclusion criteria (Figure 1). Therefore, owing to the limited number of papers and the lack of any reported quantitative data or consistency in outcomes, a meta-analysis/meta-synthesis was not completed.

Figure 1
PRISMA flow chart of included papers



OUTCOMES

Sample size for the four papers varied from two to 23 participants, with a range of conditions included. Only one paper mentioned safety or medical guidelines, reporting that all participants underwent a medical screening that was "*conducted to a standard described by Edmonds, Lowry and Pennefather and accepted by the Royal Australian Naval School of Underwater Medicine*."[17] Details provided from the medical screening were minimal, though it was noted that special consideration was needed depending on the disability type and severity. None of the papers reported physical outcomes; however, all four included psychosocial measures, with only descriptive statistics being used in the reporting of results.

Study one,[17] from Australia, included case studies of 16 participants. Out of these 16 participants, only eight met the inclusion criteria (and will be the sole focus of this discussion), four with acquired brain injury, two with a spinal cord injury, one with spina bifida and one with post-polio syndrome. All eight participants were male with a mean age of 22.3 years. Participants, new to scuba, were trained to dive. The course was completed over a nine-month period, including 40 hours of theory, 40 hours of training in a pool, and 138 hours of training in the open sea. Psychosocial measures included anxiety levels through the State-Trait Anxiety Inventory, psychophysiological disturbance screened through the Cornell Medical Index and self-concept measured through the Tennessee Self-Concept Scale (TSCS). The paper's focus was on learning how best to safely teach people with disabilities to scuba dive, so minimal effects were reported. However, it was reported that physical self-concept improved for six out of eight completing participants.[17]

Study two,[18] from the USA, involved two groups of divers in a cross-sectional survey. In a group of seven (one female and six males, mean age 35.1 years, with one to three years of diving experience) divers with disabilities, four had a spinal cord injury, two had cerebral palsy and one an intellectual disability. The second group consisted of eight divers without disabilities from a recreational dive club, selected to match the level of experience of the divers with disabilities. The paper explored the motivation of people with and without a disability to participate in scuba diving through the modified version of the Participation Reasons Scale. Both groups reported similar motivations. The excitement of the activity and participants wishing to play and have fun were reported as the most important reasons for participation for divers with disabilities, with the least important reasons including the activity pleasing others close to the participant

and participants feeling needed and wanted by others. For divers without a disability, they rated similar reasons around excitement and having fun, but the third ranking was to relieve tension, whereas divers with disabilities rated this much lower, ranking it twentieth.[18]

Study three,[19] from Canada, interviewed two men aged 33 and 47 years with spinal cord injuries, with one year and less than one year of diving experience respectively, who were recruited from a recreational dive club. Semi-structured interviews into the experience of scuba diving as a person living with a disability were conducted. The following themes emerged from the interviews: Freedom from impairment (feeling equal to divers without a disability in the water); enhancement of social experiences (meeting new people, interacting with others both with and without a disability); enhancement of self-concept (sense of achievement, increased control and independence) and optimal experience (enjoyment of the activity and environment).[19]

Study four,[20] from Italy, involved 23 participants with no previous diving experience (seven female and 16 male, aged 16 to 30 years), three with autism spectrum disorder, four with intellectual disability, one with spinal cord injury and 14 with Down syndrome. Participants underwent a specific scuba training course delivered by Disabled Divers International (DDI) with divers obtaining a qualification that allows the diver to "*perform recreational dives, within the limits of the certification obtained, in the seas around the world.*"[20] The paper explored the cognitive effects of a specifically designed scuba diving teaching method (by DDI) using the Nepsy II battery.[21] A 'general improvement' in visual attention and understanding instruction from being involved in the DDI method was reported.[20]

Of the four papers, studies two and four were assessed as low quality,[18,20] and studies One and Three as of moderate quality.[17,19] On the McMaster appraisal tools, the three quantitative studies[17,18,20] were rated as level 5 on the hierarchy of evidence,[17] with the study designs including a multiple case study and a cross-sectional investigation. study three,[19] which included a phenomenological study scored 13/22 on the quality tool. Because of the generally low quality of the data, the detailed scores are not presented here but are available from the author on request.

Discussion

This systematic review is the first that the authors are aware of to collate research on scuba interventions for people with neurological and neurodevelopmental disability. Overall, there is a lack of research in this area, as well as only limited data provided in the few papers identified in the search. Scuba interventions for people living with disabilities were reported as being enjoyable and promoted an increase in self-concept. However, the four studies identified only provide relatively weak evidence with which to determine the psychosocial effects of scuba interventions for the included population.

Self-concept as an outcome was reported in studies one and three.[17,19] It is suggested that self-concept is lower for people with disabilities than for people without disabilities.[22,23] As self-concept directly affects mental health and well-being,[23] increases in self-concept can highlight the abilities of the person and improve overall health and wellbeing. The overall self-concept of people with disabilities can be raised by instruction in scuba diving, but the long-term effects are unknown.[17]

In study three, the two divers with spinal cord injuries expressed that they felt 'in control' of their bodies and had a new-found confidence in themselves.[19] Additionally, it was suggested that they experienced feelings of achievement and competency not only from the activity itself but from their ability to adapt to the complex environment of the ocean.[19] Several psychosocial benefits were identified, including an enhancement of social experiences, freedom from impairment and equality with other divers. Some of these positive experiences stemmed from meeting and interacting with divers both with and without a disability and working in buddy teams during dives. Additionally, the "*psychology of optimal experience*" may be used to assist with understanding their responses.[19] 'Optimal experience' is defined as an occasion "*where we feel a sense of exhilaration, a deep sense of enjoyment, which we cherish*".[24] It is suggested that scuba interventions provide this deep sense of enjoyment and the experience is cherished.

The findings from study four suggest improved cognition, in particular, visual attention and understanding instruction, for participants with an intellectual disability learning to dive.[20] Whether the specific teaching technique (DDI) could be applied to skills other than scuba, is unknown; nonetheless, an increase in visual attention and the ability to understand instructions may translate into learning new skills for daily life.

There are both similarities and differences when comparing the motivation to participate in divers with and without disabilities. Divers without disabilities ranked the relief of tension much higher (third) than people with disabilities (ranked twentieth). Divers without disabilities appear to use scuba to escape and relax both mentally and physically. This does not appear to be the same focus for divers with a disability, with statements regarding the challenge and the test of physical skills ranked higher by these individuals. Divers with a disability appear to enjoy the challenge of diving, exploring a different environment and movements that they may not have thought possible.[18]

A limitation of this study was the exclusion of non-English papers and the grey literature. Also, as this review focuses only on participants with neurological disability, intellectual disability and autism, this may have limited the number of

included papers. More research is needed, including other groups, such as those with musculoskeletal conditions that limit muscle strength, mobility or physical function and those with limited cardiovascular fitness. Exploration into pain, spasticity and mood may also provide valuable insights. Additionally, the scuba interventions described in two of the studies,[17,20] were completed over prolonged periods of time with extraordinary training measures in comparison to mainstream open water diver training. They are also relatively elaborate in the context of therapeutic or recreational programmes for people with a disability. As such it is important that future research in scuba-based interventions should also consider the burden and difficulty of providing/delivering these interventions.

Finally, since the completion of this review, one additional paper has been discovered that would meet the requirements to be included.[25] The paper explored thermal balance of divers with and without a spinal cord injury (SCI) in open water diving. It was found that divers with SCI were unable to maintain gastrointestinal temperature during short, shallow dives in 6°C water and their temperatures fell further post-dive. Overall, the results of this study do not change the overall conclusions of this paper, but do provide valuable information regarding the safety of open water diving in colder temperatures for people with an SCI.

Conclusion

Scuba diving for people with neurological disability, intellectual disability and autism appears to be a fun and enjoyable experience, increasing a person's self-concept. However, there is limited research on scuba interventions for these population groups. Future research direction should explore both the physical and psychosocial effects of scuba interventions, using robust study designs.

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Flying after diving: a questionnaire-based evaluation of pre-flight diving behaviour in a recreational diving cohort

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Key words
Altitude; Decompression sickness; Guidelines; Health surveys; Surface interval; Vacation

Abstract
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Introduction: Divers are recommended to observe a pre-flight surface interval (PFSI) ≥ 24 hours before boarding a plane following a diving vacation. Decompression sickness (DCS) symptoms may occur during or post-flight. This study aimed to examine the adherence of PFSI ≥ 24 in vacationing divers, and if any perceived signs and symptoms of DCS during or after flight were experienced.
Methods: An anonymous online survey was publicised through diving exhibitions and social media. Data included diver/diving demographics, PFSI before flight, flight details, and perceived signs and symptoms of DCS during or after flight.
Results: Data from 316 divers were examined (31% female) with the age range 17–75 years (median 49). Divers recorded 4,356 dives in the week preceding the flight, range 1–36 (median 14). Overall, 251/316 (79%) respondents reported a PFSI of ≥ 24 hours. PFSIs of < 12 hours were reported by 6 respondents. Diagnosed and treated DCS developing during, and post flight was reported by 4 divers with PFSIs ≥ 24 hours and by 2 divers with PFSIs < 24 hours. Fifteen divers boarded a plane with perceived symptoms of DCS.
Conclusions: These data suggest that most divers in this study observed the recommendations of a ≥ 24 hour PFSI with safe outcomes.

Introduction

Commercial air travel exposes a passenger to decreased atmospheric pressure, reduced partial pressure of oxygen and reduced air humidity. Passenger aircraft typically fly at 11,000 to 12,000 metres (m) altitude with a cabin pressure maintained at an equivalent maximum altitude of 2,400 m. At 2,400 m the cabin pressure of 565 mmHg is ~25% lower than at sea level (760 mmHg) and alveolar oxygen tension is reduced by 25% leading to a reduction in arterial PO_2 from 100 mmHg (sea level) to approximately 55 mmHg. Relative humidity at cruising altitude is generally 10–15%, compared to 50–60% in the average home.[1] Decreased pressure, relative hypoxia and potential for dehydration experienced during commercial flight are all known risk factors for decompression sickness (DCS).[2] The time between surfacing from a dive and flying – the Pre-Flight Surface Interval (PFSI) – is important for diving safety. The most common recommended PFSI for recreational divers is 24 hours, with most other recommendations falling within a window of 18–48 hours.[3–9]

Divers may board a plane when symptoms of DCS are already evident, and symptoms may occur or be exacerbated during or after a flight. In severe cases in-flight DCS can result in aircraft diversion to expedite recompression.[1,8–12]

Experimental studies investigating safe PFSIs after diving utilised differing controlled environments and thus excluded real life confounding vacation factors such as alcohol, heat and over-exertion, all of which may contribute to the risk of developing DCS.[13–17]

Studies have indicated that 24 hours can be regarded as a safe and effective PFSI for all divers, though there have been possible indications a 24-hour PFSI may not be appropriate for a small number of divers who might be more pre-disposed to bubbling than others.[18,19] With the popularity of overseas dive vacations requiring commercial flights this study sought to explore the compliance of divers to the PFSI of ≥ 24 hours recommendation, and the hypothesis that a PFSI of ≥ 24 hours is sufficient to prevent the development of symptoms of DCS.

Methods

An anonymous online survey was available for completion from June to December 2016 and publicised through the

DDRC Healthcare website and social media platforms, Dive 2016 (22–23 October), UK diving magazines, Divernet, and Deeper Blue websites. All data were anonymous, deduplicated and analysed using descriptive statistics. Approval from a research ethics committee is not required for studies of this type in the UK.

Results

A total of 316 responses (69% male, 31% female) aged from 17–75 years, (median 49, IQR 17) were received from divers who had flown after diving. Individual lifetime dives reported ranged from 8–15,000 (median 450, IQR 922). Dives reported in the last 12 months ranged from 1–500 (median 40, IQR 50) and totalled 17,793 dives. The maximum depth ever dived ranged from 16 to 198 metres of sea water (msw) (median 48, IQR 16).

The number of dives in the week preceding a flight ranged from 1 to 36, (median 14, IQR 11), with the median depths of the last two dives being 25 msw (IQR 10) and 20 msw (IQR 12) respectively. Multi-dive days were reported by 291 (92%) respondents, whilst 25 (8%) respondents reported only diving once a day (single dive days). Consecutive days dived without a break ranged from 2 to 28 (median 6, IQR 1).

PFSIs less than 24 hours (1 x 4 hours, 5 x 8 hours, 13 x 12 hours, 33 x 18 hours) were reported by 52 (16%) respondents, whereas 251 (79%) divers reported PFSI of 24 hours or more, the maximum being 36 hours. No interpretable PFSI data were reported by 13 divers.

Respondents with perceived symptoms consistent with DCS reported limb or joint pain, headache, dizziness or disorientation, visual disturbance, inappropriate weakness/fatigue, difficulty speaking, skin itching, tingling or rash, loss of sensation and/or numbness, problems with thinking, memory or performance, chest pain, and partial paralysis.

Eighteen divers (10 males and eight females) reported onset of symptoms during the flight or post-flight (Figure 1). Details are shown in Table 1 (during flight) and Table 2 (after flight). There was no relationship between divers with or without symptoms and age (Mann-Whitney U test $P = 0.896$), the number of dives in the preceding week (Mann-Whitney U test $P = 0.880$), maximum depth of last dive (Mann-Whitney U test $P = 0.377$), or the surface interval between the last two dives (Mann-Whitney $P = 0.728$). Fourteen divers in this group recorded PFSI ≥ 24 hours (Tables 1 and 2) Additionally, there were no differences in these 18 divers between the sexes with or without symptoms (Fisher's exact $P = 0.200$). Six divers were treated in a hyperbaric chamber with two reporting residual symptoms. None of the 18 divers discussed their symptoms with the aircrew, with five divers not telling anyone about their symptoms at all.

Fifteen divers boarded a plane when already experiencing symptoms of DCS. Neurological symptoms were most frequently reported, with some boarding the plane with more than one symptom. Symptoms were generally exacerbated in flight possibly caused by flight conditions or further evolution of the original disease. In nine of the 15 divers who developed additional symptoms during and/or after the flight, five were treated in a hyperbaric chamber with two reporting mild residual symptoms (Table 3). None of the participants reported symptoms to the flight crew or sought any medical advice.

Discussion

A diving vacation may not only expose a diver to multi-dive, multi-day diving but potentially also a range of other risk

Figure 1

Symptoms reported by 18 respondents either during or after flight who boarded a plane asymptomatically; some respondents reported more than one symptom

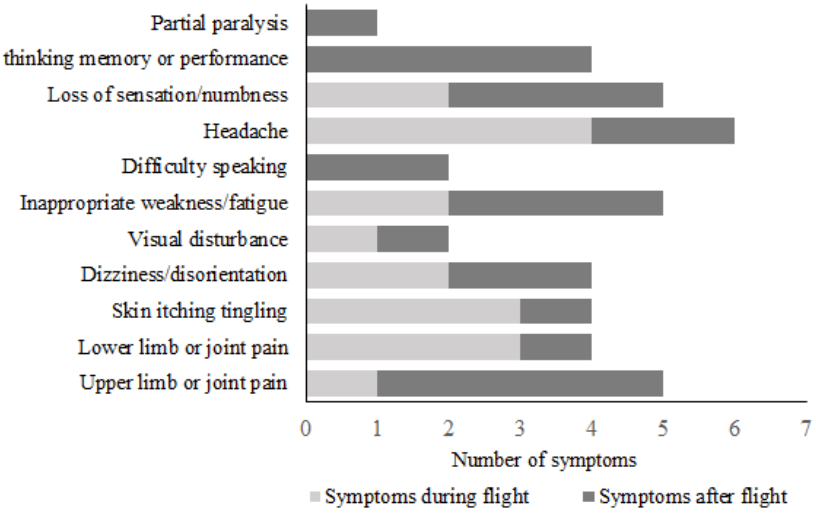

Table 1
Respondents with symptom onset during flight (n = 9); h – hours; min – minutes; PFSI – pre-flight surface interval

Sex, Age (years)	PFSI and flight	Symptoms during flying	Treated in chamber	Symptoms resolved?	Diving demographics Depths are maximum depth of dive
Male, 52	> 24 h. Direct flight 5:30 h	Dizziness and disorientation	Yes	Yes	Twenty dives in the last week, last two dives 18 and 12 msw, each dive 60 min, no stops, open circuit, air
Male, 31	18 h. Direct flight 1 h	Headache, dizziness and disorientation, visual disturbance, inappropriate weakness	Yes	Yes	One single dive to 32 msw for 33 min, 8 min at 5 msw extended safety stop because of 15m·min^{-1} ascent, open circuit, air, cold water dive
Female, 41	> 24 h. Direct flight 5 h	Upper and lower limb or joint pain, skin itching, loss of sensation. Skin rash after the flight	Yes	No, mild residual symptoms, not specified	Twelve dives in the last week, last two dives 30 msw for 28 min, and 13 msw for 47 min, 3 min at 6 msw both dives, open circuit, air
Female, 26	12 h. Direct flight 6 h	Lower limb or joint pain, headache, inappropriate weakness, skin itching. Dizziness or disorientation after the flight	Yes	Yes	Single dive days, 3 dives in the last week, last two dives, 40 msw for 40 min, and 15 msw to 60 min, no deco details, type of equipment used unclear, technical diver
Male, 62	> 24 h. Direct flight 6 h	Headache, during and after the flight	No	Yes	Six consecutive days, 24 dives in last week, last two dives, 23 msw for 60 min, and 17 msw for 58 min, no deco recorded, open circuit, nitrox
Female, 36	> 24 h. Direct flight 13 h	Loss of sensation	No	Yes	Twelve consecutive days, 14 dives in the last week, last two dives, both 25 msw for 75 min, both with 3 min at 5 msw, open circuit, nitrox
Male, 61	> 24 h. Direct flight 5 h	Lower limb or joint pain	No	Yes	Six consecutive days, 18 dives in the last week, last two dives, 28 msw for 45 min, and 27 msw for 45 min, both with 3 min at 5 msw, open circuit, air
Male, 24	24 h. Direct flight 3:30 h	Skin itching. Skin rash after the flight	No	Yes	Three consecutive days, 6 dives in the last week, last two dives, 22 msw for 30 min, and 30 msw for 18 min, no deco both dives, open circuit, air
Female, 62	> 24 h. Direct flight 4 h	Headache during and after the flight	No	Yes	Six consecutive days, 24 dives in the last week, last two dives, 10 msw for 50 min, and 6 msw for 40 min, open circuit, air

Table 2

Respondents with symptom onset after flight ($n = 9$); h – hours; min – minutes; PFSI – pre-flight surface interval

Sex, Age (years)	PFSI and flight	Symptoms after flying	Treated in chamber	Symptoms resolved?	Diving demographics Depths are maximum depth of dive
Male, 50	> 24 h. Direct flight 6 h	Headache, dizziness or disorientation, visual disturbance, difficulty speaking, loss of sensation, problems thinking, partial paralysis	Yes	No, mild residual symptoms, not specified	Five consecutive days, 13 dives in the last week, last two dives, 83 msw for 9 min, no deco details given, and 40 msw for 45 min with 12 min at 10 msw, open circuit, air
Male, 53	> 24 h. Direct flight 9 h	Upper limb or joint pain	Yes	Yes	Three consecutive days, 12 dives in the last week, last two dives, 28 msw for 40 min, and 12 msw for 30 min, both dives with stop for 3 min at 6 msw, open circuit, nitrox
Female, 51	> 24 h. Flight with changes, 7 h plus	Dizziness or disorientation, inappropriate weakness, difficulty speaking, skin itching	No	Yes	Seven consecutive days, 21 dives in the last week, last two dives, 28 msw for 59 min, and 23 msw for 55 min, no deco details given, open circuit, nitrox
Female, 43	> 24 h. Direct flight 5 h	Upper limb or joint pain	No	Yes	Ten consecutive days, 21 dives in the last week, no dive details of the last two dives provided, open circuit, air
Male, 40	> 24 h. Direct flight 5 h	Upper limb or joint pain, dizziness or disorientation	No	Yes	Two consecutive days, 7 dives in last week, last two dives, 32 msw for 60 min with 6 min deco, closed circuit, and 45 msw for 85 min with 15 min deco, open circuit, dive 2 gas 21% and 50% O_2, technical diver
Male, 54	18 h. Flight with changes 17 h	Headache	No	Yes	Ten dives in the last week, last two dives,15 msw for 40 min, and 16 msw for 40 min, stops on both dives 3 min at 6 msw, open circuit, air
Female, 56	18 h. Direct flight 6 h	Upper limb or joint pain, inappropriate weakness, loss of sensation, problems thinking	No	Yes	Six consecutive days, 9 dives in the last week, last dive, 18 msw for 70 min, open circuit, nitrox
Male, 66	> 24 h. 2 flight changes 6:30 h, 7:30 h	Inappropriate weakness or fatigue	No	No, mild residual problems, not specified	Five consecutive days, 11 dives in the last week, last two dives, 20 msw for 58 min, and 12 msw for 75 min, both dives with 3 min at 5 msw, open circuit, air
Female, 62	> 24 h. 4 flight changes, 2:20 h, 3:15 h, 12:30 h, 1 h	Lower limb or joint pain, problems thinking or performance	No	Yes	Seven consecutive days, 23 dives in the last week, last two dives, 14 msw for 63 min, and 28 msw for 70 min, both dives 3 min at 5 msw, open circuit, nitrox

Table 3

Respondents with symptoms before flight and treated for DCS after flight (n = 5); * Mild residual symptoms post treatment, detail not specified; h – hours; min –minutes; PFSI – pre-flight surface interval

Sex, Age (years)	**PFSI and flight**	**Symptoms before flight**	**Treated in chamber**	**What were the reasons for your symptoms?**	**Diving demographics Depths are maximum depth of dive**
Male, 55	24 h. Direct flight 4 h	Upper limb pain, continued during flight	Yes	"*A bend, other divers also thought it was a bend*"	Five consecutive days, 12 dives in the last week, last two dives 30 msw for 45 min with deco 9 msw for 10 min and 3 msw for 9 min. 20 msw for 45 min with deco 9 msw for 15 min and 3msw for 11 min, open circuit, air
Female, 49	24 h. Direct flight 4 h	Headache, dizziness, continued during flight	Yes	"*Carbon monoxide poisoning*"	Six consecutive days, 20 dives in the last week, last two dives 24 msw for 60 min, and 15 msw for 45 min, both dives with "*5 msw safety stop*", open circuit, air
Female, 47	24 h. Indirect flight	Headache, dizziness, inappropriate weakness. Loss of sensation during flight, difficulty speaking and thinking after flight	Yes *	"*Buddy, divers, and doctor put the symptoms down to migraine and seasickness*"	Five consecutive days, 17 dives in the last week, last dive 15 msw with no further details, open circuit, air
Male, 51	> 24 h. Indirect flight	Skin rash. Headache during and after flight	Yes	"*Dehydration, which brought on a cerebral bend*"	Three consecutive days, 6 dives in last week, last two dives 27 msw for 46 min, and 23 msw for 50 min, deco both dives "*3 min safety stop*", open circuit, air
Female, 17	> 24 h. Direct flight 3:30 h	Inappropriate weakness, skin rash. Upper limb pain during the flight. Headache, dizziness, and problems thinking after flight	Yes *	"*Fatigue, back pain due to bad posture, nausea due to traveling*"	Fourteen consecutive days, 16 dives in the last week, last two dives 15 msw for 50 min with 5 min at 5 msw safety stop, and 4 msw for 50 min no deco needed, open circuit, nitrox

factors for developing DCS. Studies from which the current guidelines for flying after diving are derived are not always able to account for confounding factors frequently associated with vacation diving.[10–16]

This study reported a range of PFSIs from 4 hours to more than 36 hours, though the majority (79%) of respondents waited ≥ 24 hours before boarding a flight. Despite observing some noticeably short PFSIs most divers did not suffer any symptoms of DCS, whilst interestingly, most divers who reported perceived symptoms had observed a ≥ 24 hour PFSI. Notwithstanding the possibility that not all the symptoms reported might have been diagnosed as DCS, it is worth noting that the divers treated in a chamber and those with residual symptoms had all observed a PFSI of ≥ 24 hours. However, it could be argued that some divers in this group may have extended their PFSI to > 24 hours due to feeling of uncertainty concerning their health. While most of the divers in this study had undertaken multi-dive days and/or consecutive dive days, one respondent reported one single dive to 32 msw, a PFSI of 18 hours, a 15 msw·minute^{-1} ascent, and subsequently developed DCS symptoms during flight and was diagnosed and treated in a chamber on return to the UK.

PFSI guidelines for recreational divers need to be straightforward. Ideally, they need to be compatible with commercially driven elements of a dive vacation. Significantly increasing the recommended PFSI may represent a marginal increase in safety but could reduce the viability of diving vacations if an extended period were to be occupied by observing a long PFSI at the expense of dive frequency and opportunities. Additionally, our study did not request details regarding any "*complete non-diving days*" taken, or not, during the dive vacation that may have impacted any outcome involving DCS. A balance is required, and our data generally support 24 hours being a pragmatic PFSI with a significantly reduced risk of developing DCS after take-off.

An interesting observation was that none of the divers who reported symptoms consulted the flight crew to establish if supplementary oxygen was available. Also recognised in previous studies, is the boarding of planes with symptoms of DCS already developing.[11,12] Data in our study showed pre-existing symptoms were exacerbated by the flight, with increased reports of neurological symptoms post flight. The financial impact of revealing symptoms of DCS before a flight may play a defining role, as may the multitude of well-documented reasons why under-reporting is prevalent for any illness. Some divers may not take out adequate insurance or could be bound by work/employment commitments to return home not appreciating the possible severity of the consequences of failing to seek appropriate medical help.[20]

An additional factor for non-treatment may be a lack of appropriate medical or recompression facilities, with the best option being to fly elsewhere for management. However, this should be done after diving medical consultation.

LIMITATIONS

This study relied on logbook entries and recall, with the recall element being a significant weakness with possible bias, and the logbook detail not always complete in some instances. All anonymous on-line studies have issues with bias and misreporting, coupled with an inability to follow up records of interest. A self-selection bias is also likely with divers in this study responding if they felt they had something of note to report, resulting in 10% of the respondents reporting perceived symptoms of DCS; though not all were diagnosed and treated as such. However, studies of this type are justified in allowing researchers to gather data that may otherwise remain unreported.

Conclusion

The data presented in this study demonstrate that UK sport divers undertake multi-dive, multi-day diving vacations and then board a flight after observing a wide range of PFSIs. Most divers observe a PFSI ≥ 24 hours and safely fly without developing DCS. Some divers fail to adhere to minimum recommended PFSIs and some will board a flight despite experiencing symptoms of DCS. The reasons for this behaviour are likely complex and beyond the scope of this study. A small proportion of those observing a minimum PFSI of 24 hours develop symptoms of DCS which are generally mild and may simply represent slow progression of existing disease or may represent exacerbated effects from the reduced pressure environment in an aircraft. The confounding factors that cause this subgroup to be susceptible to developing DCS remain the subject of conjecture and as such vigilance for symptoms should be exhibited by all commercial airline passengers after scuba diving despite the risk being small if a 24-hour PFSI is observed.

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The lower limit for FEV_1/FVC in dive medical assessments: a retrospective study

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Key words
Fitness to dive; Health surveillance; Lung function; Military diving

Abstract
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Introduction: Interpreting pulmonary function test (PFT) results requires a valid reference set and a cut-off differentiating pathological from physiological pulmonary function; the lower limit of normal (LLN). However, in diving medicine it is unclear whether an LLN of 2.5% (LLN-2.5) or 5% (LLN-5) in healthy subjects constitutes an appropriate cut-off.
Methods: All PFTs performed at the Royal Netherlands Navy Diving Medical Centre between 1 January 2015 and 1 January 2021 resulting in a forced vital capacity (FVC), forced expiratory volume in one second (FEV_1) and/or FEV_1/FVC with a Z-score between -1.64 (LLN-5) and -1.96 (LLN-2.5) were included. Records were screened for additional tests, referral to a pulmonary specialist, results of radiological imaging, and fitness to dive.
Results: Analysis of 2,108 assessments in 814 subjects showed that 83 subjects, 74 men and nine women, mean age 32.4 (SD 8.2) years and height 182 (7.0) cm, had an FVC, FEV_1 and/or FEV_1/FVC with Z-scores between -1.64 and -1.96. Of these 83 subjects, 35 (42%) underwent additional tests, 77 (93%) were referred to a pulmonary specialist and 31 (37%) underwent high-resolution CT-imaging. Ten subjects (12%) were declared 'unfit to dive' for various reasons. Information from their medical history could have identified these individuals.
Conclusions: Use of LLN-2.5 rather than LLN-5 for FEV_1/FVC in asymptomatic individuals reduces additional investigations and referrals to a pulmonary specialist without missing important diagnoses, provided a thorough medical history is taken. Adoption of LLN-2.5 could save resources spent on diving medical assessments and protect subjects from harmful side effects associated with additional investigations, while maintaining an equal level of safety.

Introduction

Fitness to dive assessments are performed to identify potential medical risks that can lead to harm in diving.[1] Despite differences in fitness required for recreational, commercial and military diving, evaluating the pulmonary tract is paramount in these assessments, as the lungs and airways must adapt significantly to submersion.[2] Pulmonary function tests (PFTs), especially spirometry, are used frequently to evaluate pulmonary status.[1,3,4]

Because pulmonary functions are biological variables with significant variability, a valid set of reference values is necessary to separate physiologic from pathologic pulmonary function.[4,5] The acceptable lower limit of normal (LLN) in asymptomatic individuals has varied over the years and among reference sets.[6] Traditionally, fitness to dive standards have suggested that LLN be a percentage of a predicted value based on sex, age and height. This LLN was usually defined as between 70% and 80% of predicted value, as was common for pulmonary medicine.[7] With the introduction of the Global Lung Initiative (GLI-2012), spirometry results are regarded as normally distributed parameters, with the results for each individual represented as Z-scores.[8] Z-scores have been found to better represent pulmonary status than percentages of predicted values.[8,9] Several studies have shown that defining the lower limit of normal (LLN) as a fixed ratio leads to extensive under- and over diagnosing in younger and older adults respectively.[9,10]

Despite the use of more reliable reference sets, such as the GLI-2012, the cut-off for LLN in fitness to dive assessments aiming to differentiate normal variations from potentially harmful findings remains unclear, especially in the absence of pulmonary complaints.[5] Pulmonologists have regarded the lowest 2.5% of the population (LLN-2.5), equivalent to a Z-score of -1.96 or lower, as potentially pathologic in asymptomatic individuals. By contrast, the Royal Netherlands Navy has adopted a more conservative cut-off, using the lowest 5% of the population (LLN-5), equivalent to a Z-score of -1.64 or lower, as the lower limit when transitioning from the European Respiratory Society

(ERS-1993) to the GLI-2012 dataset.[11] To determine whether LLNs below these cut-offs are potentially pathological, subjects must undergo additional testing, such as full-body plethysmography to determine the static lung volumes, bronchial challenge tests to exclude bronchial hyperreactivity and/or referral to a pulmonary specialist for additional tests like radiological imaging such as high-resolution computerized tomography (HRCT) or testing for dynamic hyperinflation.

This study was designed to determine the effect of using LLN-5 or LLN-2.5 as the cut-off value for PFTs in asymptomatic divers. We hypothesised that lowering the LLN to LLN-2.5 would not result in missing relevant diagnoses in fitness to dive assessments.

Methods

The Royal Netherlands Navy Diving Medical Centre medically assesses divers, submariners and inside chamber tender personnel (i.e., hyperbaric physicians and nurses) annually. All fitness to dive assessments were performed according to European Diving Technology Committee (EDTC) guidelines, except that PFT results have been interpreted relative to the GLI-2012 reference set beginning on 01 January 2015.[3,11] All data were stored in an electronic medical database.

All medical assessments of military divers between 01 January 2015 and 01 January 2021 showing a forced vital capacity (FVC), forced expiratory volume in one second (FEV_1) and/or FEV1/FVC with a Z-score between -1.64 (LLN-5) and -1.96 (LLN-2.5) were included in the analysis. Age, sex, ethnicity, height and smoking status were recorded. Medical assessments were evaluated to determine whether a subject had undergone additional testing at the Diving Medical Centre or was referred to a military pulmonary specialist at the Central Military Hospital (Utrecht, the Netherlands). Other parameters recorded including the results of radiological imaging and whether the subject was declared fit or unfit to dive.

According to national law and legislation, retrospective analyses are not required to be evaluated by a medical ethics committee. The methods used to handle personal details and privacy were in agreement with the guidelines of the Association of Universities in the Netherlands and the Declaration of Helsinki.

Results

During the 6-year study period, 814 subjects underwent 2,108 medical assessments. Of these 814 subjects, 83 (10%) had PFTs lower than LLN-5 but higher than LLN-2.5 (i.e., Z-scores between -1.96 and -1.64). These Z-scores may represent FVC, FEV_1, FEV_1/FVC-ratio or combinations of these variables. These 83 subjects included 74 (89%) men and nine (11%) women, of mean age 32.4 (SD 8.2) years and height 182 (7.0) cm. These results, as well as the PFT results, are shown in Table 1. None of the FVC and only four FEV_1 values were lower than LLN-5 (Z-score range FVC: -1.44 to +2.87, FEV_1: -2.25 to +1.27). Thus, the predominant variable with a Z-score in the targeted range was FEV_1/FVC. To give our population more context: 49 subjects of the total population (6%) had a FEV_1/FVC lower than LLN-2.5 (Z-score range -2.92 to -1.97, mean -2.33), with FVC ranging from -1.33 to 2.86 (mean 0.87) and FEV_1 from -2.8 to 1.04 (mean -0.92).

Of the 83 subjects with Z-scores between -1.64 and -1.96, 35 (42%) underwent additional investigations at the Diving Medical Centre (specified in Table 2) and 77 (93%) were referred to a pulmonary specialist in the military hospital, with 31 (37%) undergoing high-resolution computerised tomography (HRCT). Some of the included subjects may have undergone additional evaluations at the Diving Medical Centre before being referred to a pulmonary specialist.

In total, 10 (12%) of the subjects did not meet the criteria for 'fit to dive'. Four of these subjects chose not to pursue a diving career after having failed to meet the PFT requirements (all had a FEV_1/FVC between LLN-5 and LLN-2.5) on their first assessment. The other six subjects were declared 'unfit to dive' after additional testing. Two

Table 1
Baseline characteristics of 83 study subjects; data are mean (SD). FVC – forced vital capacity; FEV_1 – forced expiratory volume in one second

Parameter	Total (*n* = 83)	Male (*n* = 74)	Female (*n* = 9)
Age (years)	32.4 (8.1)	32.3 (8.1)	33.2 (8.2)
Height (cm)	182 (6.8)	183 (6.6)	175 (4.4)
FVC (L)	6.14 (0.82)	6.32 (0.67)	4.77 (0.56)
FVC (Z-score)	0.75 (0.83)	0.77 (0.82)	0.58 (0.85)
FEV_1 (L)	4.30 (0.55)	4.42 (0.45)	3.41 (0.42)
FEV_1 (Z-score)	-0.52 (0.72)	-0.51 (0.71)	-0.61 (0.72)
FEV_1/FVC (%)	0.70 (0.02)	0.70 (0.02)	0.71 (0.02)
FEV_1/FVC (Z-score)	-1.80 (0.83)	-1.80 (0.82)	-1.75 (0.85)

Table 2
Tests and referrals performed after PFT results in 83 divers with spirometry Z-scores between -1.64 and -1.96

35 (42%) additional investigations at the Diving Medical Centre: - 35 spirometry - 5 end-expiratory lung volume tests during exercise - 2 bronchial challenge tests -2 exercise-induced bronchoconstriction tests
77 (93%) referrals to a pulmonary specialist at the Central Military Hospital: - 77 spirometry - 31 high-resolution computerised tomography • 26 subjects without clinically significant findings • 1 subject with minimal air trapping in secondary lobules, determined by the pulmonary specialist as not being a risk for diving • 1 subject with severe air trapping and a history that included pneumonia and pneumothorax • 3 unexpected findings: schwannoma, haemangioma and paraseptal emphysema.

of these subjects had FEV_1/FVC lower than LLN-2.5 after retesting and were declared medically unfit for diving. Two subjects failed bronchial challenge testing after having met the spirometry requirements at a later stage. One subject showed reversibility on spirometry after testing with salbutamol and was later diagnosed with asthma by a pulmonary specialist. Finally, one subject had a medical history that included pneumonia and traumatic pneumothorax, for which he was referred to a pulmonary specialist, with HRCT showing trapped air. All divers underwent bronchial challenge testing at the start of their diving career and are subjected to both spirometry and exercise tolerance testing annually to meet national legislation requirements.

Discussion

This study indicated that use of LLN-2.5 (Z-score of -1.96) rather than LLN-5 (Z-score of -1.64) as a cut-off point for spirometry would reduce the number of additional tests and referrals to a pulmonary specialist without missing significant diagnoses. Diagnoses that led to disqualification from diving were also identified through history taking or the finding of reversibility on spirometry. Adoption of the LLN-2.5 cut-off would have saved time and resources associated with additional investigations and referral to a pulmonary specialist in 10% of our diving population.

These results are in agreement with the recommendations of the GLI-2012 taskforce for evaluating asymptomatic individuals,[8,10] as well as with a study in aviators showing that using LLN-2.5 rather than LLN-5 reduced the number of referrals significantly.[12] In the latter study, the reduction of referrals was more profound in men aged > 40 years, but was also observed in men aged < 25 years and in women. Because men constituted 89% of our study population, with too few women included for subgroup analysis, we cannot validate these findings for female divers specifically.

A higher-than-expected percentage of our population (10%) fell within the range between LLN-5 and LLN-2.5. By definition this should constitute only 2.5% of the total population We could not find seasonal effects or other explanations for this finding. However, (Dutch) Navy divers cannot be considered a representative sample of the general population due to selection bias in the Armed Forces. With a FVC generally higher than normal and a regular FEV_1, the FEV_1/FVC-ratio tends to be lower. A long-standing military diving career does not seem to negatively affect pulmonary function, although sufficiently powered prospective research is not available.[13,14] Another remarkable observation in this study was the low percentage of clinically significant findings on HRCT, as previous studies have shown that this percentage could be as high as 34%.[15,16] While this could be expected, as PFTs are a functional test and imaging relates to structural changes, this contradicts earlier findings of frequent anomalies on HRCT in dive medical assessments of healthy subjects.[15,16] We feel these findings emphasise the safety of refraining from both additional testing and referral to a pulmonary specialist in subjects with a FEV_1/FVC between LLN-5 and LLN-2.5.

Fewer false-positive PFT results would save time and resources during the process of dive medical assessments, which in our military population reduces the downtime of important operational assets of the Royal Netherlands Navy. Additionally, every additional test may provide findings of unknown clinical significance, which could lead to disqualification of a diver due to legal issues. The findings of this study are in agreement with studies that emphasise the need for more clinically driven assessments of divers rather than a legal approach consisting of annual assessments with mandatory boxes that have to be ticked.[1] For instance, a Dutch Marine who recently broke the world record speed marching (i.e., running a full marathon in military attire and a 18 kg rucksack) still had to participate in an exercise tolerance test for his dive medical assessment to ensure his fitness met the professional standards.[1,17] The argument could be made that this could have been concluded from the history taking alone.

STRENGTHS AND LIMITATIONS

To our knowledge this is the first study that assesses the LLN of PFTs in military divers. PFTs are a cornerstone of dive medical assessments, and, due to the ongoing COVID-19 pandemic, are likely to remain of great value. This emphasises the great importance of separating physiologic from pathologic pulmonary function. Moreover, a sensible policy for additional investigations or referral to a pulmonary specialist, preferably with experience in diving medicine, can save resources and prevent exposure to the potentially harmful effects of radiation, as well as reducing the likelihood of clinically insignificant findings.

This study had several limitations. First, the study population was biased towards healthy and physically fit young adults, predominantly men. Although this makes the results more relevant to military diving personnel, care should be exercised in extrapolating these results to other populations. Although these results may not be applicable to leisure or sports diving or submariners, the study population resembles commercial divers, suggesting that our results may be relevant to this group. Moreover, it could be argued that the GLI-2012 does not optimally represent our population, as the Z-score of the FVC is on average 0.75, where 0 would be expected. However, our sample size of 83 is too small to conclude the GLI-2012 is invalid for the Dutch military diving population. We feel the GLI-2012 is currently the most appropriate dataset for evaluating PFTs in Dutch military personnel, although future research might lead to the development of a specific dataset, as has been proposed for other specific populations.[18]

Secondly, our results were gathered using our earlier published algorithm.[10] We are aware that other dive medical physicians use different reference sets for interpreting PFT results, as well as different strategies that prompt additional tests and referrals to pulmonary specialists. Studies are needed to determine whether our results can be replicated in other populations. As retrospective studies have their limitations, for instance in the case of incomplete records or unclear deviations from the algorithm, a prospective study with a sufficiently long follow-up is required to ensure the suggested more permissive range does not result in harm.

Finally, using slow vital capacity (SVC) rather than FVC to evaluate PFTs may yield more accurate results, because SVC is less affected by technique than FVC. However, at time of this study, the GLI has not published Z-scores associated with SVC, which would have required the use of the outdated ERS-1993 dataset for interpretation of the PFT results. Because the GLI-2012 dataset is more accurate than the ERS-1993 reference values, use of a more reliable dataset would likely provide better results. Additionally, PFTs are performed under supervision of qualified and experienced pulmonary function technicians until reliable end-of-test criteria are met. These findings indicate that the presented data using FVC are valid and support the conclusion of this study.

Conclusion

Adopting the GLI-2012 LLN-2.5 rather than LLN-5 as a cut-off point for FEV_1/FVC in asymptomatic individuals reduces the number of additional tests and referrals to a pulmonary specialist without missing important diagnoses. Taking a thorough history and performing spirometry according to professional standards would save time and resources spent on dive medical assessments and protect subjects from harmful side effects of additional tests, such as radiation. Use of LLN-2.5 for FEV_1/FVC would also avoid findings of unknown clinical significance, which often trigger further tests and potentially disqualify subjects for diving, while maintaining an equal level of safety for divers.

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Case reports

Adjuvant hyperbaric oxygen treatment of acute brain herniation after microsurgical clipping of a recurring cerebral aneurysm: a case report

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Key words
Brain injury; Cerebral ischaemia; Hyperbaric medicine; Intracranial haemorrhage; Surgery

Abstract
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Introduction: Acute brain herniation is a life-threatening neurological condition that occasionally develops due to severe complications following cerebral aneurysm clipping. Strategies for managing acute brain herniation have not improved substantially during the past decade. Hyperbaric oxygen treatment (HBOT) may alleviate harmful effects of cerebral hypoxia, which is one of the most important pathophysiological features of acute brain herniation and, therefore, may be useful as an adjuvant therapy for acute brain herniation. A case treated with adjuvant HBOT is reported.
Case report: A 60-year-old asymptomatic man presented with a recurring left middle cerebral artery bifurcation aneurysm with previous stent-assisted embolisation. After craniotomy for surgical clipping of the aneurysm, disturbance of consciousness and right hemiplegia occurred. Computed tomography (CT) images suggested simultaneous cerebral ischaemia and intracranial haemorrhage. Pharmacologic treatment resulted in no improvement. A CT scan acquired five days after surgery showed uncal and falcine herniation. HBOT was administered five days after surgery, and the patient's condition dramatically improved. He became conscious, and his hemiplegia improved following seven sessions of HBOT. Simultaneously, CT images showed regression of the acute brain herniation.
Conclusions: The patient had recovered completely at one year post-treatment. HBOT may be effective in the treatment of acute brain herniation following cerebral aneurysm clipping.

Introduction

Acute brain herniation is associated with high mortality and morbidity.[1] It may be caused by severe brain oedema secondary to several medical conditions, including cerebral ischaemia and intracranial haemorrhage, either of which may complicate surgery for aneurysm clipping. Although intracranial pressure monitoring and decompressive craniectomy are used to treat acute brain herniation, their efficacy remains controversial.[2]

Hypoxia is the most important pathophysiological characteristic of acute brain herniation.[1] Hyperbaric oxygen treatment (HBOT), defined as respiration of 100% oxygen at a pressure > 101 kPa, improves hypoxia in the brain tissue.[3,4] However, HBOT's utility as an acute brain herniation treatment is unknown. Herein is reported, to the best of the author's knowledge, the first case of acute brain herniation treated with adjuvant HBOT.

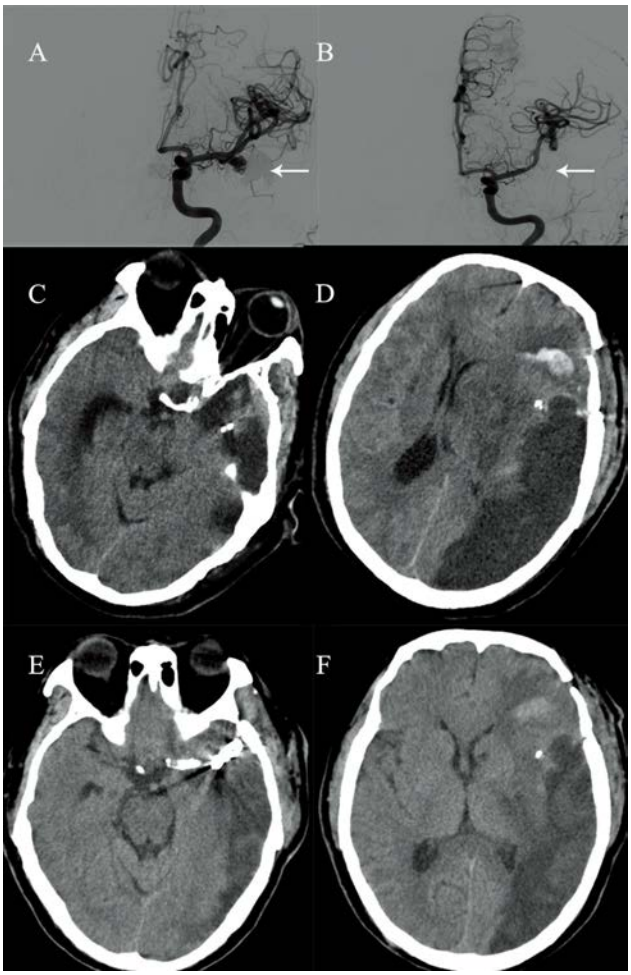
Case report

Consent for publication of this case was obtained from the patient via his immediate family.

A 60-year-old asymptomatic man was admitted to our hospital due to a recurring left middle cerebral artery (MCA) bifurcation aneurysm with a history of two stent-assisted coil embolisation procedures (Figure 1A). He had been diagnosed with a subarachnoid haemorrhage with headache four years prior and had chronic hypertension and kidney stones for 10 years.

After hospitalisation, the aneurysm was completely clipped (Figure 1B). The saccular aneurysm was 30 mm in dome diameter and 5 mm in neck diameter. Balloon dilation was performed to the M1 segment of the MCA because vasospasm was found using intraoperative digital subtraction angiography (DSA). Patency of the left superior branch of the MCA and perforating arteries was visible on

Figure 1
Digital subtraction angiography (DSA) and computed tomography (CT) images. DSA images show a recurring left middle cerebral artery bifurcation aneurysm with a history of two stent-assisted coil embolisation procedures, (A) at admission (arrow) and (B) after complete surgical clipping of the aneurysm (arrow). After attempted pharmaceutical treatment CT images acquired 5 days after surgery (C) show compression of the left cerebral peduncle and (D) a rightward shift of the cerebral midline. Following seven sessions of hyperbaric oxygen therapy CT images show (E) alleviated compression of the left cerebral peduncle and (F) an unbiased cerebral midline



intraoperative DSA images, while the left inferior trunk of the MCA was not visible (Figure 1B). Surgery lasted for 6 h and no cerebrospinal fluid drainage tube was placed.

Postoperatively, the patient suffered somnolence and right hemiplegia without sedatives, with a Glasgow Coma Scale score of 5. Head computed tomography (CT) performed 1-day post-surgery showed slight distortion of the left side of the brainstem due to simultaneous cerebral ischaemia and intracranial haemorrhage. He maintained his airway and was not intubated. Drugs including mannitol, steroids, midazolam, nimodipine, aspirin, clopidogrel, and hydroxyethyl starch were administered. However, no significant improvements occurred. CT images acquired five days post-surgery revealed a narrowed cisterna interpeduncularis, brainstem distortion, and cerebral midline shift (Figure 1C and D). Pupil diameter and light reflexes were normal.

HBOT was initiated on day 5 post-surgery along with drug administration. The multiplace hyperbaric oxygen chamber was pressurised to 203 kPa over 30 min, maintained at 203 kPa for 1 h, and then depressurised over 30 min. During the compression and decompression phases, the patient respired air; during the steady pressure period (203 kPa), he respired 100% oxygen. In total, seven consecutive daily sessions of HBOT were performed. Medical staff accompanied the patient in the chamber to monitor his electrocardiograph, blood pressure, respiratory rate, and general condition. During each HBOT session, his vital signs were stable and within the normal limits, and there were no signs of ear barotrauma or signs of cerebral oxygen toxicity.

After session one, the patient showed increased pain sensitivity. Following session three, he was conscious with mixed aphasia. After session seven, he demonstrated significant improvements in muscle strength and speech. CT images showed no midline shift and no brainstem compression (Figure 1E and F).

At the 3-month follow-up, the patient had hemiparesis and normal speech. CT angiography showed no recurrent aneurysm. At the 1- and 3-year follow-ups conducted via telephone, he had completely recovered.

Discussion

Simultaneous cerebral ischaemia and intracranial haemorrhage were the primary injuries in this patient, while secondary brain oedema led to acute brain herniation. Cerebral ischaemia was present in the region of the left inferior trunk of the MCA, while intracranial haemorrhage may have occurred due to procedure-related complications. Despite controversy regarding the effects of HBOT on cerebral ischaemia and intracranial haemorrhage,[4,5] we chose this intervention because (i) the lack of improvement after medication and risks associated with a decompressive craniotomy were of concern, and (ii) as oxygen is a limiting factor in brain injury recovery, HBOT's potential role in improving brain tissue oxygenation and metabolism was considered important.[3]

EFFICACY OF HBOT

The patient's acute brain herniation receded after treatment with medication and HBOT, resulting in symptomatic and CT image improvement. HBOT for traumatic brain injury has received considerable attention in the past several decades despite the controversy surrounding it.[6] However, few reports of HBOT being used to treat patients after

cerebral aneurysm clipping exist. One study noted that early HBOT improved the postoperative outcomes of patients with intracranial aneurysm.[7] Owing to the acute brain herniation, our patient was in more critical condition than those in the previous study; nonetheless, treatment with HBOT was associated with a good outcome. It is speculated that the HBOT protocol, specifically the timing and dosage, contributed to the good anecdotal outcome.

Regarding timing, early HBOT may help resolve brain hypoxia, which causes brain injury in acute brain herniation.[1,7] Although our patient had consciousness disturbances, his pupil diameters and reflexes were normal, indicating that he was in the early stages of acute brain herniation, prior to the occurrence of oculomotor nerve compromise. Currently, no consensus exists regarding appropriate HBOT timing.[8] Several studies initiated HBOT early after injury, while some suggested that HBOT should begin one month later, citing concerns surrounding oxygen toxicity.[8] We did not observe seizures (an oxygen toxicity sign), providing some reassurance that our early timing was appropriate.

The dose of HBOT is likewise crucial.[8] HBOT at 203 kPa elevates normal brain tissue oxygen pressure from 35 to 240 mmHg and increases brain oxygen diffusion.[9] At 203 kPa, the ability of aquaporin 4 to alleviate cerebral oedema is the strongest.[10] Here, repetitive HBOT was administered, and the patient's condition gradually improved. This might be associated with gradual elevation in cerebral tissue oxygen pressure.[11]

HBOT is considered safe when administered at pressures < 304 kPa for < 2 h.[12] The present case provides qualified support (albeit in only a single case) for the safety of HBOT at 203 kPa for the critically ill patient. We performed monitoring to ensure timely detection and intervention in the event of changes in patient condition.[12]

LIMITATIONS

As HBOT and appropriate medications were used simultaneously, both treatments may have contributed to the regression of acute brain herniation. However, the patient's abrupt improvement after HBOT and lack of improvement after earlier use of medication alone suggests that the improvements are related, at least in part, to HBOT.

Conclusion

This case suggests that HBOT may be an effective and safe treatment for critically ill patients with acute brain herniation following complicated cerebral aneurysm clipping. As this was a single case report, clinical studies with larger sample sizes are required to further elucidate a role for HBOT in this setting.

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A neoprene vest hastens dyspnoea and leg fatigue during exercise testing: entangled breathing and cardiac hindrance?

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Key words
Cardiac function; Cardiopulmonary testing; Dyspnea; Immersion pulmonary oedema; Snorkelling; Wetsuit; Work of breathing

Abstract

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Symptoms and contributing factors of immersion pulmonary oedema (IPO) are not observed during non-immersed heart and lung function assessments. We report a case in which intense snorkelling led to IPO, which was subsequently investigated by duplicating cardiopulmonary exercise testing with (neoprene vest test – NVT) and without (standard test – ST) the wearing of a neoprene vest. The two trials utilised the same incremental cycling exercise protocol. The vest hastened the occurrence and intensity of dyspnoea and leg fatigue (Borg scales) and led to an earlier interruption of effort. Minute ventilation and breathing frequency rose faster in the NVT, while systolic blood pressure and pulse pressure were lower than in the ST. These observations suggest that restrictive loading of inspiratory work caused a faster rise of intensity and unpleasant sensations while possibly promoting pulmonary congestion, heart filling impairment and lowering blood flow to the exercising muscles. The subject reported sensations close to those of the immersed event in the NVT. These observations may indicate that increased external inspiratory loading imposed by a tight vest during immersion could contribute to pathophysiological events.

Introduction

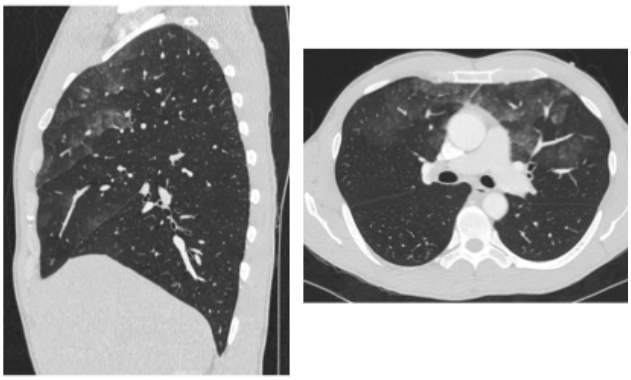
Immersion pulmonary oedema (IPO) can be life-threatening.[1] It is more likely if cardiovascular function is impaired,[2,3,4] but it can occur in fit subjects during immersed exercise.[5–7] However, to date no non-immersed investigation reproduces symptoms and physiological features of an immersed event. Yet recognising individual thresholds of functional tolerance should provide clues to tailor safer patterns of immersed activity. A contribution of inspiratory effort to IPO was evidenced either during exercise or when coping with the breathing loads coming with immersed activities.[8,9] Here we report how subjective sensations and potential physiological features of a strenuous swimming-induced IPO event were elicited during tailored cardiopulmonary testing.

Case report

A 44-year-old fit male fireman was referred for maximal exercise testing after enduring a swimming-induced episode of IPO. The subject was a regularly trained triathlon competitor, with a two pack-year current smoking history and no known cardiovascular disease. The event occurred during rescue training (12°C lake water) with snorkel-swimming over 1 km, followed by 50 m swimming and 3 m depth diving to rescue a dummy 'victim'. The subject used a 5 mm whole body neoprene suit, a mask and a 20 mm lumen snorkel. The 1 km distance was accomplished in 13 min 45 s (< 20 min expected) and after 5 min rest the subject swam to the dummy but was unable to dive and grab it, feeling out of breath. His legs could not continue finning, and he was taken back to shore by a buddy with severe dyspnoea, cough and bloody sputum.

After 30 min of oxygen mask breathing (12 $L \cdot min^{-1}$) the respiratory symptoms had disappeared and the subject went home. Two hours later he attended the emergency department. At examination no signs of heart failure or leg thrombophlebitis were found. Lung wheezes were heard bilaterally. Vital signs were normal. ECG, transthoracic echocardiography and computed tomography (CT) pulmonary angiography were performed, together with blood analysis of B-type natriuretic peptide, D-dimers and troponin. The CT showed anterior ground glass changes in the upper lobes and fine thickening of interlobular septa reflecting venous congestion (Figure 1).

Figure 1
Sagittal and transverse tomodensitometric computed tomography scans taken two hours after the IPO occurrence



IPO was diagnosed. One week later, chest X-ray and pulmonary function tests were normal (FVC = 5.8 L, FEV1 = 4.4 L, FEF50% = 4.79 $L \cdot s^{-1}$).

Two months later, a standard bicycle ergometer maximal exercise test was performed (standard test – ST). The subject wore shorts and T-shirt, and after a 3 min warm-up at 50 W, workload increased by 25 W every minute. Measurements during the test were as previously described.[10] The maximum oxygen consumption (VO_2max) was 57.1 $ml \cdot min^{-1} \cdot kg^{-1}$ (163 % predicted) and maximum of work rate was 400 W. Resting blood pressure was high (169/109 mm Hg), but the blood pressure profile during maximal exercising was normal. A 24 h ambulatory recording of blood pressure confirmed hypertension.

After one month, the exercise test was repeated with exactly the same protocol but included the wearing of a 5 mm thick neoprene vest (neoprene vest test – NVT). During this trial, both dyspnoea intensity and lower limb fatigue (Borg scales) rose earlier and were significantly higher at any given work rate than during the first test (Figure 2). The NVT ended earlier, at 350 W and 55.2 $ml \cdot min^{-1} \cdot kg^{-1}$ VO_2max, with severe breathlessness. Minute ventilation and breathing frequency rose faster during the NVT than ST (Figure 3), but tidal volume was similar at each level in the two tests (max 3.1 L in ST and 3.2 L in the NVT). The patient appraised his sensations as equivalent to those experienced during the immersed event with a feeling of "*occurring death*" before bloody spitting. No significant dynamic hyperinflation, and no mechanical ventilatory limitation were seen. Heart rate and diastolic blood pressure rose earlier than during the ST. Systolic arterial pressure and pulse pressure plateaued earlier and remained lower during the NVT than the ST (Figure 4). In the ear lobe arterialised capillary blood PaO_2 and SaO_2 were not decreased during maximum exercise in either the ST and NVT. The subject described the intensity and unpleasantness of dyspnoea during the NVT as similar to those during the field event.

Figure 2
Dyspnea and leg fatigue scores (Borg scales) during the two cardiopulmonary exercise tests; NVT – neoprene vest test; ST – standard test; W – workload in watts

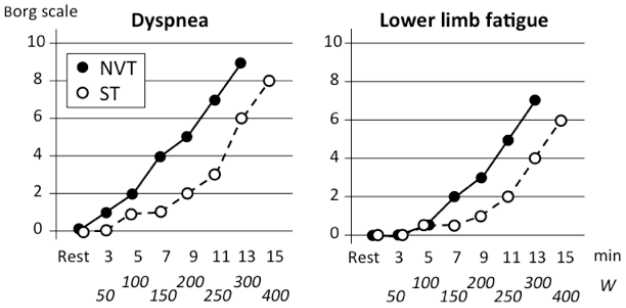


Figure 3
Minute ventilation and breathing frequency during the two cardiopulmonary exercise tests; NVT – neoprene vest test; ST – standard test

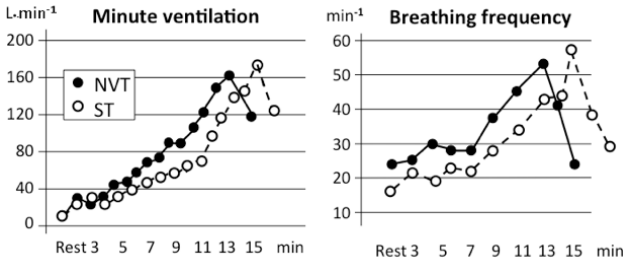
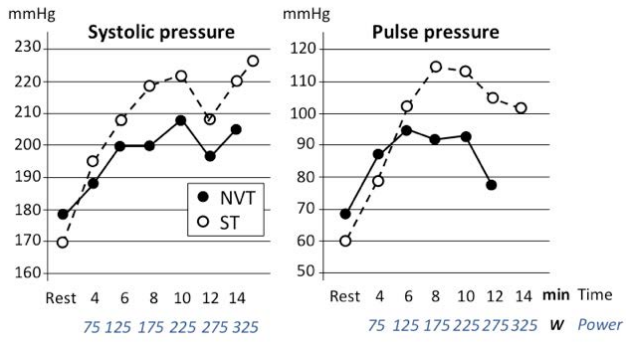


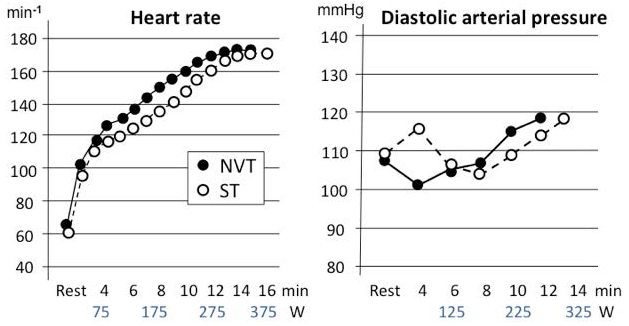
Figure 4
Systolic arterial pressure and pulse pressure during the two cardiopulmonary exercise tests; NVT – neoprene vest test; ST – standard test



Discussion

Completing the 1 km surface swim in less than 14 min i.e., much faster than required, required high-level exercise. The elastic restriction of the neoprene suit reinforced the effects of hydrostatic pressure[11] to reduce both total vascular capacity in the thorax and lung compliance,[12] as venous return, right heart preload and lung blood

Figure 5
Heart rate and diastolic blood pressure during the two cardiopulmonary exercise tests; NVT – neoprene vest test; ST – standard test



volume increased.[8,13] During immersion the neoprene suit also loaded lung mechanics by requiring supplementary inspiratory effort[14,15] and increasing the transpulmonary pressure load.[8] Finally, the snorkel enlarged the anatomical dead space, which also requires an increased ventilatory volume.[16] Together the loads of immersion, elastic suit and snorkel substantially increased inspiratory effort during the 14 min high intensity swim. Adding dead space and chest strapping cause severe dyspnoea and exercise intolerance in healthy subjects cycling on land.[16] The physiological strains and sensations in that study appear similar to those in IPO.

Most of the differences observed between the ST and NVT tests were likely explained by the "abnormal" restrictive effect of the neoprene vest.[17] One study concluded that with "abnormal" restrictive constraints on tidal expansion, the intensity and unpleasantness of dyspnoea reflect the awareness of increased neural respiratory drive needed to achieve any given level of ventilation during exercise.[17] A higher inspiratory power of breathing was required to achieve tidal volume at each exercise level during the NVT than the ST. Minute ventilation rose faster during the NVT in keeping with the previous results.[16] At high exercising levels, ventilatory muscles require increased blood flow, diverted from the working limb muscles.[18] Accordingly, during the NVT the earlier rises of heart rate and diastolic blood pressure at the highest workloads (Figure 5) likely conveyed the higher sympathetic activity instrumental in re-routing part of the cardiac output from the lower limbs towards respiratory muscles, leading to earlier leg fatigue.

The first systolic blood pressure value was 9 mmHg higher in the NVT than the ST (Figure 4). Higher anxiety at beginning of a second maximal exercise test or simple measurement variability[19] might explain this. However, we consider that despite baseline sympathetic possibly being higher during the NVT, the plateauing of systolic arterial pressure and pulse pressure to remain roughly 10 mmHg and 20 mmHg respectively lower than during the ST (Figure 4) could be consistent with restricted stroke volume during this second part of the NVT i.e., at the highest energy expenditure levels.[18] Immersed cumulative inspiratory effort was found to correlate with end-stage right heart preloads and right to left ventricle ratios,[8] thus paving the way for imbalance of ventricular outputs and consequently fluid extravasation into lung interstitium.[20] During the NVT the additionally loaded inspiration due to the restrictive effect of the vest elasticity may have caused lowering of pleural-mediastinal pressure thus increasing both right heart preload and left ventricle afterload while possibly decreasing the left ventricle preload through leftward interventricular septal displacement.[21,22] Such a restricted left ventricular preload would then impede the rise of stroke volume, consistent with reduced systolic and pulse pressures in the NVT. In addition, the larger sympathetic activation during the NVT would have added to left ventricular afterload and further impeded the stroke volume.

Immersion-linked conditions are instrumental in developing IPO.[23] IPO was first described during cold water diving.[24] Immersion in cold water causes peripheral vasoconstriction, lung congestion, increased pulmonary arterial pressure and increased left ventricle afterload.[25] Immersion may also increase airway resistance and breathing work.[26] In the present case the water temperature could have triggered peripheral vasoconstriction and contributed to increase central blood volume and right heart preload.[24,27] However, it is also possible that during the vigorous swimming, skin vasoconstriction was suppressed, given the combination of the large muscular heat production and the suit-hindered heat dispersal into water.[28] The higher work of breathing in water than air is described,[15,29,30] but neither the additional inspiratory load of wet suit, nor its haemodynamic burden have been reported to the best of our knowledge. A small effect of a wet suit on maximal expiratory flow has been described.[31]

The development of haemoptysis and pulmonary oedema during the field event suggest pulmonary capillary stress failure[32,33] suggestive of cardiopulmonary stresses greater than those of the NVT. Our interpretation of the haemodynamic change during the NVT, possibly suggesting increased congestion of right heart and pulmonary circulation with restrained stroke volume would mimic a loss of left ventricular diastolic compliance.[34,35] This also takes place during pulsus paradoxus as it develops during high inspiratory efforts as in acute asthma[36] and whose cardiac features root in normal breathing.[37] Pulsus paradoxus is defined by a more than 10 mmHg inspiratory lowering of pulse pressure linked to right ventricle overdistension and the simultaneously reduced left ventricular filling through parallel biventricular interdependence.[36] This pattern would likely support the feeling of suffocation featured in congestive heart failure at rest[38] and repeatedly reported during IPO. Therefore we submit that the duplicated cardiopulmonary exercise testing sheds some light on symptoms repeatedly reported during the development of IPO. The faster increase

in dyspnoea during the NVT than the ST reflected the required higher inspiratory effort. In turn, this might have prompted a parallel circulatory impairment indirectly evidenced by the restriction of systolic and pulse pressures at the highest levels of exercising power together with higher leg fatigue. In status asthmaticus, pulsus paradoxus and dyspnea are lowered when breathing load is lessened through replacing air by heliox inhalation.[39] Accordingly we hypothesise that, beside the greater inspiratory effort caused by the elastic vest, part of the additional dyspnoeic sensations in the second half of the NVT might have been linked to ventricular imbalance and restriction of left stroke volume.

IPO results from extravasation of plasma into interstitial airway spaces, leading to decreased airway luminal caliber and later to impaired pulmonary gas exchange. Ultimately this causes arterial hypoxaemia and alveolar flooding.[40,41] Arterial blood gases were not altered in either test, and DLCO was not assessed so that no direct evidence of altered lung gas exchange was obtained. The absence of hypoxaemia is nevertheless consistent with the gradual development of right to left ventricular imbalance[17,33] which would eventually result in impaired gas exchange. According to this pathophysiological paradigm various forms of reversible myocardial dysfunction may then follow during immersed activities.[2,3,42] The higher myocardial work and a raised sympathetic stimulation would also pave the way for myocardial ischaemic events.[43]

The occurrence of IPO has been suspected to predict the development of arterial hypertension,[24] and the reverse connection was described.[44] In the present case, the high blood pressures on first test day led to ambulatory monitoring, diagnosing moderate hypertension, and appropriate treatment was started after the second exercise test. Some degree of hypertension-linked left heart diastolic dysfunction and a higher left ventricular afterload bolstered by sympathetic activation at high exercising levels might have enhanced the delay of left ventricular emptying as compared to the right.[45,46]

In conclusion, these observations underscore the potential respiratory burden imposed by an elastic wet suit during substantial swimming effort. We suggest they may also shed some new light on the role of work of breathing in developing IPO through interlocked haemodynamic and respiratory alterations.

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Letters to the Editor

Diving-related disorders in breath-hold divers could be explained with the distal arterial bubble hypothesis

In the June 2021 issue of Diving and Hyperbaric Medicine,[1] Dr Kohshi and colleagues, expressed a degree of skepticism that distal arterial bubble formation[2,3] can be the main explanation for cerebral decompression sickness (DCS) in breath-hold divers. We have previously suggested that in the bifurcating tree of a distal artery, the vessel's wall becomes thinner and wall surface to volume ratio of the vessel increases; both of these circumstances enhance nitrogen diffusion from the surrounding tissue into the blood. A local reduction of blood flow, also leaves enough time to enhance nitrogen diffusion from tissue into the blood. If an active hydrophobic spot (AHS) is located in the distal artery, a nanobubble at the AHS could develop into a larger decompression bubble, blocking perfusion. In repeated breath-hold diving, a local bubble would remain almost stable because of a very small oxygen window in the arterial blood, and would continue to expand in further dives.

Kohshi and colleagues claimed: "*However, this hypothesis cannot explain why one or more large ischaemic lesions are not invariably accompanied by multiple small ones in the subcortical areas*".[1] There are two prerequisites for the expansion of distal arterial bubbles: enhanced diffusion of nitrogen with respect to the volumetric blood flow; and existence of an AHS. The distribution of AHS is highly variable between individual sheep, and within their blood vessels. I assume a similar variability exists in divers. Thus, if the diffusional conditions are appropriate for gas loading but there are no AHS, bubbles would not appear. If, however the diffusional conditions are appropriate for gas loading in a somewhat large artery, all arteries of the next generations having AHS would have decompression bubbles. The authors stress: "*The ischaemic lesions…were situated in the terminal zone…and border zone. They are so-called...low flow*".[1] Local low perfusion, which allow enough time for nitrogen loading, is exactly a major cause for distal arterial bubble formation.[2] The authors also conclude: "*The dramatic and rapid response suggests the presence of bubbles in the cerebral arteries. Nitrogen accumulation in fat tissues increases throughout repetitive breath-hold diving*".[1] These are exactly the appropriate parameters in distal arterial bubble formation. As is remarked in the review: "*Why lesions in breath-hold diving mainly involve the brain but not spinal cord is an unresolved question*".[1] I believe the distal arterial bubble is the answer.

Alessandro Marroni (personal communication) reported one case of clear clinical neurological symptoms in breath-hold diver without any detectable bubbles on echocardiography. A viewpoint on neurological decompression sickness in breath-hold diving claimed: "*Most studies reported no or rare circulating bubbles after repeated breath-hold dives. Consequently, other mechanisms should be considered*",[4] and other authors suggested: "*However, comparing the incidence of DCS in breath-hold diving with the low prevalence of venous gas emboli with scuba divers who often produce venous gas emboli in higher quantities with relatively low rate of injury, clearly the pathogenesis is more complex*".[5] These viewpoints and Marroni's observation contradict the preference expressed by Kohshi and colleagues for veno – arterial shunts as the main cause for breath-hold DCS: "*The prevalent theory of brain involvement is that arterialised venous gas bubbles passing through right to left shunts may be the plausible mechanism*".[1]

In summary, I am confident that the distal arterial bubble formation is the best explanation for the mechanism of cerebral DCS in breath-hold diving, rather than bubbles shunting from the venous to the arterial circulation and directed mainly to the brain.

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Ama diver; Decompression sickness; Gas micronuclei; Letters (to the Editor); Nanobubbles; Taravana

Reply: Diving-related disorders in breath-hold divers could be explained with the distal arterial bubble hypothesis

Thank you for the opportunity to respond to Dr Arieli. First of all, no clear mechanism of decompression sickness (DCS) in breath-hold (BH) diving has been confirmed or established, and we are all only guessing at various possibilities and hypotheses for the diving disorders.[1] Repetitive BH diving mainly causes stroke-like neurological events consistent with ischaemic brain lesions, and moreover the clinical characteristics of DCS in BH divers are different from those in scuba diving.

Dr. Arieli's hypothesis that cerebral DCS is caused by distal arterial bubbles developed from nanobubbles is interesting;[2] it seems to explain the formation of venous bubbles. However, we consider that gas micronuclei underlying decompression bubbles are circulating in all blood vessels, and that they may expand as a result of nitrogen transfer from the blood and surrounding tissues in repetitive BH dives. Microbubbles (less than 21 micron) formed from gas micronuclei can pass through capillaries of all tissues; they may grow mainly in venous capillaries. Hence, venous bubbles have been detected in some BH divers,[1,3] similar to the presentation in scuba diving.

We have presented some cases of stroke-like neurological disorders in Japanese Ama divers whose magnetic resonance imaging (MRI) of the brain showed signal changes in the 'external' watershed areas and the territories of the perforating arteries.[3] Watershed infarcts of the brain are grouped into two main categories based on their location in either internal or external regions.[4] The former are located at the junctions of the cortical arterial territories with deep perforating arteries, showing the rosary-like pattern in the centrum semiovale; they are mainly affected by hypoperfusion due to arterial stenosis or haemodynamic impairment. In contrast, the latter 'external' infarcts occur at the junctions of the distal fields of cortical arteries and are usually wedge-shaped, and their cause may be embolic rather than haemodynamic in nature.[4] Other ischaemic lesions involve the territories of the perforating arteries in the basal ganglia and brainstem.[3] While lacunar infarcts resulting from occlusion of a single perforating artery are considered to be due to atheromatous changes, one-third may include emboli from cardiac or carotid sources.[5] The MRI findings of Ama divers with stroke-like neurological disorders support the hypothesis that an embolic mechanism plays a crucial role in the pathogenesis of infarcts in the external watershed areas and the territories of perforating arteries; that is, the most plausible aetiology is occlusion of the cerebral arteries by emboli.[3]

Venous nitrogen bubbles formed following BH dives are trapped or retained in the pulmonary arteries. The 'trapped' bubbles are compressed during each dive and may therefore arterialise.[1] Circulating bubbles in arteries will increase in size during ascent; some of the bubbles flow into the arteries of the brain, especially at the junctions of the cortical arteries or in the perforating arteries. Moreover, even small bubbles may cause endothelial damage,[1] and may provoke thrombus formation and affect arterial occlusion of the brain. The bubble seeds are the first step in neurological DCS for BH divers, and this may be followed by expansion of bubbles is due to nitrogen influx from the end of occluded arteries and surrounding brain tissue. At present, the above hypothesis seems to best explain the mechanisms of neurological DCS in Ama divers.

In conclusion, based on MRI findings of the brain in Ama divers, repetitive BH dives cause stroke-like neurological disorders compatible with cerebral arterial embolism. We consider that nitrogen bubbles arterialised across the pulmonary circulation play a key role in brain damage in BH diving, although the mechanisms are not clear.

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Key words
Ama diver; Cerebral embolism; Cerebral infarction; Decompression sickness; Letters (to the Editor); Magnetic resonance imaging

Pulmonary barotrauma after helicopter underwater escape training

I read with great interest the case report by Lindblom and Tosterud presenting a case of pulmonary barotrauma and cerebral arterial gas embolism after shallow submersions in a pool, breathing compressed air.[1] Their report cites the two other published reports on this subject.[2–3]

In late 1997 I participated in a conference in Birmingham discussing subjects related to helicopter evacuation training for passengers travelling offshore to work in the oil and gas industry. The majority of the participants were representatives from safety training centres. I have filed my presentation at that meeting, but regrettably I am unaware of any conference proceeding or proper reference to the meeting. However, I believe that it would be of interest to reiterate the case report I presented. The information below is retracted from the manuscript I submitted at the time.

The incident took place in an offshore safety training centre outside Bergen, Norway in May 1997. At the time, training was provided for pilots in the offshore helicopter companies as well as crew members of selected helicopter wings of the Royal Norwegian Air Force (RNoAF). During flight, these crew members were provided with small (0.2–0.4 L) 200 bar compressed air cylinders with a demand regulator. The auxiliary gas supply was provided to allow breathing in the event of evacuation from an upside-down ditched helicopter in sea. The crew members were regularly trained in the use of this breathing apparatus in a helicopter model submerged in a 4 m shallow freshwater pool. The training programme would include familiarisation to minimal submersion of the head at the shallow part of the pool, evacuation from the helicopter model in the upright position at surface and finally evacuation from the rotated model at a water depth of approximately 1 m.

The patient was a 28-year-old male, working mainly for the Norwegian Coast Guard (Lynx helicopter). He was previously healthy and had been examined annually according to the relatively strict requirements for military air crew members. He had participated in two simulated helicopter ditches the same day without any problem. He experienced no technical/practical problem during the last (third) ditch, but complained of restrosternal pain about 15 min later. He had no coughing, but the pitch of his voice changed and he experienced vertigo. He was immediately transferred to the naval base and medically examined by myself approximately 30–40 min after the incident. He felt no problem at that time, and medical examination – including a thorough neurological examination – was normal. He had reported vertigo and neurological affection could not be eliminated. Due to this he was recompressed and treated according to a modified USN Treatment Table 6. He was transferred to Haukeland University Hospital the same evening-Neurological examination was without pathological findings. Chest X-ray demonstrated air in the mediastinum. EEG was normal. He was re-examined with a chest CT 1–2 days later with completely normal findings. He returned to air service without restrictions, but has not been followed up by me.

This incident happened 24 years ago and further medical details are unavailable. From a medical perspective, this story adds little additional information to the three cases already published,[1–3] but underscores that pulmonary barotrauma may occur after helicopter underwater evacuation training (HUET). The reason for bringing this to the attention of the journal readership relates to the ongoing debate on HUET training for helicopter passengers travelling offshore. HUET is one of a large number of factors affecting successful evacuation of a ditched helicopter.[4] Passengers travelling offshore by helicopters are commonly supplied with an emergency breathing system (EBS), and the European Commission has prescribed that "*All persons on board shall carry and be instructed in the use of emergency breathing systems*".[5] The European Union Aviation Safety Agency (EASA) Acceptable Means of Compliance states that the "*EBS …should be an EBS system capable of rapid underwater deployment*".[6] There are three commonly applied EBS systems available for such use:

- A conventional SCUBA open-circuit air breathing system with a small container of pressurised gas connected to a demand regulator.

- A rebreather (simply a rebreather bag connected to a mouthpiece) to be filled with expired gas from the passenger.
- A hybrid rebreather with a small cylinder of compressed gas connected to the rebreather bag injecting compressed air once the EBS is immersed.

The UK Civil Aviation Authority has published a draft standard for EBS[7] and categorised them as "*Class A*" and "*Class B*" (CA EBS and CB EBS). CA EBS should be designed to allow deployment in air as well as submerged, while a CB EBS will require deployment in air. CA EBS is accordingly a more robust system. The miniaturised scuba is generally considered to comply with the CA EBS. Passenger training in the use of these EBS varies between jurisdictions. Canada has established a mandatory in-water training programme including underwater breathing of compressed air. UK (OPITO) offshore workers are inducted with theoretical and 'dry' training in compressed air breathing. In Norway offshore workers are exempted by the national civil aviation authority from the EBS training, but basic offshore safety induction and emergency training includes HUET training by means of the rebreather system. One important explanation for these differences is the risk assessment of pulmonary barotrauma associated with pressurised underwater breathing systems. The fidelity of the training will depend on whether the participants are inducted by theoretical training only or whether they are exposed to underwater breathing. Risk assessment will depend on the expected incidence of complications and their nature. Future risk assessments of HUET training with pressurised breathing gas should consider the present case description as well as the three previously published reports[1–3] of shallow water pulmonary barotrauma and cerebral arterial gas embolism.

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Case reports; Cerebral arterial gas embolism; Diving; Injuries; Letters (to the Editor)

Obituary

Peter Wynne McCartney OAM, MBBS, MMed (Anaesthesia), DipDHM

Peter McCartney, the father of Diving and Hyperbaric Medicine in Tasmania, died on 03 September 2021, aged 90 years. Peter was a visionary and he has inspired many young doctors to practice hyperbaric medicine.

Born in Johannesburg, South Africa, youngest of eight children, he learnt Zulu, Afrikaans and English as a child. Following an unconventional schooling, Peter enrolled to study science and engineering, but transferred to medicine and graduated from the University of the Witwatersrand.

During his intern year in Rhodesia, he met his future wife, Dr Penny Leighton. Romance ensued and they were married in Johannesburg in 1960, launching their enduring and loving 61-year partnership. A move to the UK, saw sons Paul and Phillip added to the family. Following the UK, they moved to the island of St Helena, where Peter and Penny were the only doctors for 6000 people. Peter found an amazing piece of marine archaeology when snorkelling at St Helena – a catenary stone (Figure 1). He investigated its origins (including carbon dating surface material) and concluded it originated in the Mediterranean region and was more than 2,500 years old. It remains located at the Royal Hobart Hospital (RHH) hyperbaric facility.

Peter and Penny returned to South Africa in 1963, where children Anne and Bern were born. He started his Anaesthesia (M. Med (Anaes) Pretoria) and his diving and hyperbaric medicine career (South African Naval Base, Simonstown). In 1972 the family moved to Hobart, Tasmania, where Peter was employed at the RHH where he was able to apply his skillsets of anaesthesia and diving medicine.

Figure 1
Catenary stone

The first RHH Hyperbaric chamber, built in 1967, installed in 1974, was a Kirkswood twin-lock chamber (diameter 48-inch, length 12 ft 8 inches). Peter became involved with the facility as an anaesthetist and became the hospital's first Director of Diving and Hyperbaric Medicine. Records of the first chamber dives have been lost. The first documented clinical record of chamber operation under Peter's supervision was in 1980 (Dive 32 for the chamber). The topside supervising doctor was Penny McCartney, with Peter inside the chamber attending his patient. The final treatment using this pioneering RHH chamber (22 February 1993 – Dive 1242), was also supervised by Peter.

Whilst operating the first RHH hyperbaric facility during the 1980's Peter planned and designed a next generation recompression chamber that was capable of housing five patients at once. His planning process included engineering, occupational health and safety and patient comfort (sound baffling under the floor, huge 750 mm 6 ATA portholes for natural light and floor level entry for staff), psychology (via colour selection and ambience). In addition, Peter personally lobbied political support to fund the new facility (at $1.6 million). This was achieved despite many local antagonists, and without Australian (and New Zealand) standard 4774.2 for guidance. The chamber was constructed locally by Tasmania's Hydro Electric Commission. Peter literally *saw the future* when designing the new RHH facility. It was purpose-built for dysbaric illness, and medical cases including ICU patients. The "*McCartney*" chamber commenced manufacture in 1991, was commissioned 30th October 1992, and first patients were treated 25 February 1993. Peter supervised the first Tasmanian hyperbaric nurse course in January/February 1993.

Peter remained as Director of the Department of Diving and Hyperbaric Medicine at the RHH until his retirement at the end of 1997. He was succeeded in 1998 by Drs Margaret Walker and David Smart. The McCartney facility operated from 1993-2015, with 10,816 compressions, treating 34,473 patients. When Peter commenced with the first chamber in the 1970's, such numbers of patients receiving HBO treatment could never have been imagined.

Peter's enthusiasm for the discipline has spawned many generations of hyperbaric physicians (of whom the author is one), equipping us with a fundamental interest in the science of diving and hyperbaric medicine. Peter also published several scientific papers. He was a committee member with the South Pacific Underwater Medicine Society from

1986–1990 and was awarded the Order of Australia medal in 1990 for services to diving and hyperbaric medicine.

Peter particularly enjoyed diving, snorkelling, camping, and sailing, particularly when shared with his family. Peter was a man of many parts: a dedicated family man, a visionary, a dreamer, a raconteur, a keen intellect, an enquiring mind, a teacher, always kind and generous. To Peter, every day was a "*great*" day; an opportunity to meet, to explore, to think and to enjoy.

Peter's parting words to me every time we had been together were to "*go gently*". In the words of Zulu, *hamba kahle*, Peter.

Professor David Smart
Royal Hobart Hospital

Notices and news

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

SPUMS President's message

Neil Banham

Another year has passed by already!

Much has changed over the last year, with widespread uptake of COVID-19 vaccination reducing the burden of severe illness, hospitalisation and deaths, which we should be very thankful for.

However, much has stayed the same. With travel restrictions still in place in New Zealand and in some states of Australia, we have again been curtailed in our plan to have an in-person Annual Scientific Meeting (ASM) with a diving program in Tutukaka next year. The 2022 ASM will again be held mainly virtually and coordinated by Greg Van der Hulst and his team from New Zealand. We hope that a 'normal' SPUMS ASM can be held in 2023, most likely in Australia.

Over the last few months, thanks to a funding grant from the Australasian Diving Safety Foundation (ADSF), back issues of *Diving and Hyperbaric Medicine* (DHM) and its predecessor *SPUMS Journal* are now able to be accessed via the DHM website. This enables public access to individual articles dating back to the publication of the first SPUMS Newsletter by Carl Edmonds in 1971.

Many thanks for the support of John Lippmann and ADSF for making this possible, and to Nicky Telles for making it happen.

John Lippmann and ADSF are keen to support a variety of initiatives that relate to diving safety, including research and diving safety promotions. John can be contacted on johnl@adsf.org.au

The ANZHMG Introductory Course in Diving and Hyperbaric Medicine will again be held in Fremantle in 2022 (21 February–04 March) details are on the SPUMS website https://www.spums.org.au/content/approved-courses-doctors

This course is always fully subscribed, so if you are considering attending, apply soon.

I wish all SPUMS members and their families a safe and enjoyable festive season and a happy New Year. May the 51st year of our society be a good one.

Neil Banham
SPUMS President

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

The new Diploma of Advanced Diving and Hyperbaric Medicine was launched on 31 July 2017. Those interested in training are directed to the ANZCA website https://www.anzca.edu.au/education-training/anzca-diploma-of-advanced-diving-and-hyperbaric-me.

Training

Documents to be found at this site are:

- Regulation 36, which provides for the conduct of training leading to the ANZCA Dip Adv DHM, and the continuing professional development requirements for diplomats and holders of the ANZCA Certificate of DHM;
- ANZCA Advanced DHM Curriculum which defines the required learning, teaching and assessment of the diploma training programme; and
- ANZCA Handbook for Advanced DHM Training which sets out in detail the requirements expected of trainees and accredited units for training.

Examination dates for 2022

Written examination	See website for dates
Viva examination	See website for dates

Accreditation

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

Transition to new qualification

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

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

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

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

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

Royal Australian Navy Medical Officers' Underwater Medicine Course 2021

Date: 14–25 March 2022

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$1,355.00 (ex GST), this is yet to be confirmed.

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*

SPUMS Facebook page

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

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

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

Notices and news

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

EUBS President's message

Jean-Eric Blatteau

I am very honored to participate in the long cycle of Presidents who have followed one another regularly since the creation of the EUBS in 1971.

I was surprised when looking at the archives that very few French colleagues participated in the Executive Committee or the Presidency of EUBS. Among the first presidents of EUBS, I found an eminent French doctor, Dr Xavier Fructus, who fully contributed to the epic era of deep diving with the company Comex. All of this brings us back to a time when the primary goal was to push the limits, trying to break records in depth- and dive-time.

Today, due to robotic advances, deep human incursions are no longer the priority. On the other hand, recreational and professional diving has developed considerably, we realize that diving accidents persist despite adherence to procedures and that new clinical entities in diving accidents are emerging. The 'practice of diving' is now responsible for an accidentology which raises new questions – so despite already 50 years of EUBS, we still have a lot of work ahead of us in the area of diving medicine.

In our field, I welcome the initiatives of 'regrouping' as has happened in 2008 with SPUMS and our joint journal, Diving and Hyperbaric Medicine, which proves to be a real success. More recently, for hyperbaric medicine, the upcoming planned merger of EUBS with the European Committee for Hyperbaric Medicine (ECHM, under the leadership of Daniel Mathieu and Jacek Kot), seems to me to be a very good thing.

We will still have work to increase our visibility and our interactions with other societies, because in a small field like ours, the pooling of skills and collective intelligence should make it possible to better know our specificities and strengthen our practice.

Finally, I would like to congratulate my predecessor, Ole Hyldegaard, for having been able to steer EUBS through this difficult period of COVID-19 pandemic, which unfortunately is not yet over. Thanks to Ole, Peter Germonpré, our Honorary Secretary and also thanks to the entire Executive Committee, our Society has been able to remain active by offering recommendations on COVID-19 both in diving and hyperbaric medicine, and the organization of on-line webinars, which were well attended, not only by EUBS members but also by scientists and clinicians from around the entire globe. These webinars were a big success and we hope to be able to all meet again at EUBS congresses and to be able to interact face-to-face.

Thank you for your confidence in our ExCom team, and see you soon.

Jean-Eric Blatteau
EUBS President

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.

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

EUBS Notices and news

EUBS 2020–2022 Annual Scientific Meeting

The 46th EUBS Annual Scientific Meeting postponed due to the COVID-19 pandemic, was again postponed and has now been announced for 31 August – 03 September 2022, in Prague, Czech Republic.

While at the time of this publication the pandemic is still very much a reality, EUBS hopes to welcome its members, many friends and scientists from around the globe for our '2020–2022 meeting', to finally be able to gather in person and renew/strengthen our professional and personal relationships.

Keep monitoring the website https://eubs2020.com/ for all news and updates.

Our next Annual Meetings will be scheduled as:
2023 – Porto, Portugal
2024 – Brest, France
2025 – Turku, Finland

EUBS Annual General Assembly

Also due to the COVID-19 pandemic, our annual EUBS General Assembly has been held in a 'virtual meeting' format. All EUBS Full Members in good standing had a chance to express their opinion and vote online regarding the 'current affairs' decisions for our Society. A high 46% of our members voted and the results of the vote, the full GA presentation and all accompanying documents are still available on the EUBS website, in the 'Members Area' section.

During the GA, we also voted for a change in the Constitution and Bylaws of the Society. The main change is that instead of having three 'Members-at-Large' in the Executive Committee, each for a period of three years, we will now have four, each for four years. This means that the next ExCom elections will be held for a four year position as Member-at-Large. The new Constitution and Bylaws text is also available on the EUBS website.

EUBS Executive Committee

To replace Oscar Camacho from Porto (Portugal), after serving a three-year term, Evangelos Papoutsidakis, from Barceelona (Spain) has been elected as new Member-at-Large 2022. The Executive Committee wish to express their gratitude for Oscar's contributions to the ExCom activities. We have also elected a new Vice President for the EUBS: Bengusu Mirasoglu from Istanbul (Turkey) was approved by the vast majority of EUBS members.

Finally, there will be a change of Presidents, with Jean-Eric Blatteau taking over the Presidency from Ole Hyldegaard, who becomes 'Immediate Past President'. As the chain of seats progresses, Jacek Kot becomes 'Past President' and we have to say goodbye to Costantino Balestra; as the Chairman of the Research and Education Committee. 'Tino' will however remain present in many of the ExCom discussions. The composition of the new ExCom 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/
Twitter: @eubsofficial
Instagram: @eubsofficial

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

EUBS membership

Do not 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 DHM Journal, (if selected) a print copy of the e-journal for your convenience, reduced registration fee at our Annual Scientific Meetings (this alone already reimburses your membership fee), reduced membership fees at selected Affiliate Societies, access to the GTÜM 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 note – that payment by PayPal is by far the easiest and also the cheapest way to pay your membership fee. Bank transfer is also available, however, this means you will have to pay the banking costs for international money transfers (EUBS is registered in the UK, which is now outside of Europe). Please select "*all banking costs carried by the sender*" when you make the transfer. Also, the money

transfer may take up to one week and may fail for some obscure reason. Therefore, unless you are in the UK, we do not recommend this payment option. Using 'Transferwise' is another option to reduce or avoid banking costs and have ensure 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!

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

In case you have any suggestions for adding or correcting the info posted, please contact us at webmaster@eubs.org.

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.

SIMSI Società Italiana di Medicina Subacquea ed Iperbarica

The Italian Society of Underwater and Hyperbaric Medicine (SIMSI) is still confident to grant those expected educational and training opportunities.

Date: 28–29 January 2022, Niguarda Hospital, Milan

"*Respira, Ripara, Rigenera*" (Breathe, Repair, Regenerate)

A meeting dedicated to the potential of hyperbaric oxygen that, thanks to a very active national diving and hyperbaric medical network (24/7/365), is tested daily both in case of outpatient routine treatments and in emergency scenarios.

Date: 02–04 December 2022, Padua

"*SIMSI XXV Biennial Congress*", University of Padova

Coinciding with the celebrations for the 800th anniversary of the University of Padua.

To take advantage of an early-bird fare, please keep up-to-date with 'Your membership' and 'Your invite', by regularly visiting https://simsi.it/. Here you will find the latest updates on news, meetings, initiatives, sector events under the aegis of SIMSI.

Remember your SIMSI membership means you are entitled to a 10% discount for your EUBS membership.

Gerardo Bosco and Vincenzo Zanon

Courses and meetings

Scott Haldane Foundation

As an institute dedicated to education in diving medicine, the Scott Haldane Foundation (SHF) has organized more than 300 courses all over the world, over the past 28 years. SHF is targeting on an international audience with courses world wide.

Due to the COVID-19 pandemic some courses are rescheduled. Fortunately, we were able to find new dates for all postponed courses. Below are the up and coming SHF-courses in the first quarter of 2022.

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

2022

3rd week of January
Internship different types of diving (2d) Royal Dutch Navy-Den Helder NL

February (5 hours)
Refresher course the diving medical in practice, NL

25–26 March
Medical Examiner of Divers part 1 (level 1), Bunnik/Zeist, NL

31 March, 01–02 April
Medical Examiner of Divers part 2 (level 1), Amsterdam Univ. Med. Centre, NL

14–21 May
Medical Examiner of Divers part 2 (level 1), Bonaire, Dutch Caribbean

On request Internship HBOt (level 2d certification), NL/Belgium

The course calendar will be supplemented regularly. For the latest information see: www.scotthaldane.org. Please also check the COVID-19 News update on this website for the latest schedule changes.

German Society for Diving and Hyperbaric Medicine (GTÜM)

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

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Foundation of Diving Research, SDR

Saturday 26 March 2022, AMC, Amsterdam: Symposium to celebrate the 50 year anniversary of the Dutch Stichting Duik Research (SDR, Foundation of Diving Research).

Topics: 50 years research by SDR; diving cardiology; safety of professional diving; diving to perform coral biotope research and open sea under water archaeology; physiological adaptations of diving mammals. 4 cp.

Visit: http://www.duikresearch.org/ or http://www.diveresearch.org/
For more information: n.a.schellart@amsterdamumc.nl

Baltic International Symposium on Diving and Hyperbaric Medicine

The second Baltic International Symposium on Diving and Hyperbaric Medicine will be held on 09–11 June 2022, in Gdynia, Poland as an on-site meeting.

Let's meet at the Baltic Sea to discuss matters of interest and exchange knowledge with a group of expert lecturers. As we all know, a personal conversation will not replace any email or zoom contact.

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

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

Diving and Hyperbaric Medicine: Instructions for authors (summary)

(updated August 2021)

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

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

There is on-screen help on the platform to assist authors as they assemble their submission. In order to submit, the corresponding author needs to create an 'account' with a user name and password (keep a record of these for subsequent use). The process of uploading the files related to the submission is simple and well described in the on-screen help provided the instructions are followed carefully. The submitting author must remain the same throughout the peer review process.

Types of articles

DHM welcomes contributions of the following types:

Original articles, Technical reports and Case series: up to 3,000 words is preferred, and no more than 30 references (excluded from word count). Longer articles will be considered. These articles should be subdivided into the following sections: an **Abstract** (subdivided into Introduction, Methods, Results and Conclusions) of no more than 250 words (excluded from word count), **Introduction, Methods, Results, Discussion, Conclusions, References, Acknowledgements**, **Funding** sources and any **Conflicts of interest**. **Legends/captions** for illustrations, figures and tables should be placed at the end of the text file.

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

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

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

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

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

Formatting of manuscripts

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

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

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

Instructions for authors (Full version)
DHM Key words 2021
DHM Mandatory Submission Form 2020
Trial design analysis and presentation
English as a second language
Guideline to authorship in DHM 2015
Helsinki Declaration revised 2013
Is ethics approval needed?

DIVER EMERGENCY SERVICES PHONE NUMBERS

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ADSF
AUSTRALASIAN DIVING SAFETY FOUNDATION

Scholarships for Diving Medical Training for Doctors

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

There are two categories of scholarships:

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

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