Diving and Hyperbaric Medicine

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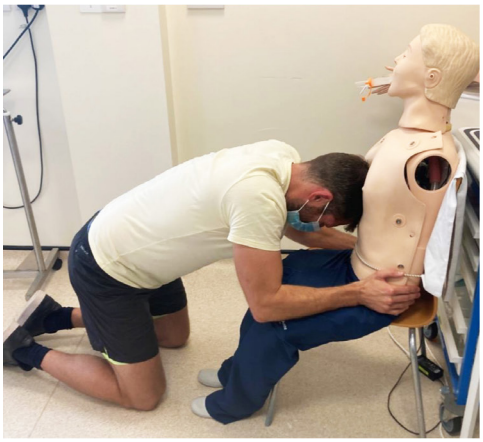
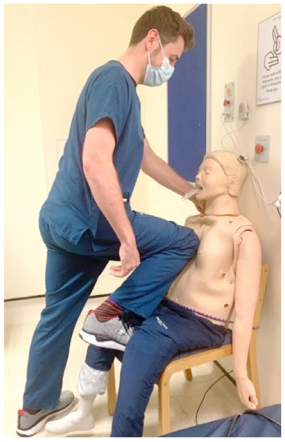


Manual and mechanical CPR in a diving bell

Decompression in transfer under pressure diving

Hyperbaric oxygen treatment in children

Australian snorkelling and breath-hold fatalities

Vitality and health in Dutch submariners

Hyperbaric oxygen in retinal artery occlusion

Glucose management in diabetics during HBOT

Utility of chest X-rays in diving medicals

Inner ear DCS in Western Australia

Gradient factors for military diving

Diving with psychotropic medication

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Effect of HBOT on cardiac troponin levels

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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

I write this having just returned from a fabulous trip to Europe where I had the honour of presenting to Professor Gerado Bosco's Master class in diving and hyperbaric medicine at Padova, Italy. It was an amazing experience lecturing in one of the oldest universities in the world. I am very grateful to Prof Bosco for this opportunity, and for the amazing hospitality shown by him and wife Natalie. From there I went to Porto for the EUBS meeting; a triumph of organisation, science and (again) hospitality by Óscar Camacho and his team. I offer my congratulations on a superb job by them.

This third instalment of DHM for 2023 is a big issue with a terrific mix of material of interest to those whose primary interest is either diving or hyperbaric medicine. There are two original articles by our British colleagues that focus on the challenge of providing effective cardiopulmonary resuscitation (CPR), particularly the chest compression component of CPR, in the cramped environment of a diving bell, both manually and using a mechanical device. Although a rare scenario, this highly original work provides guidance for those unfortunate enough to find themselves needing to resuscitate a dive buddy in this setting. Continuing in the occupational diving space Jan Risberg and colleagues summarise decompression procedures for 'transfer under pressure' diving; a diving style that shares methodologic elements with both surface oriented 'bounce' diving and saturation diving. This paper will be a valuable resource for groups engaging in this type of diving.

Figden Ayden reports the experience from a large series of 329 paediatric patients treated for various indications with hyperbaric oxygen. Caring for children in the hyperbaric setting has its own unique set of challenges and it is extremely useful to see such a substantial body of experience reported, with hints of optimising the experience of this particular patient group. John Lippmann publishes the latest in his series of thematically linked papers on fatalities in Australian diving, with analysis of contributing causes. This paper focuses on snorkelling and breath-hold diving; an important tourist activity in Australia, often undertake by inexperienced older participants with medical comorbidities.

Antoinette Houtkooper and colleagues evaluate the potential long-term adverse effects on health of service aboard submarines. Although there are many potential hazards to health associated with living in a submarine, there is little data on actual long-term health outcomes. This paper is a valuable contribution to this poorly researched area and will be a of great interest to naval health authorities. Jeremy Williamson and colleagues present observational data describing outcomes after treatment of central or branch retinal artery occlusion with hyperbaric oxygen. These potentially devastating but sporadic events would be virtually impossible to study in randomised trials and observational studies like this are critical to building knowledge about the efficacy of hyperbaric intervention.

Brenda Laupland and colleagues respond to expanding knowledge of the potential for hyperbaric oxygen to lower blood glucose levels with a survey to evaluate how hyperbaric units monitor and manage blood glucose levels in diabetic patients undergoing treatment. Jack Meintjes and colleagues address the controversial issue of whether routine chest X-rays are justified as part of medical evaluations for naval divers and submariners. Perhaps not surprisingly, the answer may be context sensitive, where 'context' refers to the expected prevalence of relevant pulmonary disease in the subject community.

Jeremy Mason and colleagues present data describing a cohort of recreational divers treated for inner ear decompression sickness (IEDCS) in Western Australia. This paper adds to the growing body of literature confirming that, despite a previous association with deep mixed gas diving, IEDCS can occur after recreational scuba air dives, and that there appears to be an association with persistent (patent) foramen ovale. Sven de Ridder and colleagues evaluate optimal decompression gradient factors for Belgian military divers by mapping gradient factor calculations on dives of known (and adequately safe) outcomes established through testing in large research programs.

Jack Marjot and colleagues present data describing the effect of hyperbaric oxygen exposure on myocardial troponin levels in patients with moderate risk of ischaemic heart disease undergoing hyperbaric treatment. Their report of a lack of any effect on troponin leak is reassuring in the face of speculation that hyperbaric oxygen might be harmful to compromised myocardium.

There are two review articles. Abraham Querido and colleagues describe the use of psychotropic medications and diving. This article is of high practical relevance to doctors who perform diving medicals and frequently encounter divers or diver candidates using such medications (which is increasingly common in modern society). Nicole Wong and colleagues review the known effects of hypoxia on the human electroencephalogram. This review was a necessary prequel to a study evaluating hypoxia recognition by divers that is currently underway.

Finally, Petra Magri Gatt and colleagues describe a case of cutis marmorata decompression sickness in an unusual distal distribution. This might have been precipitated by changes in perfusion during the dive associated with the configuration of wetsuit thermal protection worn.

Professor Simon Mitchell
Editor, Diving and Hyperbaric Medicine Journal

Cover photo: Different potential methods of delivering chest compressions in a diving bell environment as described by Johnson et al. in this issue.

Original articles

Delivering manual cardiopulmonary resuscitation (CPR) in a diving bell: an analysis of head-to-chest and knee-to-chest compression techniques

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Keywords
Cardiovascular; Deaths; Diving deaths; Diving incidents; Diving medicine; Diving research; Resuscitation

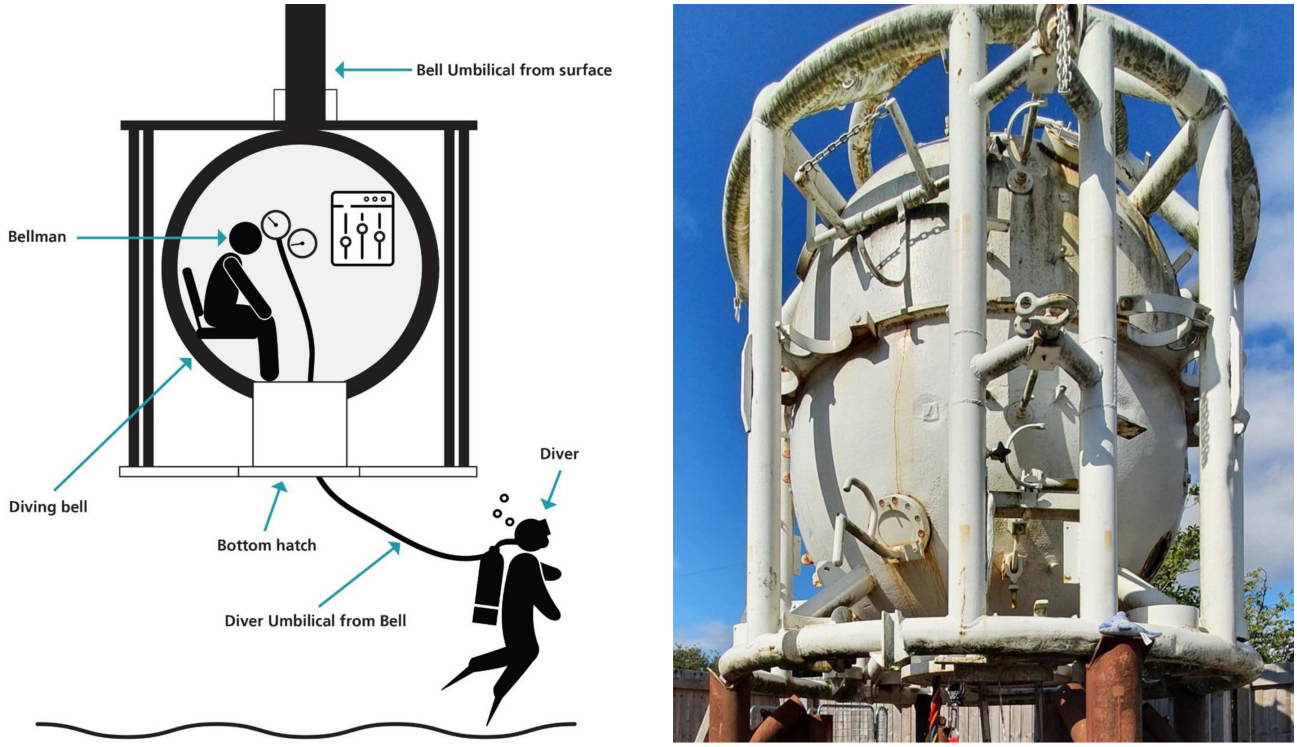
Abstract
(Johnson G, Bryson P, Tilbury N, McGregor B, Wesson A, Hughes GD, Hughes GR, Tabner A. Delivering manual cardiopulmonary resuscitation (CPR) in a diving bell; an analysis of head-to-chest and knee-to-chest compression techniques. Diving and Hyperbaric Medicine. 2023 September 30;53(3):172–180. doi: 10.28920/dhm53.3.172-180. PMID: 37718290.)
Introduction: Chest compression often cannot be administered using conventional techniques in a diving bell. Multiple alternative techniques are taught, including head-to-chest and both prone and seated knee-to-chest compressions, but there are no supporting efficacy data. This study evaluated the efficacy, safety and sustainability of these techniques.
Methods: Chest compressions were delivered by a team of expert cardiopulmonary resuscitation (CPR) providers. The primary outcome was proportion of chest compressions delivered to target depth compared to conventional CPR. Techniques found to be safe and potentially effective by the study team were further trialled by 20 emergency department staff members.
Results: Expert providers delivered a median of 98% (interquartile range [IQR] 1.5%) of chest compressions to the target depth using conventional CPR. Only 32% (IQR 60.8%) of head-to-chest compressions were delivered to depth; evaluation of the technique was abandoned due to adverse effects. No study team member could register sustained compression outputs using prone knee-to-chest compressions. Seated knee-to-chest were delivered to depth 12% (IQR 49%) of the time; some compression providers delivered > 90% of compressions to depth.
Conclusions: Head-to-chest compressions have limited efficacy and cause harm to providers; they should not be taught or used. Prone knee-to-chest compressions are ineffective. Seated knee-to-chest compressions have poor overall efficacy but some providers deliver them well. Further research is required to establish whether this technique is feasible, effective and sustainable in a diving bell setting, and whether it can be taught and improved with practise.

Introduction

There are many potential causes of acute illness in a saturation diver, including equipment and gas supply problems, trauma, and environmental issues. Although commercial divers are typically viewed as a healthy population, they are ageing; a large proportion are over the age of 45 and a significant number are 60 and over. Cardiac arrest can occur at any age and for a multitude of reasons, with medical causes becoming increasingly common with increasing age.[1] Sudden cardiac arrest is also more common in the male population that forms the vast majority of commercial divers.[2] Observations from recreational diving suggest that the majority of incidents are cardiac in origin, with some correlation with age.[3] There have also been well-publicised cardiac arrests in a saturation environment in recent years.[4,5]

Diving bells (Figure 1) are spherical containers with an internal diameter of 1.5–2.5 m which transport 2–4 divers and their personal equipment. Space inside the diving bell is extremely limited. Divers exit the bell to work, returning to the bell when it is time to return to the ship. One diver remains in the bell during the dive to monitor progress and undertake safety-related activities. Divers remain in a hyperbaric environment for continuous periods of up to a month before decompressing. The decompression process takes several days, with its duration governed by the depth

Figure 1
Diving bell schematic (left) and exterior appearance of a real diving bell



to which divers were compressed and the company's specific diving tables. Decompressing too quickly leads to harm, including decompression illness, which can be fatal.[6]

Effective management of a cardiac arrest in the diving bell environment is extremely challenging, with many factors affecting the provision of basic life support (BLS). The bell is small, and many have insufficient room in which to lie a casualty flat on the floor (Figure 2); some bell floors have protrusions rendering them unsuitable for casualty management. This has led to the development of techniques for the provision of BLS to a casualty in a seated or semi-recumbent position; these techniques include head-to-chest compressions, seated knee-to-chest compressions[7] and prone knee-to-chest compressions.[8] Whilst these techniques are taught on a variety of courses, no evidence informing their effectiveness or sustainability has been identified.

Early defibrillation, a mainstay of conventional cardiac arrest management, is not possible in a diving bell; there are currently no devices able to withstand the operating conditions. Defibrillation is possible within the saturation chamber on the ship but extricating a casualty from the seabed to the ship can take up to 40 minutes. In the absence of effective CPR during extrication, ongoing treatment in the chamber is likely to be futile.

Effective chest compressions are defined in current guidelines as delivered to a depth of at least 5 cm and not more than 6 cm, at a rate of 100–120 beats per minute (bpm),[9] whilst allowing effective recoil and keeping non-compression time to a minimum is vital.[10,11]

This study evaluated the efficacy, safety, and sustainability of three alternative chest compression techniques, in comparison to conventional manual chest compressions, when performed by a team of expert CPR providers.

Methods

TEAM

The team of expert CPR providers included three emergency medicine consultants, one emergency medicine research fellow, one critical care paramedic (and offshore medic) and one emergency medicine charge nurse; all were advanced life support providers or instructors and deliver CPR on a regular basis as part of their professional role.

The study team also included a diver medical technician (an active commercial diver), an offshore medic without extended life support training, and an anaesthetic nurse. All had received basic life support training and would be expected to deliver life support when needed in their professional roles, but none had ever delivered CPR outside of a simulated setting.

Figure 2
Diving bell interior with a simulated victim requiring resuscitation in the sitting position

SETTING AND EQUIPMENT

Data collection took place in the simulation centre of the Royal Derby Hospital. Chest compression efficacy data were captured using the Laerdal Resusci Anne QCPR manikin; this manikin has been used in multiple evaluations of CPR efficacy.[12–14]

OBJECTIVE

To assess the efficacy of existing techniques for providing chest compressions to a casualty in a diving bell environment. The techniques evaluated were head-to-chest compressions, seated knee-to-chest compressions, and prone knee-to-chest compressions.

PRIMARY OUTCOME

The primary outcome measure was the percentage of compressions delivered to target depth (50–60 mm).

SECONDARY OUTCOMES

The following secondary outcomes were recorded.

1. Depth of compressions
2. Difference between depth of compressions for each technique and the gold standard
3. Proportion of compressions with full recoil
4. Proportion of compressions delivered at target rate (100–120 bpm)
5. Rate of compressions
6. Proportion of compressions delivered with correct chest position
7. Sustainability of compressions
8. Adverse events reported by providers during the delivery of chest compressions

GOLD STANDARD

The 'gold standard' comparator was conventional manual chest compressions delivered by expert CPR providers.

DATA COLLECTION

Gold standard data acquisition

The team of expert CPR providers were allowed to familiarise themselves with the manikin and its outputs. Following a rest period, each provider then delivered chest compressions with the manikin supine on a trolley at an appropriate height.

Head-to-chest compressions

The manikin was placed in a seated position on a chair against a wall to simulate the conditions found in a diving bell environment (Figure 3). Head-to-chest compressions were delivered by all members of the study team for a 2-minute period (or until exhaustion).

Seated knee-to-chest compressions

The manikin was placed in the same seated position as for head-to-chest compressions (Figure 3). Knee-to-chest compressions were delivered by all members of the study team for a 2-minute period (or until exhaustion).

Prone knee-to-chest compressions

The manikin was placed face down across a seated provider's knee, with the knee placed in the centre of the chest (Figure 3). Hands were placed on the manikin's back, and compressions delivered by either lifting the knee, pushing down on the manikin's back, or both. Compressions were delivered by all members of the study team for a 2-minute period (or until exhaustion).

STOPPING CRITERIA

Testing of a chest compression technique was abandoned if no provider could deliver effective compressions for at least 30 seconds, or if provider harms (e.g., injury) related to the delivery of compressions were identified.

FURTHER EVALUATION

Techniques for which compression data could be effectively gathered were then evaluated further using a group of up to 20 emergency department doctor and nurse volunteers (different individuals for each technique), all of whom were trained in the provision of basic life support and who were expected to deliver chest compressions as part of their professional role. Each provider again delivered

Figure 3
Head-to-chest CPR (left); seated knee-to-chest CPR (middle); prone knee-to-chest CPR (right)

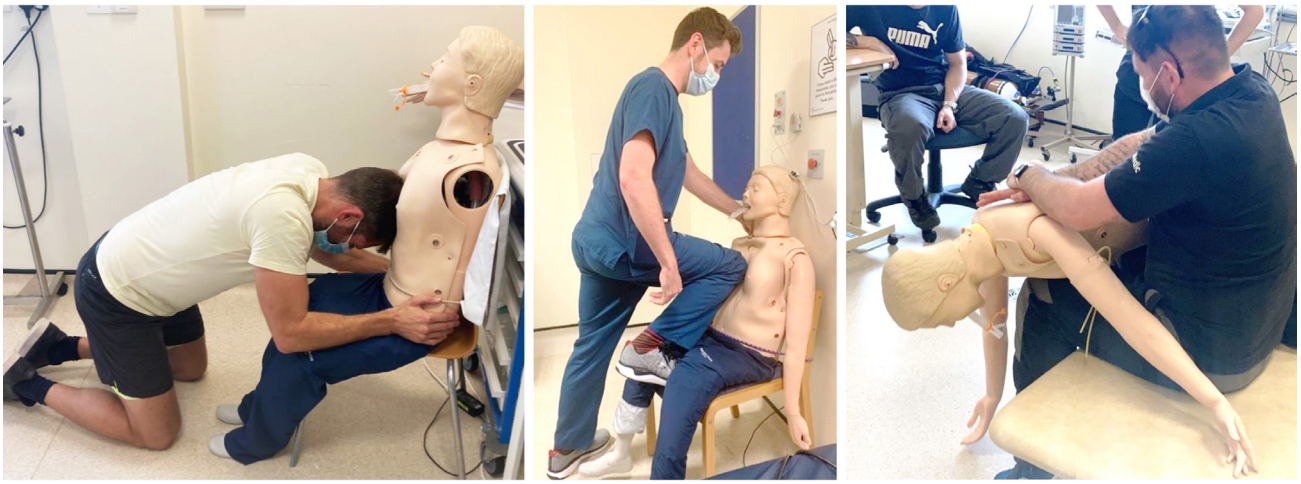


Table 1
Outcomes for conventional chest compressions delivered by expert CPR providers (compression-only); IQR – interquartile range

Provider number	Compression depth (%)	Recoil (%)	Mean depth (mm)	Rate (%)	Mean rate (bpm)	Position (%)
1	99	98	56	99	111	51
2	98	98	54	81	117	100
3	99	98	61	95	112	71
4	98	53	58	64	120	100
5	97	65	54	89	117	100
6	84	99	51	97	116	100
Median (IQR)	98 (2)	98 (25)	55 (4)	92 (14)	117 (4)	100 (22)

chest compressions for 2 minutes (or until exhaustion); no volunteer delivered more than one technique to avoid fatigue.

ADVERSE EVENTS

Any discomfort or injuries sustained during the delivery of chest compressions were recorded.

SUSTAINABILITY

If a provider ceased compressions prior to completion of the planned time period then their total time was recorded. At the end of each data collection period each provider was asked their opinion on whether the technique could be sustained for a period of 40 minutes; this interval represents the theoretical maximum time between commencing CPR in a diving bell and offloading a casualty onto the ship.

DATA ANALYSIS

Study team data were compared to volunteer data using the Mann-Whitney U test; if no significant difference was identified between the groups then pooled data are presented.

Results

GOLD STANDARD (CONVENTIONAL CHEST COMPRESSIONS)

The median percentage of compressions delivered to depth was 98%, interquartile range (IQR) 1.5%. Further efficacy data can be found in Table 1.

HEAD-TO-CHEST COMPRESSIONS – STUDY TEAM

Efficacy data for this technique can be seen in Table 2. Two study team members could not complete the planned 2-minute period of head-to-chest compressions, and no study team member felt that head-to-chest compressions would be sustainable for 40 minutes. All but one provider reported side effects, including headache and neck pain, from head-to-chest compressions.

HEAD-TO-CHEST COMPRESSIONS – FURTHER EVALUATION

The median percentage of compressions delivered to the required depth (study team and volunteer) was 32% (IQR

Table 2
Outcomes for head-to-chest compressions delivered in the seated position for up to two minutes, compression only; sustainability assessed by each provider; *emergency department staff volunteer; bpm – beats per minute; IQR – interquartile range

Provider number	Compression depth (%)	Recoil (%)	Mean depth (mm)	Rate (%)	Mean rate (bpm)	Position (%)	Adverse effects	Sustainable for 40 min?
1	80	57	55	94	102	100	Head pain	No (1 min 50 s)
2	64	60	51	98	115	100	Head pain	No
3	10	97	40	7	94	100	Head pain	No
4	7	60	44	99	113	100	Head pain	No
5	46	21	48	100	111	100	Headache	No
6	70	51	51	96	107	100	Headache	No
7	0	80	35	30	124	100	–	No
8	18	7	45	74	101	86	Head pain	No
9	78	66	55	99	109	100	Head pain, felt unwell	No (1 min 22 s)
10*	5	99	44	0	84	100	Head and neck pain	No
Median (IQR)	32 (61)	60 (24)	47 (58)	95 (7)	108 (11)	100 (0)	–	–

60.8%). The change in median compression depth compared to the gold standard was -14.5% (8 mm). Further efficacy data can be found in Table 2.

The combination of poor efficacy findings and adverse effects meant that only a single member of emergency department staff was recruited before further evaluation was abandoned on safety grounds.

SEATED KNEE-TO-CHEST COMPRESSIONS – STUDY TEAM

Two study team members could not complete the planned 2-minute period of seated knee-to-chest compressions, and only one study team member felt that seated knee-to-chest compressions would be sustainable for 40 minutes. No study team members reported adverse effects from delivering seated knee-to-chest compressions.

The median percentage of compressions delivered to the required depth was 15% (IQR 42%). The change in median compression depth compared to the gold standard was -21.8% (12 mm). Further efficacy data can be found in Table 3.

SEATED KNEE-TO-CHEST COMPRESSIONS – FURTHER EVALUATION

Twenty members of emergency department staff delivered seated knee-to-chest compressions; two were unable to complete the full 2-minute compression period. The median percentage of compressions delivered to the required depth was 7.5% (IQR 51.3%). The change in median compression depth compared to the gold standard was -25.5% (14 mm). Further efficacy data can be found in Table 4.

One provider felt that seated knee-to-chest compressions could be sustained for 40 minutes, with a further 13 stating it might be sustainable; multiple participants observed that the opportunity to swap providers every few minutes would improve its sustainability. The remaining six providers felt that seated knee-to-chest compressions could not be sustained for 40 minutes regardless of the ability to swap providers.

There was no significant difference between the proportion of compressions to depth using seated knee-to-chest delivered by the study team and that delivered by emergency department staff volunteers ($P = 0.45$); the median proportion of compressions to depth in pooled data was 12% (IQR 49%). The change in median compression depth for pooled data when compared to the gold standard was -25.5% (14 mm).

PRONE KNEE-TO-CHEST COMPRESSIONS

Prone knee-to-chest compressions could not be delivered by any study team member to a sufficient depth to register a sustained output from the manikin. There are therefore no efficacy data to present; no further evaluation was performed.

Discussion

This study represents the first evaluation of the three alternative chest compression techniques widely taught and employed in the commercial diving industry. None of the

Table 3

Outcomes for seated knee-to-chest compressions delivered in the seated position by study team members for up to two minutes, compression only; sustainability assessed by each provider; bpm – beats per minute; IQR – interquartile range

Provider number	Compression depth (%)	Recoil (%)	Mean depth (mm)	Rate (%)	Mean rate (bpm)	Position (%)	Adverse effects	Sustainable for 40 min?
1	48	67	49	4	95	100	No	No (44s)
2	34	80	47	99	106	100	No	No
3	3	58	39	89	112	100	No	No
4	6	53	41	69	110	82	No	No (18s)
5	0	60	37	99	110	100	No	Yes
6	98	25	60	100	107	100	No	No
7	12	97	41	84	117	100	No	No
8	15	63	43	94	105	100	No	No
9	92	46	58	91	114	100	No	No
Median (IQR)	15 (42)	60 (14)	43 (14)	91 (15)	110 (6)	100 (0)	–	–

Table 4

Outcomes for seated knee-to-chest compressions delivered in the seated position by emergency department staff for up to two minutes, compression only; sustainability assessed by each provider; bpm – beats per minute; F – female; IQR – interquartile range; M – male

Volunteer	Sex	Profession	Depth (%)	Recoil (%)	Depth (mm)	Rate (%)	R a t e (bpm)	Position (%)	Adverse effects	Sustainable for 40 min?
1	F	Nurse	1	100	38	19	93	100	No	Yes
2	M	Doctor	99	100	60	89	103	100	Knee pain	Maybe
3	F	Nurse	0	100	40	89	120	100	No	Maybe
4	M	Doctor	19	100	45	92	116	100	Calf pain	No
5	F	Nurse	1	100	39	95	108	100	No	No (56 s)
6	F	Doctor	59	100	52	84	99	100	Back pain	Maybe
7	F	Doctor	97	95	59	30	96	100	Knee pain	No (1 min 30 s)
8	F	Doctor	0	89	37	0	90	100	Knee pain	Maybe
9	M	Paramedic	0	100	29	96	104	100	Hip pain	Maybe
10	M	Doctor	50	87	49	100	108	100	No	Maybe
11	M	Paramedic	97	70	52	38	99	100	Hip pain	Maybe
12	F	Nurse	10	100	45	0	84	100	Hip pain	Maybe
13	F	Doctor	49	77	49	97	107	100	No	No
14	M	Nurse	58	100	49	97	107	100	Leg pain	Maybe
15	M	Doctor	12	81	41	62	102	100	No	No
16	F	Nurse	0	100	34	35	92	100	Leg pain	Maybe
17	M	Doctor	3	100	41	98	116	100	Knee pain	Maybe
18	M	Doctor	5	99	39	82	103	100	No	No
19	M	Doctor	1	100	36	71	101	100	No	Maybe
20	M	Doctor	0	100	29	83	103	100	No	Maybe
Median (IQR)	–	–	8 (51)	100 (7)	41 (11)	84 (58)	103 (9)	100 (0)	–	–

techniques have efficacy comparable to conventional chest compressions and none were perceived to be sustainable for a prolonged period. However, given that environmental limitations frequently prohibit the use of conventional chest compressions, alternative techniques are undoubtedly necessary if a casualty is to be given a meaningful chance of recovery from cardiac arrest. At least one recent incident has shown that neurologically intact survival is possible after a prolonged period with low cerebral oxygen delivery in the hyperbaric environment.[5] In the absence of defibrillator availability, high quality CPR is therefore the mainstay of treatment and essential to maintain cerebral and coronary blood flow.[11] Whilst neurologically intact survival after prolonged CPR is frequently perceived to be unlikely, consciousness and awareness during high quality CPR have been demonstrated, highlighting the value of effective CPR in optimising outcomes.[15]

Head-to-chest compressions were found to have an unacceptably high incidence of adverse events, with almost all providers reporting head and/or neck pain; this is even more concerning given the short study period (two minutes). The only study-team member who did not report an adverse event (Provider 7) was also the only member who failed to deliver any compressions to an adequate depth (mean 35 mm). Conversely, the two providers unable to complete the full 2-minute test period were those with the highest percentage of compressions delivered to an appropriate depth. This suggests that delivering effective compressions is not sustainable and is likely to be associated with adverse events.

The seated knee-to-chest compression data are more nuanced. The median compression depth from the pooled data (12%) suggests that this technique is ineffective; this was unaffected by provider gender (of relevance given most commercial divers are male). However, two providers delivered 98% of compressions to depth and five providers delivered 90%; conversely, many providers delivered none of their compressions to target depth. The reason for this variation is not yet established by this study, but may well be related to technique or biomechanical issues.

The perceived sustainability of seated knee-to-chest compressions was variable. Four providers (two study team members and two volunteers) were unable to complete two minutes of compressions, whilst four were able to deliver compressions of comparable depth to expert conventional CPR for the full 2-minute period. Multiple providers felt that seated knee-to-chest compressions could potentially be sustained for 40 minutes, especially if alternating of providers was possible. It is well-known that even conventional resuscitation techniques in a hospital environment are extremely tiring, and that provider-effectiveness decreases after as little as two minutes of delivering chest compressions.[16] Real-world assessment of seated knee-to-chest compression sustainability is needed.

Resuscitating a casualty in a seated position in a diving bell is undertaken through necessity, and the effectiveness of head-up CPR is not yet well-evidenced. It has been suggested that in some settings head-up CPR achieves improved cerebral blood flow compared to conventional CPR, but that this improvement requires a period of supine chest compressions first; the latter would not always be possible in a diving bell setting.[17]

No provider was able to administer prone knee-to-chest compressions effectively. Correct positioning of the casualty on the rescuer's knee was difficult, even with a manikin weighing considerably less than a diver and unencumbered by the supplemental equipment, water and cognitive load that would be associated with a real emergency.

This study has not considered mechanical CPR, a technique commonly used in clinical settings to reduce provider cognitive load and fatigue, and shown to be non-inferior to conventional chest compression.[18] The authors are only aware of one device that can be used in a saturation environment (the NUI compact chest compression device);[19] the efficacy of this device is presented elsewhere in this issue.[20] However, it will not be universally available across the industry due to varying safety standards and financial constraints, and mechanical failures are always a possibility; techniques for providing manual chest compressions in a diving bell environment are therefore still required.

Across the pooled study participants (and with exceptions discussed above), seated knee-to-chest CPR efficacy data are poor. However, in the absence of an alternative method for manual compression delivery, and without the availability of a mechanical CPR device, the only remaining alternative would be to forego chest compressions altogether. Given the close working relationships between divers, the lack of immediately available medical help, and the prolonged extrication time in an enclosed space, it is hard to envisage that bystanders would not wish to attempt resuscitation; not being able to do so would likely increase the likelihood of psychological consequences. We must therefore develop and teach the best possible techniques given the context and environmental constraints, both to optimise outcomes in casualties and to reduce the likelihood of second victim syndrome.[21]

LIMITATIONS

This is a laboratory study of chest compressions using an intelligent manikin; it is impossible to predict whether the efficacy findings seen in this study would translate to clinical effectiveness. Despite efforts to simulate appropriate casualty positioning, it is likely that the environmental limitations of a diving bell would affect the biomechanics of delivering compressions to a casualty.

Time off the chest to deliver ventilations would be necessary during the provision of compressions to a casualty, and this

time may vary between techniques; this variation has not been assessed in this study, and pauses in chest compressions are known to be associated with poor resuscitation outcomes.[22]

Resuscitation in a diving bell would present myriad challenges in addition to those experienced by those providing chest compressions in this study, including (but not limited to): lack of familiarity with techniques; presence of extraneous equipment; wet conditions; stress and cognitive load; the need to resuscitate a colleague; physical fatigue due to recent manual labour; and the need to focus both on the management of the casualty and on bringing the bell to the surface safely. As such, positive data should be considered exploratory, rather than as evidence of likely technique effectiveness in a real-world setting. Nonetheless, given the relative infrequency of cardiac arrests in this environment coupled with the challenges of delivering clinical research on this topic, it is extremely unlikely that clinical outcome trial data will ever be available.

Finally, the absolute nature of target ranges does not translate perfectly to clinical practice. A provider delivering every compression to 49 mm is deemed to have delivered 0% of compressions to depth by the intelligent manikin; it is debatable whether delivering these compressions 1 mm deeper translates to meaningful clinical impact.

Conclusion

Head-to-chest compressions have limited efficacy and are unsafe for providers; they should no longer be taught or used. Prone knee-to-chest compressions are ineffective and the technique should be abandoned.

Further research is required to establish the feasibility, effectiveness, and sustainability of seated knee-to-chest compressions in a diving bell environment, and to incorporate them into an algorithm for the management of a casualty in a saturation diving setting. It must be established whether the provision of effective seated knee-to-chest compressions is a teachable and reproducible skill. Caution should be applied to the teaching and implementation of seated knee-to-chest compressions until further data concerning optimum techniques in a diving bell setting are available.

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An evaluation of the NUI Compact Chest Compression Device (NCCD), a mechanical CPR device suitable for use in the saturation diving environment

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Abstract
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Introduction: Provision of manual chest compressions in a diving bell using a conventional technique is often impossible, and alternative techniques are poorly evidenced in terms of efficacy and sustainability. The first mechanical cardiopulmonary resuscitation (CPR) device suitable for use in this environment, the NUI Compact Chest Compression Device (NCCD), has recently been designed and manufactured. This study assessed both the efficacy of the device in delivering chest compressions to both prone and seated manikins, and the ability of novice users to apply and operate it.
Methods: Compression efficacy was assessed using a Resusi Anne QCPR intelligent manikin, and the primary outcome was the proportion of compressions delivered to target depth (50–60 mm). The gold standard was that achieved by expert CPR providers delivering manual CPR; the LUCAS 3 mCPR device was a further comparator.
Results: The NCCD delivered 100% of compressions to target depth compared to 98% for the gold standard (interquartile range 1.5%) and 98% for the LUCAS 3 when applied to both supine and seated manikins. The NCCD sometimes became dislodged and had to be reapplied when used with a seated manikin.
Conclusions: The NCCD can deliver chest compressions at target rate and depth to both supine and seated manikins with efficacy equivalent to manual CPR and the LUCAS 3. It can become dislodged when applied to a seated manikin; its design has now been altered to prevent this. New users can be trained in use of the NCCD quickly, but practise is required to ensure effective use.

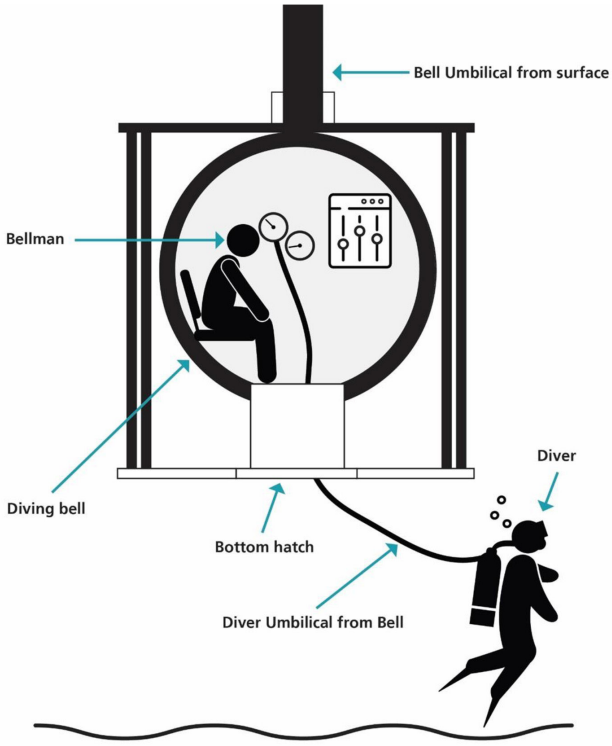
Introduction

Commercial diver deaths historically were predominantly related to trauma or equipment failure, but acute illness now underlies the majority of medical emergencies; this is due to an ageing workforce coupled with improved safety regulations and working conditions.[1]

Management of a sudden cardiac arrest whilst divers are working from the diving bell (Figure 1) is extremely challenging due to space-limited working conditions, a lack of flat surface on which to lie the casualty, the impossibility of immediate medical help, and restrictions on suitable equipment due to the hyperbaric, wet working environment. In addition, extrication times can be prolonged; it may take up to 40 minutes to bring a casualty from the working depth back to the saturation chamber on board the ship, and it may be several days before a casualty can be removed from the chamber and transported to land.

Mechanical cardiopulmonary resuscitation (mCPR) involves the use of a mechanical device to deliver chest compressions to a casualty in cardiac arrest. Whilst outcomes after cardiac arrest management with mCPR are not superior to manual CPR, mCPR has been recommended for situations where provider resource may be lacking, when a prolonged resuscitation is envisaged (to protect against provider fatigue), and in other settings where environmental or situational constraints preclude early initiation of high-quality manual CPR.[2] All of the above conditions are present in the context of a cardiac arrest in a diving bell.

Figure 1
Diving bell schematic (left) and exterior appearance of a real diving bell (right)



There has not been an mCPR device that could be used in this setting due to technical considerations: repeated exposure of the lithium battery and LCD screens to a hyperbaric environment; device size; the impact of the hyperbaric environment on device operation (e.g., a gas-driven device); and the impact of saltwater corrosion. However, the Norwegian company NUI have recently designed and manufactured the NUI Compact Chest Compression Device (NCCD) for use in the saturation diving setting, and specifically in a diving bell.[3] The NCCD (Figure 2) is a gas-driven piston device requiring manual trigger actuation to deliver each compression. The compression is delivered to a fixed depth and then held until the provider releases the trigger. The device is compact, and the piston and device body are held to the chest by a tough fabric strap encircling the casualty and secured by hook-and-loop fastening. The device is driven by gas at 1,000 kPa (10 bar) above ambient gas pressure (readily available in a saturation diving environment from the built-in breathing system) and can work whilst submerged.

Design and space constraints in some diving bells mean that there may be no flat surface on which to manage a casualty, and CPR may therefore need to be delivered with a casualty in a seated position. Manual techniques (e.g., seated knee-to-chest compressions) enable delivery of some effective compressions, but their efficacy does not compare well to conventional CPR in a laboratory environment.[4] Commercial divers also wear a neoprene hot-water suit that may affect the effectiveness of chest compressions if left in-situ. The impact of the seated position and hot-water suit on mCPR chest compression effectiveness have not previously been assessed.

There are no published data on the efficacy of the NCCD. This study evaluated:

1 The efficacy of the NCCD in providing chest compressions in a laboratory setting, compared to manual compressions administered by expert providers, and compared to the LUCAS 3 (Stryker, Kalamazoo, USA), an mCPR device in widespread use in healthcare settings.
2 The efficacy of the NCCD in a seated position, and when administering chest compressions through a (dry) hot-water suit.
3 The ability of new users to apply and use the NCCD after a short familiarisation session.

Methods

SETTING AND EQUIPMENT

Data collection took place in the simulation centre of the Royal Derby Hospital. Chest compression efficacy data were collected using the Laerdal Resuci Anne QCPR manikin. The mCPR devices used were the NUI Compact Chest Compression Device and the LUCAS 3. The hot-water suit was of a type in common industry use.

Figure 2
The NUI Compact Chest Compression Device (NCCD) in use on a manikin

PRIMARY OUTCOME

The primary outcome measure was the percentage of compressions delivered to target depth (50–60 mm).

SECONDARY OUTCOMES

Secondary outcome measures included:

1 Depth of compressions
2 Difference between depth of compressions for each device and the gold standard
3 Proportion of compressions with full recoil
4 Proportion of compressions delivered at target rate (100–120 bpm)
5 Rate of compressions
6 Proportion of compressions delivered with correct anatomical positioning on the chest
7 The impact of manikin position (supine vs seated) on the above metrics
8 The impact of a hot-water suit on the above metrics
9 Difference between the average depth of compressions for each device in each position/suit configuration, and the same device operated in its ideal working conditions (supine, no suit)
10 Percentage of compressions delivered to target depth by users new to the NCCD
11 Time taken to apply the NCCD by users new to the device
12 Other efficacy metrics as described above when the NCCD was operated by new users

GOLD STANDARD

The notional 'gold standard' was manual chest compressions delivered by expert CPR providers to a supine manikin. These data were collected in another study,[4] but are presented below for ease of comparison.

The study team of expert CPR providers included three emergency medicine consultants, one emergency medicine research fellow, one critical care paramedic/offshore medic, and one emergency medicine charge nurse; all were advanced life support providers or instructors who regularly deliver CPR as part of their professional roles.

DATA COLLECTION

Mechanical chest compression data acquisition

Study team members use the LUCAS 3 in their clinical practice and were familiar with its operation prior to this study. They received a short period (5–10 min) of device familiarisation and training in the use of the NCCD from a NUI representative.

Each mCPR device was applied to the manikin in accordance with the manufacturer's instructions and operated by the study team for a period of two minutes in the following conditions:

» Supine, no suit
» Supine, suit closed (i.e., on and zipped up)

» Supine, suit open (i.e., on but unzipped, such that the device can be applied directly to the front of the manikin)
» Seated (i.e., propped on a chair, back to a wall), no suit
» Seated, suit closed
» Seated, suit open

Application time and new user efficacy data

Twelve volunteer participants (eight nurses and four doctors) were recruited from the Royal Derby Hospital emergency department's staff as a convenience sample. They received the same period of device familiarisation and training as the study team in the use of the NCCD from a NUI representative.

Working in pairs, they were asked to apply the NCCD to a seated manikin and to deliver compressions for a 2-minute period. Volunteer one of each pair delivered compressions in the first attempt.

Time taken until delivery of the first compression was recorded. Following debrief and discussion the pair had a further attempt with volunteer two of each pair delivering compressions. Data from both attempts are reported for each pair.

Results

GOLD STANDARD (CONVENTIONAL CHEST COMPRESSIONS)

The median percentage of conventional chest compressions delivered by the study team to the required depth was 98% (interquartile range [IQR] 1.5%). Further efficacy data can be found in Table 1, and a fuller description of the acquisition of the gold standard data can be found elsewhere.[4]

MECHANICAL CHEST COMPRESSIONS

Data from both the NCCD and the LUCAS in all manikin positions and suit configurations can be seen in Table 2. In a supine position when operated by the study team both the NCCD and the LUCAS delivered compressions to the appropriate depth as well as, or better than, the gold standard in almost all suit configurations; the only supine suit configuration where an mCPR device did not perform equivalently to the gold standard was with the NCCD with an open hot-water suit (NCCD 94%, gold standard 98%).

There are no data for the LUCAS in the seated position because it could not be effectively applied; its mass and centre of gravity caused it to fall from the chest and no compressions could be delivered.

The NCCD became dislodged prior to the completion of the 2-minute compression period in all seated configurations on first attempts; data presented were therefore gathered after adjusting the fitting/application process during repeat attempts.

With a seated manikin and no hot-water suit in place the NCCD delivered 100% of compressions to an appropriate depth with full recoil. It performed inferiorly to the gold standard with the hot-water suit in either the open or closed position.

The mean compression rate with the NCCD in the supine, no hot-water suit configuration was 95 beats per minute (bpm), with only 5% of compressions delivered within the target rate range. The mean rate during all other data collection sessions for the NCCD was within the target range.

The change in mean compression depth for each device, position and suit configuration, when compared to both the gold standard and to that device's 'supine, no suit' configuration, can be seen in Table 3. The presence of a hot-water suit in any configuration had an impact on the depth of compression delivered with the NCCD; the LUCAS was comparatively unaffected.

Volunteer testing

One pair of volunteers were recalled to clinical duty and were unable to perform a second data collection period; their single use of the NCCD is presented with the five other paired data sets in Table 4.

The median percentage of compressions delivered to the target depth with the NCCD by new users was 97% (IQR 37%). The median application time was 61 s, and all pairs showed improvement between their first and second attempts (1st attempt median 67 s, 2nd attempt 45 s).

The median rate of compressions delivered by novice users using the NCCD was 100 bpm (IQR 10).

The percentage of compressions delivered with full recoil varied widely (median 91%, IQR 85%). It was noted that users achieving poor recoil had applied the device tightly which compressed the manikin's chest at baseline, rendering full recoil impossible.

Discussion

In a laboratory setting the NCCD delivered a greater percentage of compressions to target depth and with full recoil than expert providers performing conventional CPR. Whilst further assessment of device performance when operated by saturation divers is required, these early results are encouraging, and suggest that the NCCD may be a suitable device for the delivery of mCPR in a saturation diving setting.

Table 1
Outcomes for conventional chest compressions delivered in the supine position by expert CPR providers for four minutes, without the presence of a hot-water suit; bpm – beats per minute; IQR – interquartile range

Provider number	Compression depth (%)	Recoil (%)	Mean depth (mm)	Rate (%)	Mean rate (bpm)	Position (%)
1	99	98	56	99	111	51
2	98	98	54	81	117	100
3	99	98	61	95	112	71
4	98	53	58	64	120	100
5	97	65	54	89	117	100
6	84	99	51	97	116	100
Median (IQR)	98 (2)	98 (25)	55 (4)	92 (14)	117 (4)	100 (22)

Table 2
Outcomes for the NUI compact chest compression device (NCCD) and the LUCAS 3 mechanical CPR device, supine and seated, with a hot-water suit in a variety of configurations; bpm – beats per minute

Device	Manikin position	Hot-water suit position	Compression depth (%)	Recoil (%)	Mean depth (mm)	Rate (%)	Mean rate (bpm)	Position (%)
NCCD	Supine	No suit	100	100	59	5	95	100
NCCD	Supine	Suit closed	100	100	52	98	115	100
NCCD	Supine	Suit open	94	100	51	96	115	100
Lucas	Supine	No suit	98	100	54	98	101	100
Lucas	Supine	Suit closed	100	100	53	94	102	100
Lucas	Supine	Suit open	99	100	56	99	101	100
NCCD	Seated	No suit	100	100	58	98	111	100
NCCD	Seated	Suit closed	0	100	41	99	113	38
NCCD	Seated	Suit open	82	100	51	91	116	2
Lucas	Seated	No suit	–	–	–	–	–	–
Lucas	Seated	Suit closed	–	–	–	–	–	–
Lucas	Seated	Suit open	–	–	–	–	–	–

The NCCD is mechanical but not automatic; it requires manual activation of the piston by a trigger. The proportion of compressions delivered at the target rate was variable, and adherence improved over time. Across all tests the median percentage of compressions delivered at target rate by the study team was 98% (IQR 4%); this is more consistent and accurate than expert providers delivering conventional CPR. The percentage of compressions delivered at an appropriate rate by volunteers, with less exposure to NCCD use than study team members, was more variable; this suggests that a longer period of device familiarisation, together with simulated practice, is required to ensure that compressions are delivered at an appropriate rate by users new to the NCCD. A standardised approach to device training and familiarisation is required.

Resuscitation in a diving bell may have to take place in a fully seated position for at least some of the resuscitation effort, as it may be the only position possible due to space constraints. The NCCD was applied and used effectively in a seated position, with compression depths similar to the gold standard. However, it became loose or dislodged within the 2-minute test period both with and without the presence of a hot water suit, and had to be re-applied to complete data collection.

There is some evidence that head-up CPR, where compressions are delivered with the casualty in the reverse Trendelenburg position, may have a positive impact on outcomes after cardiac arrest.[5] However, it is felt to require a period of 'priming' with the casualty in a supine position in order to be effective; this may not be possible in a diving bell. The efficacy of head-up or seated CPR is not yet well-evidenced, and is possible that the position may reduce cerebral blood flow even during effective chest compressions.

Table 3
Change in mean compression depth compared to gold standard, and to each device baseline (supine, no suit); NCCD – NUI compact chest compression device

Device	Manikin position	Hot-water suit position	Mean compression depth (mm)	Change from gold standard (%)	Change from 'supine, no suit' (%)
NCCD	Supine	No suit	59	7.3	–
NCCD	Supine	Suit closed	52	-5.5	-12
NCCD	Supine	Suit open	51	-7.3	-14
LUCAS	Supine	No suit	54	-1.8	–
LUCAS	Supine	Suit closed	53	-3.6	-2
LUCAS	Supine	Suit open	56	1.8	4
NCCD	Seated	No suit	58	5.5	-2
NCCD	Seated	Suit closed	41	-25.5	-31
NCCD	Seated	Suit open	51	-7.3	-14

Table 4
Efficacy data and application times for the NUI compact chest compression device (NCCD) when operated by emergency department staff/novice device users; bpm – beats per minute; IQR – interquartile range

Team	Member roles	Application time (s)	Depth (%)	Recoil (%)	Rate (%)	Depth (mm)	Rate (bpm)	Position (%)
1	Nurse	67	97	100	65	53	101	100
	Nurse	45	64	100	39	50	99	100
2	Nurse	67	18	87	3	45	131	44
	Nurse	65	0	94	6	47	79	100
3	Nurse	76	97	5	0	53	87	100
	Nurse	61	61	100	9	50	93	100
4	Doctor	80	99	3	19	52	96	100
5	Doctor	55	98	100	56	53	100	100
	Doctor	40	99	0	75	58	103	100
6	Nurse	50	100	91	100	62	109	100
	Nurse	35	99	25	97	56	105	97
Median (IQR)	–	61 (20)	97 (37)	91 (85)	39 (63)	53 (5)	100 (10)	100 (0)

The safe removal of a hot-water suit will take time, and compression delivery during this period will be impossible. Given that reducing the no-flow fraction of a resuscitation is associated with better outcomes,[6,7] it was therefore important to assess the efficacy of the NCCD with the suit still in place; if suit removal could be avoided whilst still delivering effective compressions then this would reduce the no-flow fraction of diver resuscitation. Whilst both the NCCD and the LUCAS delivered effective compressions through the hot-water suit (either open or closed) in a supine position, both had at least one failed attempt where the device came loose or fell off before completion of the 2-minute test period. Applying the device was more difficult, with the suit becoming trapped and interfering with the strap or backboard. The presence of a hot-water suit (either open or closed) also reduced compression depth for the NCCD in both supine and seated positions; the most significant impact was with a closed suit in a seated position where the mean compression depth was reduced by over 30% in comparison to normal operation. Presence of the hot-water suit also impacted the accuracy of device positioning. It is therefore important that in practice the suit is removed, or cut away from the upper body, prior to application of the NCCD.

The median compression depth when the NCCD was applied and operated by new users compared favourably

Figure 3
The NCCD application process showing application of the NCCD with added neck strap (images courtesy of NUI)

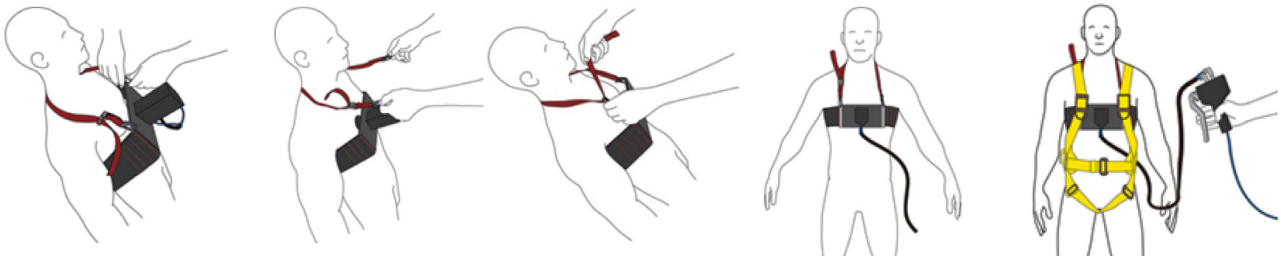
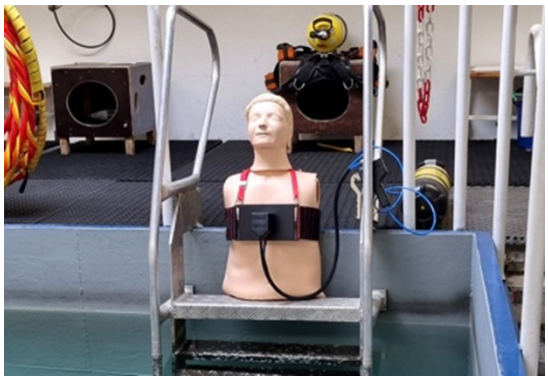


Figure 4
Updated NCCD with neck strap in use on a manikin (image courtesy of NUI)



to the gold standard, but with greater variation between providers compared to when the device was operated by the study team. The device was applied rapidly even by first-time users, and this time decreased with practise; this again highlights the importance of appropriate device training, coupled with the opportunity for simulated practice, prior to any real-world use of the device. It is worth noting that all volunteers were already trained in the delivery of both manual and mechanical CPR using other devices.

DEVICE CONSIDERATIONS

The NCCD requires manual trigger activation to deliver each compression, and its piston will remain extended as long as the trigger is depressed; this has potential safety implications for the patient as the chest could be held in the compressed position indefinitely. The user will need to deliver compressions continuously whilst being mindful of the compression rate. A key benefit of mCPR devices such as the LUCAS 3 is their ability to free up a team member and cognitively offload the team,[2] as they deliver compressions automatically and without user input once applied and started; neither of these benefits is offered by the NCCD.

Whilst the NCCD could be applied effectively in the seated position, it fell off multiple times during testing. This problem is likely to be compounded in a wet environment and with users less familiar with its operation. These findings were fed back to NUI, who have subsequently altered the design to include a neck strap, improving stability in the seated position without impacting the delivery of compressions (Figures 3 and 4). This modified device has not been tested in this study.

Both the group of expert CPR providers and the volunteers received only a short period of device training and familiarisation. Improvements in rate (expert providers), application time (volunteers) and effectiveness of application in a seated position were noted with repeated experience. This highlights the importance of adequate time for device training and familiarisation, together with the need for simulated practice, and is not an unexpected finding.[8]

The various design considerations that allow its use in a saturation diving environment (low profile, light, waterproof, non-electrical) may also render it suitable for use in a variety of non-standard resuscitation settings, including confined spaces, challenging casualty extrications, and other settings which mimic the restrictions present in a diving bell.

STUDY LIMITATIONS

This study reports efficacy metrics from an 'intelligent manikin' and any extrapolation to potential clinical outcomes should be cautious. However, given the relative infrequency of cardiac arrests in saturation diving and the inherent difficulty in performing research in that setting, meaningful clinical data collection is likely to be impossible. The industry is therefore reliant on simulation studies such as this, coupled with extrapolation of data from other settings, to inform best practice.

This small exploratory laboratory study was performed in 'ideal conditions'; further work should involve testing in a simulated, or actual, saturation diving setting, with the mCPR device operated for a longer period of time. Users in this assessment should be equipped authentically for modern saturation diving to evaluate any impact diving equipment may have on the users' ability to operate the device. This work should also explore the efficacy of the device when used by non-expert CPR providers. It should also evaluate the impact of the device on provision of ventilations during CPR. An optimal approach to device

training and familiarisation should be developed, and the rate of skill atrophy should be assessed to inform refresher training requirements.

The NCCD strap was noted to compress the manikin's chest when applied tightly, reducing the ability to record full recoil; it is not clear whether this effect would be present with real casualties.

Conclusions

The NCCD can deliver chest compressions at a rate and depth in line with existing guidelines, and in both a supine and seated position, as effectively as expert CPR providers delivering conventional chest compressions.

The presence of a hot water suit reduced device efficacy and hindered application. A seated casualty position led to the device becoming dislodged, requiring adjustment or re-application; this has led to a modification in device design (incorporation of a neck strap).

New users can apply the device quickly and use it effectively but require device training and familiarisation to use it optimally. It is vital that potential users receive appropriate training and practise prior to using the device in a medical emergency.

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Decompression procedures for transfer under pressure ('TUP') diving

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Abstract
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Background: There is an increasing interest in 'transfer under pressure' (TUP) decompression in commercial diving, bridging traditional surface-oriented diving and saturation diving. In TUP diving the diver is surfaced in a closed bell and transferred isobarically to a pressure chamber for final decompression to surface pressure.
Methods: Tables for air diving and air and oxygen decompression have been compared for total decompression time (TDT), oxygen breathing time as well as high and low gradient factors (GF high and low). These have been considered surrogate outcome measures of estimated decompression sickness probability (P_{DCS}).
Results: Six decompression tables from DadCoDat (DCD, The Netherlands), Defence and Civil Institute of Environmental Medicine (DCIEM, Canada), Comex MT92 tables (France) and the United States Navy (USN) have been compared. In general, USN and DCD procedures advised longer TDT and oxygen breathing time and had a lower GF high compared to MT92 and DCIEM tables. GF low was significantly higher in USN procedures compared to DCD and one of the MT92 tables due to a shallower first stop in many USN profiles compared to the two others. Allowance and restrictions for repetitive diving varied extensively between the six procedures. While USN procedures have been risk-assessed by probabilistic models, no detailed documentation is available for any of the tables regarding validation in experimental and operational diving.
Conclusions: Absence of experimental testing of the candidate tables precludes firm conclusions regarding differences in P_{DCS}. All candidate tables are recognised internationally as well as within their national jurisdictions, and final decisions on procedure preference may depend on factors other than estimated P_{DCS}. USN and DCD procedures would be expected to have lower P_{DCS} than MT92 and DCIEM procedures, but the magnitude of these differences is not known.

Introduction

Commercial diving is conventionally described as either surface-oriented or saturation. Most diving is surface-oriented: the diver enters and exits the water from a diving platform at surface. The diving depth is typically restricted to 50 metres of seawater (msw), and bottom time is usually restricted to a few hours to avoid excessive decompression time in the water. Surface decompression with oxygen ($SurDO_2$) may extend the bottom time somewhat for surface-oriented diving, but this diving method has faced criticism for a high incidence of decompression sickness (DCS).[1] Surface-oriented diving requires limited resources with respect to manning, training, equipment and breathing gas. For deeper work, saturation diving is used. The saturation diver will stay in a pressure chamber for many days or weeks, pressurised to the approximate ambient pressure at the diving work site. The diver will be transferred from the saturation chamber complex to the worksite by means of a closed diving bell and will typically be 'locked out' for 4–6 h per day. One important benefit of saturation diving is the amount of immersed working time, the drawback is the high cost of equipment and support organisation as well as a prolonged decompression time at the end of the saturation period. Decompression rate for saturation diving is typically in the range of 15–25 msw·d^{-1}.

Closed bell no-saturation diving is commonly associated with the term 'bounce diving'. While bounce diving is not clearly defined, it has a historical origin with the 'mini bell' system used in North Sea petroleum related diving in the 1980s.[2] The bottom time for deep bounce dives was limited and the diving procedures were optimised to exploit most of it as in-water diving time. This was achieved by rapid compression with the divers in the closed bell. Decompression was commonly done by a sequence of gas shifts and the final part of the decompression in a deck decompression chamber. The method has later generally been considered unsafe and Hamilton and Thalmann wrote "*DCS incidence has not been reported formally, but it was probably in the range of 10–20% for the more stressful dives*".[3] This quote was stated at a meeting in 1976.[4] Imbert

has reported the experience from Comex Services Company with heliox tables.[5] These tables, designed for diving down to 120 msw with bottom times up to 120 min achieved a 3.8% DCS incidence. The divers were decompressed in a closed bell breathing compressed air followed by oxygen. These historical 'bounce' decompression procedures were proprietary commercial products and have not been available to us.

Modern no-saturation closed bell diving is commonly termed 'transfer under pressure' (TUP) diving. Such diving is usually based on air as the breathing gas in the bottom phase and air or oxygen as the decompression breathing gas. Oxygen breathing during decompression increases the efficiency of the decompression and permits longer bottom times compared to conventional in-water air decompression. Depth is typically limited to 50 msw. Previous bounce decompression procedures as well as modern TUP procedures are based on isobaric transfer from the diving bell to the deck decompression chamber. Beyond this fact the differences are too extensive to allow a meaningful comparison.

To reduce the probability of DCS, the United Kingdom Health and Safety Executive (HSE)[6] has enforced bottom time limitations for surface-oriented diving. Similar restrictions apply in Norwegian waters.[7] These bottom time restrictions are relaxed for TUP decompression compared to in-water and surface decompression. This relaxation is motivated by the observed lower DCS incidence in TUP diving compared to conventional in-water and surface decompression methods.[1] A comparison of allowed[6,7] bottom times for a selected number of table depths is presented in Table 1.

To the best of our knowledge, there has only been a small number of TUP diving systems produced in recent years and TUP diving is not widely used in North Sea petroleum-related diving. However, the interest in TUP diving has been increasing as the diving method may be a cost-effective underwater intervention method bridging conventional surface-oriented diving and saturation diving. In 2019 the Norwegian Oil and Gas Association (NOROG, presently Offshore Norway) approached the editorial team of the Norwegian Diving and Treatment Tables (NDTT)[8] requesting a review of available TUP decompression tables. The editorial team produced an internal report in Norwegian language and presented the conclusions at the Bergen International Diving Seminar the same year. The report has not been published in the open domain. The current manuscript is an extended and restructured version of the original Norwegian report.

The objective of the present study is to review available decompression procedures applicable to TUP diving. Such diving has not been unequivocally defined. We will use the term for diving with a closed bell. After diving is finished, the divers will revert to the bell and be transferred isobarically to a deck decompression chamber without interventing decompression to surface pressure. Decompression after compressed air work shares some commonalities with TUP after immersed diving but falls beyond the scope of this work. For this work, we have limited our search of candidate procedures to those using air or nitrogen-oxygen (nitrox) as the breathing gas during the bottom phase and air, nitrox or oxygen as the breathing gas during decompression. We have not reviewed procedures for air or nitrox saturation diving.

Table 1

Schedules selected from transfer under pressure (TUP) candidate tables for comparison. Three to six bottom times (BT1–BT6) have been selected for each of the six chosen table depths. Schedules were selected to match United Kingdom Health and Safety Executive and Norsok-allowed[6,7] maximum bottom time for a non-TUP dive (BT lim. non-TUP), the longest allowed bottom time for a TUP-dive (BT lim. TUP) and an intermediate bottom time. Schedules printed in all tables are identified by bold typeface. Some tables lack schedules for the preferred bottom times, these have been identified with superscripts as follows: [1]DCD dry or wet bell, [2]DCD TUP, [3]MT92 12 msw O_2, [4]MT92 6 msw O_2, [5]DCIEM. Maximum bottom times for a direct ascent dive (No decompression [NoD] time) according to the US Navy Diving Manual Rev 7[9] are shown for comparison

Table depth (msw)	NoD time (min)	BT lim. Non-TUP (min)	BT lim. TUP (min)	BT 1 (min)	BT 2 (min)	BT 3 (min)	BT 4 (min)	BT 5 (min)	BT 6 (min)
15	92	180	240	180[1]	210[2,5]	**240**			
18	63	120	180	**120**	**140**	**180**			
24	39	70	180	70[2]	**80**	**100**	**140**	160[3,4]	180[1,5]
30	25	50	110	50[2]	**60**	80[2]	**90**	110[2]	
36	15	35	85	30[3]	40[2]	**60**	80[2]	**90**	
42	10	30	65	**30**	40[2]	**60**			

Methods

The study was initiated with a literature review. Searches were designed to identify TUP decompression procedures. Pubmed (https://www.ncbi.nlm.nih.gov/pubmed/) was searched with search terms *TUP* AND *diving* OR *Transfer under pressure*. The Rubicon Research Repository (http://archive.rubicon-foundation.org/xmlui/) was searched (last time 29.10.2019 due to the site subsequently closing) using the indexed term *Transfer under pressure*. Additionally, a Google (https://www.google.com) internet search on combinations of «TUP» «Decompression table» and «Decompression tables» was undertaken.

Independent of these searches the authors have reviewed diving procedures published by the United States (US) Navy,[9] Defence and Civil Institute of Environmental Medicine (DCIEM, Canada),[10] Comex MT92 table (France),[11] DadCoDat (DCD, the Netherlands),[12] National Oceanographic and Atmospheric Administration (NOAA, USA),[13] Norwegian Diving and Treatment Tables[8] and the Royal Navy (United Kingdom)[14] for their applicability for TUP diving. These procedures hold a number of decompression tables applicable for various diving procedures, e.g., air in-water decompression, air and oxygen in-water decompression, and SurDO_2. The combination of a specific table depth and bottom time will be termed *schedule*. We will use the term *profile* and *decompression profile* for the staged decompression stops and switches of breathing gas for a given schedule.

Three major diving contractors, operating in the North Sea, were contacted requesting access to decompression tables applicable for TUP diving.

To be considered for detailed review, the procedures should be publicly available, they should be based on compressed air or nitrox as the bottom gas and air, nitrox or oxygen as the breathing gas during decompression. The reason for not including other mixed gases (e.g., helium-oxygen or helium-oxygen-nitrogen) is the assumed limited relevance of these gases in commercial surface-oriented diving to depths not exceeding 50 msw. One of the tables – the DCD TUP tables – did not meet one of these criteria since these tables are commercial products not published in the open domain. This table was nevertheless included in the study for reasons described in the results and discussion sections.

We have included all publicly available decompression procedures intended for use with air or nitrox as the bottom gas and air or nitrox and oxygen as the decompression breathing gas. We expected this search strategy to include procedures developed for in-water decompression as well as closed bell decompression. The consequences of including in-water decompression procedures for TUP diving will be discussed later.

We have reviewed several parameters for each decompression table and summarised the findings in Table 2. A comparison of DCS probability has been assessed based on total decompression time (TDT) and oxygen breathing time. Increasing TDT and oxygen breathing time is expected to reduce the probability of DCS[15] though this reduction cannot be quantified. The distribution of decompression stops will likely influence this probability. Previous studies have reported increased DCS incidence and venous gas embolism when deep decompression stops were introduced.[16,17] However, we have no means of quantifying the effect of changing either TDT, oxygen breathing time or the distribution of decompression stops on DCS probability. Concerning comparison of TDT and oxygen breathing time, a comparison of all depth and bottom time combinations would be ideal. We found this to be too exhaustive and we have therefore compared a limited set of schedules (Table 1). We have chosen the table depths 15, 18, 24, 30, 36 and 42 msw. These table depths were selected due to their operational relevance. These table depths would allow the longest extension of bottom time using TUP compared to conventional surface-oriented diving according to UK and Norwegian regulations.[6,7] Whenever possible we have tried to review schedules for the longest allowed[6,7] bottom time for dives with TUP decompression, the longest allowed bottom time for decompression without TUP and a third bottom time midway between these two limits. The stipulated bottom time limitations as well as the schedules selected for comparison are presented in Table 1. Some of the published decompression tables did not provide schedules for each of the three preferred bottom times for every table depth. In these cases, we have chosen to analyse a schedule for a bottom time as close as possible to the preferred shared by all or a majority of the tables. A total of twenty-five schedules have been reviewed. Thirteen of these schedules were available for comparison across all tables.

We have retrieved TDT for each schedule as they were printed in the original decompression tables. It is common operational practice to breathe compressed air for 5 min after every 20–30 min of hyperbaric oxygen breathing to reduce likelihood of pulmonary and central nervous system (CNS) oxygen toxicity. DCD procedures include these 'air breaks' in the listed TDT in contrast to the other procedures. A 5 min air break after 30 min of oxygen breathing is mandated by USN and is recommended by the DCIEM procedures. MT92 doesn't provide information on air breaks. To facilitate comparison, we have included a 5 min air break for every 20 min of oxygen breathing when calculating TDT, independent of the advice given by the publisher.

We have calculated gradient factors[18] (GF) for the controlling compartment at the deepest stop (GF low) as well as GF at the time of surfacing (GF high) using the software Deco Planner version 4.5.1 (Global Underwater Explorers, High Springs, FL) configured with a descent and ascent rate of 10 msw·min^{-1}. Ascent rate and descent rate varies between

Table 2

Comparison of six TUP decompression table candidates published in four diving procedures; further details are presented in Appendix 1; DCD – DadCoDat; DCS – decompression sickness; msw – metres of seawater; NEDU – Navy Experimental Diving Unit; $SurDO_2$ – surface decompression with oxygen; TUP – transfer under pressure; USN – United Sates Navy; VGE – venous gas emboli

Feature	DCIEM	MT92 12 msw MT92 6 msw	DCD dry or wet bell DCD TUP	USN
Latest revision	2009	2012	2014 and 2015	2018
Algorithm/ parameter set	Kidd & Stubbs Serial perfusion	Haldanian	Haldanian	Thalmann E-L VVAL79
Algorithm/ parameter set published in public	No/No	Yes/No	No/No	Yes/Yes
Validation method	Experimental	Field experience	Field experience	Experimental/ probabilistic
Validation criteria	DCS and VGE	DCS	DCS	DCS
DCS estimate available for TUP candidate profiles	No	No	No	Yes
Publisher	Defence R&D Canada	French government	DadCoDat	USN (NEDU)
Deepest decompression stop breathing oxygen (msw)	9	12 (MT92 12 msw) and 6 (MT92 6 msw)	15 (TUP) and 9 (wet and dry bell)	9
Shallowest decompression stop (msw)	9	6	3	6
Air break	Recommended	Not stipulated	Mandatory	Mandatory
Compatibility with air tables	Yes	Yes	Yes	Yes
Compatibility with $SurDO_2$ tables	Yes	???	Yes	Yes

the tables, but changing these to 18 msw·min^{-1} will affect GFs by 2% or less which we consider of no practical consequence. The oxygen fraction (FO_2) in air was rounded to 21%. Breathing gas inspired O_2 fraction (F_IO_2) of the built-in breathing system (BIBS) was set to 85% as will be discussed later. The decompression model was ZH-L16B.[19]

We have presented the difference in TDT and O_2 breathing time by modified Bland-Altman plots and sorted them according to expected exposure severity. We have considered two different parameters describing decompression stress. First, the 'PrT' index (PrT = pressure x square root of time). PrT has been shown in epidemiological surveys[1] as well as statistical probabilistic models[20] to be positively associated with DCS. We have calculated PrT using Bar as the pressure unit (1 Bar = 10 msw) and minutes as the time unit. The second measure of decompression stress would be to use the estimated DCS probability (P_{DCS}) calculated by a probabilistic model. The P_{DCS} for schedules based on the USN decompression tables ($P_{DCS-USN}$) has been retrieved from Appendix E in Navy Experimental Diving Unit (NEDU) report 12-01.[20] We have used $P_{DCS-USN}$ as listed for 'VVAL-79 air/in-water O_2, 20 feet of seawater (fsw) last allowed stop' calculated by the NMRI98 probabilistic model.[15] The NMRI98 model estimates P_{DCS} at any time during or after decompression as a function of supersaturation in three compartments. Probability of DCS is calculated by the integration of this function. The NMRI98 model presumes exponential gas uptake and linear gas elimination, modified by the extent of hyperoxia in the breathing gas. The arguments to use $P_{DCS-USN}$ as the decompression severity index are further described in Results and Discussion.

We have presented table depths in units of msw although the SI unit Pa would be scientifically correct for a description of ambient pressure. However, most of the published decompression tables use msw as the depth and pressure unit, and comparisons and practical application of this work are facilitated by using the msw unit. The USN Diving Manual (USNDM)[9] has published depth in fsw. We have deliberately rounded 10 fsw = 3 msw to facilitate the comparison of similar table depths. This conversion implies a 2% rounding error since 10 fsw = 3.06 msw[9] and will overestimate the deepest schedule reviewed in this work (140 fsw/42 msw) by

0.3 msw. We consider this rounding error to be substantially less than the accuracy of the DCS estimate[20] of the USNDM and of no practical implication for the interpretation of the data. Metric conversion of USNDM table depths (10 fsw = 3 msw) will tend to give a lower P_{DCS} for USN schedules than stipulated.[20]

STATISTICAL ANALYSES

All six tables were compared pairwise, i.e., for any outcome variable there would be fifteen pairwise comparisons. First, differences in TDT, oxygen breathing time, GF low and GF high were analysed for normality with the Shapiro-Wilk test. If any of the pairwise comparisons didn't meet the requirement for normality distribution all comparisons for that test would be presented with median and interquartile range. Pairwise differences in TDT, oxygen breathing time, GF low and GF high have been statistically analysed with the Wilcoxon signed rank test. The association between $P_{DCS-USN}$ and the pairwise differences was measured with Pearson's correlation coefficient. As mentioned earlier not all tables have published all the schedules listed in Table 1. The number of comparisons for each of the twenty-five schedules thus range from 15–24. To compensate for familywise alpha inflation and Type I errors, α for each of these fifteen multiple comparison tests has been adjusted according to the Šidák correction. Statistical analysis was completed using Wizard for Mac Version 1.9.4 (www.wizardmac.com). $P < 0.05$ has been considered statistically significant.

Results

SEARCH FOR TUP CANDIDATE TABLES

Among the reviewed public decompression procedures (see Methods) we identified five tables applicable to TUP-Diving: the DCD 'BOX15';[12] DCIEM 'Table 2';[10] MT92 'Table 4';[11] MT92 'Table 5';[11] and the US Navy 'Air/O_2' table.[9] The most authoritative textbook in diving medicine[21] has no reference to TUP-procedures. The PubMed search gave no results. The Rubicon Research Repository search gave one result referring to a presentation by the UK Chief Inspector of Diving relating to North Sea offshore diving in 1979. The presentation discusses how the diving bell should be secured to the deck decompression chamber, but decompression tables were not discussed. The Google search gave references to Wikipedia, International Marine Contractors Association, N-Sea (a Dutch diving contractor), NDTT[8] and others who discuss TUP decompression, but without references to specific decompression tables. Due to the extensive internet references to N-Sea as well as a presentation of their TUP operations at the Bergen International Diving Seminar 2019 the company was approached to learn the details of the decompression tables. We were informed that N-Sea used proprietary tables developed by Prof. Wouter Sterk (Rob Borgonjen, personal communication 2019). Prof. Sterk was contacted in October 2022 requesting permission to review and analyse the decompression procedure for immersed diving with closed bell decompression breathing compressed air in the bottom phase and oxygen and compressed air in decompression. A set of tables designated 'AoxTUP2B' version October 2014 was submitted to the authors under a non-disclosure agreement. The publisher of the tables allowed the authors unrestricted access to read, review and analyse the tables. However, the specific details of individual profiles could not be shared with others. The AoxTUP2B tables are termed 'DCD TUP' in the present work and this is the sixth table included for analyses. Two other major diving contractors operating in the North Sea, Subsea 7 and Technip FMC, were contacted and submitted their decompression tables. Technip FMC had developed TUP procedures using the MT92 Table 5 (without air breaks). Subsea 7 had not developed specific TUP procedures but provided two sets of tables intended for in-water decompression breathing oxygen at 12 or 6 msw. These tables listed decompression schedules identical to or within 2 min of the MT92 Table 4 and Table 5. The decompression procedures from these companies have not been reviewed in further detail since they for all practical purposes are identical to the MT92 tables.

REVIEW OF PUBLISHED DECOMPRESSION TABLES

Details of the individual tables are presented in Appendix 1.We have summarised some of the main characteristics in Table 2. A typical arrangement for a TUP dive would be for the diver to enter the diving bell at the surface, lock the hatch and remain at surface pressure while the bell is lowered to working depth. The bell atmosphere will be compressed to ambient water pressure. United States Navy and DCIEM recommend a maximum descent (compression) rate of 23 and 18 msw·min^{-1} respectively, while DCD and MT92 don't provide advice on descent rate. The diver will be locked out of the bell once chamber and ambient pressure is equalised and return to the bell after finishing bottom time. Surfacing takes place with the hatch closed. Decompression may take place in the bell, in a deck (surface) decompression chamber or in a combination. The diver will typically breathe air or nitrox during the bottom phase and the first part of the decompression. At 12, 9 or 6 msw the breathing gas will be changed to oxygen through a built in breathing system (BIBS) with short periodic interruptions for breathing air (air breaks) to reduce the toxic effects of high pO_2. A typical profile is presented in Figure 1.

ASSESSMENT OF EXPOSURE SEVERITY

Probabilistic modelling[20] as well as operational experience[1] suggest that there is a positive association between diving exposure severity, expressed as PrT, and the outcome, expressed as P_{DCS}. However, P_{DCS} will depend on the decompression profile for any schedule. Increasing

Footnote: * Appendix 1 is available on DHM Journal's website: https://www.dhmjournal.com/index.php/journals?id=318

Figure 1

Typical dive profile for a TUP dive to 24 msw for 180 min. The diver breathes air (black line) during the bottom phase and oxygen (red line) at 9 msw and shallower. Oxygen breathing is interrupted for 5 min every 30 min (arrows) when the diver breathes compressed air ('air break'). The diver will be compressed and decompressed in the diving bell and a deck decompression chamber (broken line). This profile corresponds to the USN Diving Manual[9] 80 fsw/180 min in-water oxygen decompression procedure. Comparison of tables in the present work is based on a standardised 5 min air break for every 20 min of oxygen breathing

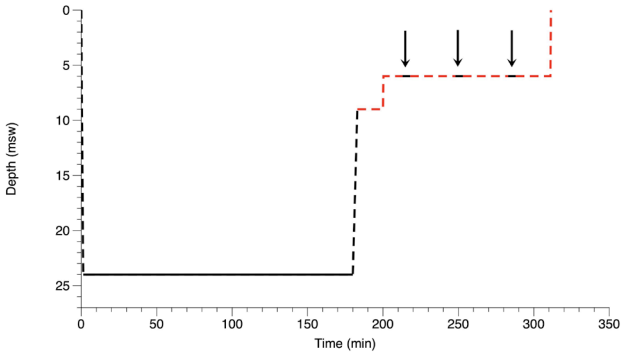
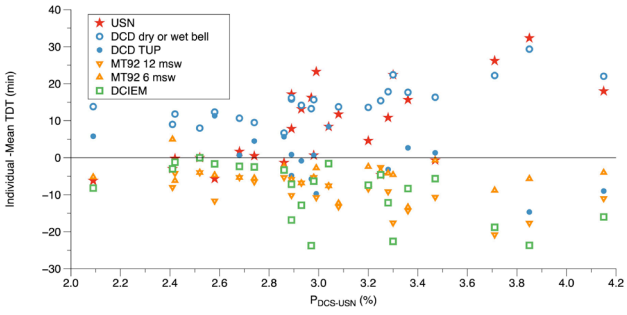


Figure 3

Differences in total decompression time (TDT) for twenty-five different schedules from six different decompression tables presented in a modified Bland-Altman plot. Each symbol represents a schedule with a specified table depth-bottom time combination (see Table 1). The difference between the stipulated TDT for a specific table and the mean TDT for all tables is plotted on the Y-axis. The schedules are sorted (X-axis) according to the expected DCS incidence for the USN schedule[20] ($P_{DCS-USN}$). Be aware that P_{DCS} will be different for the other tables. Some tables are missing certain schedules. DCD – DadCoDat; DCIEM – Defence and Civil Institute of Environmental Medicine; TUP – transfer under pressure; USN – United States Navy



decompression time or oxygen breathing time will reduce P_{DCS}. We have compared PrT and $P_{DCS-USN}$ for the twenty-five USN profiles listed in Table 1. As is shown in Figure 2, PrT and $P_{DCS-USN}$ is highly correlated up to a PrT of 31 ($r = 0.94$, $P < 0.001$). When PrT exceeds 31, the relationship is lost ($r = 0.29$, NS). Accordingly, PrT will not be a valid surrogate measure of the outcome of a dive adhering to USN air/in-water O_2 decompression table, at least not for dives with PrT > 31. We would expect that a decision to prefer a certain procedure in part would depend

Figure 2

Relationship between PrT (see text) and estimated probability of DCS ($P_{DCS-USN}$) according to the USN NMRI98 probabilistic model[20] for twenty-five US Navy Diving Manual air/in-water O_2 -schedules[9] compared in the present work (Table 1). Health and Safety Executive[6] and Norsok[7] regulations restrict bottom times for TUP diving to an upper PrT threshold of 42 (vertical broken line)

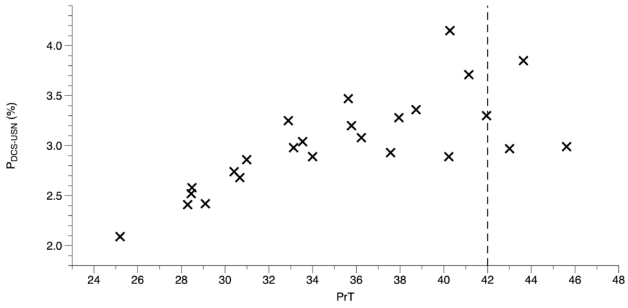
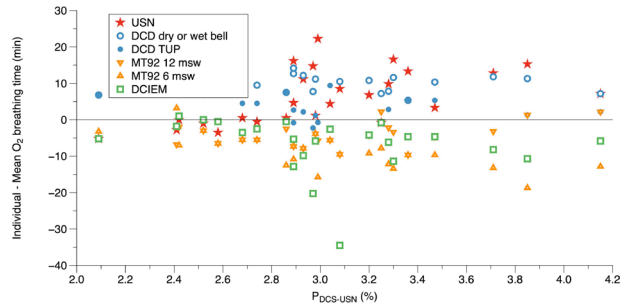


Figure 4

Differences in decompression oxygen breathing time (O_2 breathing time) for twenty-five different schedules from six different decompression tables presented in a modified Bland-Altman plot. Each symbol represents a schedule with a specified table depth-bottom time combination (see Table 1). The difference between the stipulated oxygen breathing time for a specific table and the mean oxygen breathing time for all tables is plotted on the Y-axis. The schedules are sorted (X-axis) according to the expected DCS incidence for the USN schedule[20] ($P_{DCS-USN}$). Be aware that P_{DCS} will be different for the other tables. Some tables are missing certain schedules. DCD – DadCoDat; DCIEM – Defence and Civil Institute of Environmental Medicine; TUP – transfer under pressure; USN – United States Navy



on the expected P_{DCS}. Given the inaccuracy of using PrT as a surrogate outcome measure, we have decided to present differences in outcome variables (TDT, oxygen breathing time, gradient factors) in increasing order of $P_{DCS-USN}$ instead. This decision will be discussed later.

COMPARISON OF DECOMPRESSION TABLES

Total decompression time and oxygen breathing time

Total decompression time (TDT) and oxygen breathing time vary between the published tables. Figures 3 and 4 give graphical presentations of this variation in modified Bland-Altman plots. The Bland-Altman plots compare the

Table 3

Pairwise comparison of six tables for differences in total decompression time; the data are median (interquartile range) minutes. A positive sign indicates that the table of the corresponding row has a longer TDT than the intersecting column. Statistically significant differences are indicated in bold ($P < 0.01$). A statistically significant positive correlation (Pearson correlation coefficient) between $P_{DCS-USN}$ and the difference in TDT between the tables of the intersecting row and column is indicated by * ($P < 0.05$) and ** ($P < 0.01$). DCD – DadCoDat; DCIEM – Defence and Civil Institute of Environmental Medicine; d/w bell – dry or wet bell; TUP – transfer under pressure; USN – United Sates Navy

Table	DCD d/w bell	DCD TUP	MT92 12 msw	MT92 6 msw	DCIEM
USN	**-8 (11)***	7 (24)	**16 (23)****	**11 (20)****	**10 (32)****
DCD d/w bell		15 (11)	22 (10)**	20 (10)*	22 (20)**
DCD TUP			**6 (8)**	6 (9)	**9 (5)**
MT92 12 msw				**-3 (7)**	-2 (9)
MT92 6 msw					3 (11)

Table 4

Pairwise comparison of six tables for differences in decompression oxygen breathing time; the data are median (interquartile range) minutes. A positive sign indicates that the table of the corresponding row has a longer oxygen breathing time than the intersecting column. Statistically significant differences are indicated in bold ($P < 0.01$) or italics ($P < 0.05$). DCD d/w bell: DCD dry or wet bell. Other table abbreviations are as per Table 2. A statistically significant positive correlation (Pearson correlation coefficient) between $P_{DCS-USN}$ and the difference in oxygen breathing time between the tables of the intersecting row and column indicated by * ($P < 0.05$) and ** ($P < 0.01$). DCD – DadCoDat; DCIEM – Defence and Civil Institute of Environmental Medicine; d/w bell – dry or wet bell; TUP – transfer under pressure; USN – United Sates Navy

Table	DCD d/w bell	DCD TUP	MT92 12 msw	MT92 6 msw	DCIEM
USN	-7 (11)*	2 (14)	**10 (14)**	**13 (17)****	**8 (21)**
DCD d/w bell		*5 (5)*	**15 (5)**	**20 (5)***	**14 (10)**
DCD TUP			10 (5)	15 (5)	**10 (4)**
MT92 12 msw				5 (10)**	*0 (7)***
MT92 6 msw					-4 (4)

stipulated TDT or oxygen breathing time for one profile to the mean of all profiles for each schedule. Each comparison is presented on the Y-axis and ordered in increasing order of expected incidence of DCS according to the USN procedure[9] on the X-axis ($P_{DCS-USN}$). As an example: for the 36 msw/60 min schedule the USN profile has an estimated DCS incidence of 3.47%. The mean TDT from all profiles for this schedule would be 68 min. MT92 12 msw, DCIEM, MT 6 msw, USN and DCD TUP recommend 57, 62, 67, 67 and 84 min of TDT respectively. The difference between the individual TDTs and the mean TDT (-11, -6, -1, -1 and +16 min respectively) can be seen as vertically stacked symbols at $P_{DCS-USN}$ 3.47% in Figure 3.

The figures suggest that USN and DCD TUP tables in general stipulates longer TDTs and oxygen breathing times than MT92 and DCIEM. Statistical analyses of these differences confirm this impression (Tables 3 and 4). Other statistical comparisons are summarized in Tables 3 and 4. The difference in TDT comparing either USN or DCD dry or wet bell to the MT92 and DCIEM increase as $P_{DCS-USN}$ increase (Table 3). Less consistent correlations were present for oxygen breathing time (Table 4). Differences in TDT and oxygen breathing time were not normally distributed for all pairwise comparisons and central location and distribution have been presented with median and interquartile ranges.

Gradient factors

GF low and GF high are measures of supersaturation in the controlling compartment at the deepest planned decompression stop and immediately after surfacing. None of the tested profiles exceeded 100% for GF low or GF high. As shown in Figure 5 and Table 5, USN and MT92 6 msw procedures have significantly higher GF lows than the other four procedures. GF high was significantly lower in USN and the two DCD procedures compared to the two MT92 and DCIEM procedures (Figure 6 and Table 6).

REPETITIVE DIVING

Repetitive diving is probably of limited practical interest for commercial TUP-diving. We will nevertheless provide a short summary comparing table characteristics for such diving.

Independent of depth and bottom time of the preceding dive, the DCIEM and USN tables allow a new single dive after an 18 h surface interval, DCD TUP tables 16 h while MT92 and DCD dry or wet bell tables allow a new single dive after 12 h. The decompression obligation for a repetitive dive is calculated based on a bottom time penalty. MT92 and USN impose a bottom time penalty as a nominal addition to the

Figure 5

Gradient factor low (GF low) for twenty-five different schedules (Table 1) from six TUP candidate tables presented in increasing order of estimated incidence of DCS for USN decompression schedule[20] ($P_{DCS-USN}$). Be aware that P_{DCS} will be different for the other tables. DCD – DadCoDat; DCIEM – Defence and Civil Institute of Environmental Medicine; TUP – transfer under pressure; USN – United States Navy

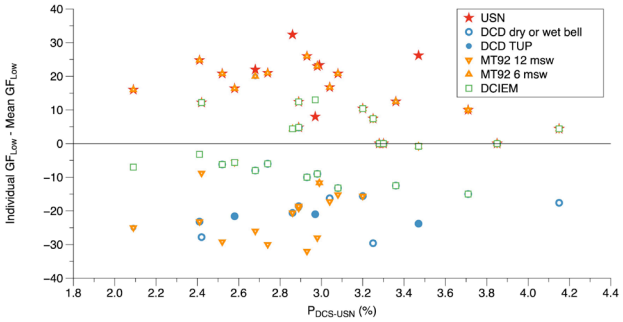


Figure 6

Gradient factor high (GF high) for twenty-five different schedules (Table 1) from six different TUP candidate tables presented in increasing order of estimated DCS incidence for the USN decompression schedules[20] ($P_{DCS-USN}$). Be aware that P_{DCS} will be different for the other tables. DCD – DadCoDat; DCIEM – Defence and Civil Institute of Environmental Medicine; TUP – transfer under pressure; USN – United States Navy

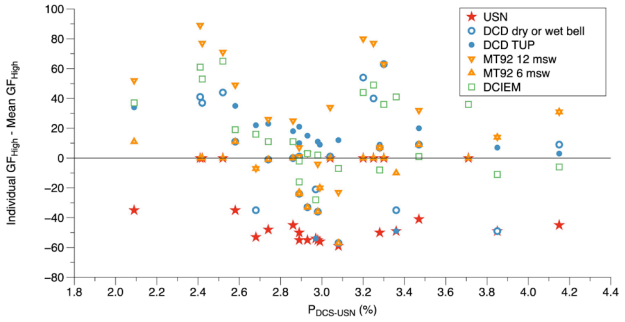


Table 5

Pairwise comparison of six tables for difference in low gradient factor (GF low); the data are median (interquartile range). A positive sign indicates that the table of the corresponding row has a higher GF low than the table of the intersecting column. Statistically significant differences are indicated in bold ($P < 0.01$) or italics ($P < 0.05$). A statistically significant positive ($P < 0.05$) or negative ($P < 0.01$) correlation (Pearson correlation coefficient) between $P_{DCS-USN}$ and the difference between the tables of the intersecting row and column is indicated by * and ## respectively. DCD – DadCoDat; DCIEM – Defence and Civil Institute of Environmental Medicine; d/w bell – dry or wet bell; TUP – transfer under pressure; USN – United Sates Navy

Table	DCD d/w bell	DCD TUP	MT92 12 msw	MT92 6 msw	DCIEM
USN	**32 (14)**	**31 (34)**	**28 (48)**$^{\#\#}$	0 (0)	**23 (28)**
DCD d/w bell		0 (6)	0 (37)	**-30 (12)**	*-18 (31)*
DCD TUP			0 (19)	*-25 (33)*	0 (23)
MT92 12 msw				**-25 (36)***	-15 (22)*
MT92 6 msw					14 (27)

Table 6

Pairwise comparison of six tables for difference in high gradient factor (GF high); the data are median (interquartile range). A positive sign indicates that the table of the corresponding row has a higher GF high than the table of the intersecting column. Statistically significant differences ($P < 0.01$) are indicated in bold. A statistically significant negative correlation (Pearson correlation coefficient) between $P_{DCS-USN}$ and the difference between the tables of the intersecting row and column is indicated by $^{\#}$ ($P < 0.05$) and $^{\#\#}$ ($P < 0.01$). DCD – DadCoDat; DCIEM – Defence and Civil Institute of Environmental Medicine; d/w bell – dry or wet bell; TUP – transfer under pressure; USN – United Sates Navy

Table	DCD d/w bell	DCD TUP	MT92 12 msw	MT92 6 msw	DCIEM
USN	11 (18)	1 (19)	**-17 (14)**	**-20 (16)**$^{\#}$	**-17 (21)**
DCD d/w bell		-7 (6)	-23 (9)	-26 (7)	-24 (8)
DCD TUP			**-15 (11)**	**-18 (5)**	**-19 (4)**
MT92 12 msw				0 (6)$^{\#}$	-1 (12)$^{\#\#}$
MT92 6 msw					1 (5)

actual bottom time. DCIEM procedures provide 'repetitive factors' – a factor by which to multiply the actual bottom time of the repetitive dive to reach an 'effective bottom time'. Decompression is then calculated based on the effective bottom time. DCD procedures don't provide guidance on repetitive diving.

The number of possible repetitive dive combinations is too large to allow analysis. We have compared bottom time penalties prescribed for the three tables allowing repetitive dives within a 12 h surface interval. We have arbitrarily chosen three different depth/bottom time combinations. The repetitive dive was presumed to take place to the same depth as the first dive (Table 7). For these three schedules

Table 7

Bottom time (BT) penalty (minutes), that is, time to be added to the actual bottom time of the repetitive dive, for three selected schedules according to DCIEM, MT92 and USN procedures. A large bottom time penalty indicates a conservative procedure. DCIEM – Defence and Civil Institute of Environmental Medicine; SI – Surface interval; USN – United Sates Navy

Depth (msw)	BT first dive (min)	SI (h)	BT repetitive dive	BT penalty (min) for a repetitive dive to the same table depth		
				MT92	DCIEM	USN
15	140	2	120	50	108	109
24	80	8	60	5	18	29
30	55	6	40	5	12	33

Table 8

Maximum allowed bottom time (min) for non-exceptional dives according to United States Navy (USN) and Defence and Civil Institute of Environmental Medicine (DCIEM) decompression tables. Bottom times equal to or exceeding United Kingdom Health and Safety Executive transfer under pressure (TUP) limits are indicated by *

Table depth (msw)	TUP bottom time limit (min)	USN	DCIEM
15	240	*	140
18	180	*	120
21	180	*	100
24	180	170	80
27	130	*	60
30	110	*	55
33	95	*	55
36	85	*	50
39	75	*	45
42	65	*	45
45	60	*	40
48	55	*	35
51	50	*	35

the USN and DCIEM procedures impose a similar bottom time penalty while MT92 imposes less penalty.

OPERATIONAL CONSIDERATIONS

All tables provide decompression schedules for air diving to 50 msw. However, most of the HSE maximum allowed bottom times exceed those permitted for DCIEM non-exceptional dives (Table 8). The USN restricts bottom time for a non-exceptional dive to 24 msw to 170 min (10 min shorter than the HSE limit[6]) (Table 8), for all other table depths the HSE-imposed bottom time limitations are within the non-exceptional dive range. None of the tables detail use of nitrox as the bottom breathing gas. All except the MT92 procedures provide routines for flying after diving.

Discussion

PROBABILITY OF DCS – SINGLE DIVES

All tables presented in this work are recognised as de facto national standards in their originating country. Most of them are additionally applied in commercial diving in foreign jurisdictions. They have been revised for decades. There is no reason to suggest that any of them should be considered unsafe, however one of the objectives of this study was to analyse whether there are systematic differences between them suggesting that one procedure might provide higher protection for DCS compared to others.

Only one of the tables, the DCD TUP tables, has been specifically developed for TUP diving, though the MT92 12 msw is designed for closed bell intervention in addition to surface supplied and wet bell use. Neither the DCD dry or wet bell nor the MT92 tables specifically mention TUP as the diving method but state that they are intended for use with diving bells. The USN Air/Oxygen and DCIEM tables were designed for in-water decompression. The divers' decompression environment may affect DCS incidence. In-water decompression with oxygen will provide the diver with FiO_2 = 100%. When decompressing in a dry environment, leakage from the breathing masks (BIBS-system) will reduce FiO_2 depending on mask fitting as will be discussed later. Immersion, thermal stress and physical activity may also affect DCS probability and validation of decompression profiles should ideally be performed as close as possible to the operational reality.

We have compared the tables with respect to gradient factor low, gradient factor high, TDT and oxygen breathing time. The USN and DCD tables in general stipulate longer TDTs and oxygen breathing times than MT92 and DCIEM (Tables 3 and 4). Longer decompression time and increased FO_2 during decompression is expected to decrease DCS incidence.[15] The difference in TDT between USN and DCD vs MT92 and DCIEM, tended to increase as $P_{DCS\text{-}USN}$ increased (the difference was positively correlated to $P_{DCS\text{-}USN}$ Table 3). The difference between the tables will thus be most pronounced for dives with a high expected DCS incidence. As expected, the increased TDT and oxygen breathing time caused surfacing GF high to be lower for USN

and DCD compared to MT92 and DCIEM (Table 6). These results suggest that USN and DCD TUP procedures should give a lower DCS incidence than MT92 and DCIEM tables.

It should be recognised that the stipulated figures for $P_{DCS-USN}$ are valid for the USN air/in-water O_2 table profiles only. While the longer TDT and oxygen breathing time in USN and DCD procedures would suggest a lower DCS incidence compared to MT92 and DCIEM tables, the distribution of decompression stops may modify this assumption. The GF low was higher in USN profiles than all other procedures except MT92 6 msw. This is due to a shallower first decompression stop in the USN profiles compared to MT92 6 msw. A high GF low implies that the inert gas supersaturation in the controlling compartment is close to the allowed tension threshold. While adding a deep decompression stop, which would reduce GF low, previously was thought to reduce DCS incidence in deeper diving, this has been difficult to confirm. Two studies[16,17] suggested increased venous gas embolism and a negative effect on DCS incidence when deep stops were introduced. Procedures with deep stops may therefore actually have a higher probability of DCS compared to other procedures with similar TDT and oxygen breathing time, but as previously stated, we have no means of quantifying this effect.

While the results suggest that DCS incidence would be lower in USN and DCD procedures than MT92 or DCIEM, we have no access to a probabilistic model allowing us to assess the effect size of this difference. However, consideration should be given when decompressing with shorter TDT or oxygen-breathing time than USN unless a higher DCS probability is acceptable. The USN air/in-water O_2 procedure is based on the Thalmann E-L probabilistic model.[20] The VVAL79 parameter set used assumes FiO_2 = 99.5% during in-water decompression at 9 and 6 msw. The same parameter set assumes FiO_2 = 85% when breathing 100% O_2 during surface decompression with oxygen accounting for some leakage in the BIBS masks. A somewhat higher DCS probability than that stipulated by $P_{DCS-USN}$[20] (Figure 2) would thus be expected when USN air/in-water O_2 procedures are used in a TUP setting when divers breathe O_2 through BIBS in the diving bell or chamber.

The assessment of the safety performance of various decompression procedures should ideally be based on experimental studies with many subjects testing all possible profiles. Table revision should be based on large epidemiological studies documenting table performance in operational diving.[22] In practice this is impossible due to logistical constraints and tables are tested with a limited number of experimental dives followed by monitoring of operational dives. Reports from operational dives have several limitations. Data on depths, bottom time and decompression times may be inaccurate since these values previously were registered manually rather than based on electronic depth monitoring. Even more importantly, operational diving will not reflect a homogenous distribution of diving depths and bottom times. It seems likely that diving to bottom times not requiring staged in-water decompression stops ('no-decompression dives) would predominate. Epidemiological studies[23] as well as the outcome of probabilistic models of USN decompression tables[20] strongly suggest that DCS incidence will increase as a function of diving depth and bottom time. Unless the profiles are presented, caution should be taken in interpreting reports of DCS incidence in operational diving. Assessment of the safety of these tables is challenged by the fact that publicly available data on experimental testing or robust epidemiological data are scarce or non-existent. This is further discussed below.

DCS PROBABILITY – REPETITIVE DIVES

Comparing repetitive dive procedures is complicated by the fact that the tables have different procedures for such diving as well as a difference in minimum surface interval to allow a new single dive. For repetitive dives with a surface interval shorter than 12 h the MT92 procedures will advise shorter bottom time penalties than DCIEM and USN (Table 7) and we would presume that this would affect DCS incidence.

The procedures may be compared with respect to minimum surface interval allowing new dives and bottom time penalties for repetitive dives. For a given surface interval a procedure not allowing new dives would be considered more conservative than those allowing repetitive dives. Similarly, a long bottom time penalty would be considered more conservative than a short bottom time penalty. A broad summary, listing the procedures and repetitive schedules (Table 7) in decreasing order of conservatism, may be presented such:

- Surface Interval > 18 h: all procedures will accept the following dive as a new single dive
- Surface interval 12–18 h: DCD TUP>>USN >DCIEM>>MT92 and DCD dry or wet bell
- Surface interval 0–12 h: DCD>>USN>DCIEM>MT92

DOCUMENTATION AND VERIFICATION OF ALGORITHMS AND PARAMETER SETS

The USN decompression tables have been developed based on a publicly accessible algorithm (Thalmann E-L[24]) and parameter set.[20] Acceptance criteria and verification have been clearly described.[20] USN tables have been revised recently (2018) by NEDU scientists. These tables are expected to be continuously developed and improved due to the institutional commitment backed up by a recognised team of scientists. A large database has allowed USN to develop probabilistic models that may predict the outcome of any decompression schedule. The present study investigated the USN Diving Manual Rev 7 air/in-water O_2 decompression schedule as one of the candidates for TUP models. The schedules in this procedure were developed using the Thalmann E-L deterministic model with VVAL79 parameter set.[20] The NEDU has developed this parameter

set to allow DCS probability to stay within USN acceptable limits. The $P_{DCS-USN}$ has been assessed with two models, one of them being NMRI98[15] calibrated with 4,335 dives. The schedules reviewed in the present manuscript have $P_{DCS-USN}$ ranging from 2.1–4.2% (Figure 2). While this is the prediction of the NMRI98 probabilistic model,[15] we are not aware of any experimental verification of the air/in-water O_2 decompression schedules published in the USN Diving manual.

The DCD procedures were initially published in 1988, but revised 2015. They are edited by two experienced physicians. The details of the underlying algorithms and parameter sets have not been published in the public domain. Operational experience with DCD tables has been reported in a proceedings document published 1990.[25] A total of eleven cases of DCS had been reported in 25,902 dives (0.04%). About 50% of all dives were 'no-decompression dives. The details of DCS cases were not described. The Dutch NDC diving school experienced ten cases of DCS during 1,091 SurDO_2 dives adhering to the DCD procedures during 1998–1998.[26] A later publication[27] reported experience with 1,607 helium-oxygen-nitrogen (trimix) dives in the North Sea during 2005 and 2006. Seven cases of DCS were reported, six of these were skin DCS occurring during a four-week period. The cluster of skin DCS was believed to be caused by insufficient heating of the diving bell and surface decompression chamber. Heating and isolation were provided and though a single neurological DCS occurred later, no further incidents of skin DCS were experienced. However, we have not been able to find reports detailing DCS incidence related to testing or operational use of either of the two DCD tables reviewed in this work.

The DCIEM air decompression tables were updated regularly until 1986. There were some minor changes in 2009, but the tables have for all practical purposes been unchanged since 1986. The algorithm has been described in general terms, but the parameter set has not been published. These tables have been extensively tested in strictly controlled experiments using DCS as well as venous gas emboli as the outcome measure.[10] However, there is no information available presenting how the tables have been adjusted based on the result of these experiments. Nishi et al.[28] claim that high bubble grades (Grade III to IV in more than 50% of the subjects) will predict a DCS incidence of more than 5%. The narrative describing the latest Canadian tables don't specify whether this has been used as an acceptance criterion. The 'Introduction' chapter in these tables states that the tables were used in 5,000 dives up to 1967, 2,000 dives during 1967–1971, more than 1,200 dives during 1983–1986 and more than 1,500 dives during 1986–1991. In total, the Canadian tables have been tested in approximately 10,000 dives. However, details are absent regarding which profiles have been tested. Accordingly, it is impossible to know the safety of individual diving methods, depths or bottom times. Sawatzky[29] reported in his thesis that 73 nitrox dives with in-water oxygen decompression were monitored with Doppler for venous gas emboli (VGE). Another report noted that 27 profiles had been tested with 276 exposures.[30] However, neither the profiles nor the DCS incidence was reported.

The MT92 tables were first published in 1974, revised in 1992 and last published 2012.[11] The underlying principles of the algorithm have been published[31] but not with sufficient details to allow an independent review. Data describing the incidence of DCS has not been published for most of the diving methods except air decompression. However, Imbert et al.[31] reported a significant reduction in DCS incidence when the 1992 tables were introduced – in particular for dives with high inert gas load (PrT > 35). Imbert and Bontoux[32] reported that the Comex database in 1987 held data for 573 man-dives from 40 different table profiles with oxygen breathing from 12 msw during decompression. Similarly, a total of 814 man-dives using 55 profiles were logged using the decompression procedures of breathing oxygen from 6 msw. However, neither the profiles used nor the number of DCS experienced are described. We are unaware of other data describing DCS incidence for dives adhering to MT92 in-water oxygen decompression procedures.

In summary, the USN procedures seem to be the best documented and validated tables published. The NEDU is staffed by scientists continuously developing the tables. We would nevertheless underscore that none of the published procedures, including those of USN, have reported DCS incidence with TUP-diving using air as the bottom gas and air and oxygen as the decompression breathing gas.

PRACTICAL AND OPERATIONAL DIFFERENCES

A selection of table characteristics is presented in Table 2. The MT92 12 msw and DCD TUP tables are the only tables specifically designed for closed-bell decompression. The DCD TUP tables contain provision for transfer from the diving bell to the surface decompression chamber with bell and chamber pressurised to 15 msw. A maximum of 15 min is allowed for such transfer without the need for extension of table bottom time. No other procedure allows flexibility for such personnel transfer.

The procedures differ for handling a situation in which oxygen-breathing must be interrupted, e.g., due to acute oxygen toxicity or BIBS failure. The DCD TUP procedures contain highly specific air decompression procedures for such cases while the DCD dry or wet bell air diving procedures don't detail emergency air decompression. The DCIEM and MT92 procedures state that air decompression should follow the conventional air decompression procedure. USN have a detailed, but complicated, procedure for conversion from oxygen-breathing to air-breathing decompression stops.

Oxygen breathing at raised ambient pressure involves a risk for acute and chronic oxygen toxicity. The details of this are beyond the scope of the present work, but the probability

of oxygen toxicity will rise as PO_2 and exposure time increases. On the other hand, resting state, non-immersed exposure and air breaks will delay toxicity onset. We believe that differences in pulmonary oxygen toxicity will mainly depend on total oxygen breathing time rather than maximum PO_2 since most of the oxygen exposure takes place at 6 and 3 msw for all but DCIEM procedures. The probability for clinically relevant pulmonary oxygen toxicity should nevertheless be small as long as air is used as the bottom breathing gas and bottom time is limited to HSE/Norsok regulations. Risberg and van Ooij[33] recommended the daily hyperoxic exposure, calculated as 'K', not to exceed 50 for multiday diving. The longest oxygen breathing time identified in this work is 113 min in the USN 24 msw/180 min profile. This profile has K = 33, significantly less than the proposed limit. However, consideration should be taken when oxygen enriched gas is breathed during the bottom phase since this may significantly enhance pulmonary oxygen toxicity.

The DCIEM advice for bottom time limitations is more restrictive than those imposed by UK and Norwegian regulators (Table 8). We would presume that this would make DCIEM procedures less relevant for commercial diving than the others.

LIMITATIONS

This study has several limitations. Most importantly we don't have the methods to assess the effect size on expected DCS incidence of a given difference in decompression time or oxygen breathing time. While we have presented these differences for some schedules, we are unable to quantify to what extent a given difference will make a relevant difference in DCS incidence. To compensate for this, we have presented the differences in increasing order of $P_{DCS\text{-}USN}$ (Figure 3 and Figure 4). For schedules with a high $P_{DCS\text{-}USN}$ we advise careful consideration if alternative procedures suggest a significant reduction in TDT or oxygen breathing time than those prescribed by USN. Secondly, we have not compared all schedules. Given the fact that we have compared at least three bottom times for each of the selected table depths, we nevertheless would presume that we have disclosed the general performance of each procedure concerning the analysed parameters.

We have standardised air breaks to 5 min for every 20 min of oxygen breathing in our calculation of TDT and total air breathing time. The reason for this is that recommendations for air breaks vary significantly between the procedures. However, we expect that most users will include an air break to reduce pulmonary and CNS oxygen toxicity. A 5 min air break is mandated for every 20 min of oxygen breathing in the DCD tables except for the 3 msw stop. Our standardisation will extend TDT marginally by 0–5 min for these tables. The USN requires a 5 min air break for every 30 min of oxygen breathing, while the DCIEM tables recommend similarly. The MT92 tables do not stipulate air breaks. Our standardisation has thus extended TDT by a maximum of 20, 15 and 10 min of air-breathing time relative to the original published decompression schedules in the MT92, DCIEM and USN tables respectively. Without standardisation of air breaks the TDT of DCIEM and MT92 will be shortened by 15–20 min for profiles with the longest oxygen breathing times. Without standardisation of air breaks the contrasts in TDT between MT92 and DCIEM tables on one side and USN and DCD tables on the other side would increase compared to those presented in Table 3.

The DCD TUP tables have been provided to the authors under a commercial-in-confidence and non-disclosure agreement. The reason for this restriction is the commercial value of the product for the publisher. The non-disclosure term is an evident concern since it will prevent independent control of the results presented in this manuscript. However, the authors recognise that N-Sea has been the single largest operator of North Sea offshore TUP diving operations in recent years, and the inclusion of the tables would be highly relevant for individuals, organisations, companies and regulators interested in developing TUP diving capacity. We believe that the benefit of including the DCD TUP tables outweighs the disadvantages. The DCD TUP tables illustrate a concern related to proprietary and confidential decompression tables. Such proprietary tables were common in the past, particularly in offshore saturation and mixed gas diving, but are less common in diving in developed countries today. There may exist TUP-tables we are unaware of since our search strategy has been based on open sources. We strongly support sharing the contents of decompression tables in the open domain. In addition, there is a need for better epidemiological data on DCS occurrence in occupational diving. Electronic monitoring of dives is comparatively inexpensive. Sharing exposure and outcome data from commercial diving would allow future studies to compare performance of decompression tables.

We have compared the relative safety of different TUP candidate decompression tables. However, we have no method to assess the absolute DCS risk of these tables when applied for closed bell decompression. We have compared decompression time and oxygen breathing time of candidate tables to those of USN air/in-water oxygen decompression tables. Even though we have referred to the expected DCS probability of USN air/in-water oxygen tables, we would like to reiterate that these estimates are valid for in-water decompression only. TUP decompression will usually avoid the thermal stress, hydrostatic forces and work related to in-water decompression. On the other hand, it is possible that leaks of the BIBS masks may give a lower inspiratory oxygen fraction compared to that of the immersed diver's breathing equipment. The direction and effect size of these factors remain to be studied.

Finally, the focus of this work has been a comparison of parameters related to DCS probability. Economic, practical,

legislative and standardisation factors will, in the end, have an important impact on table selection. Assessment of these factors is beyond the scope of this work.

Conclusions

This is a published systematic approach to the evaluation of decompression tables applicable to TUP diving. The present work has identified six candidate tables from DCD (the Netherlands), DCIEM (Canada), MT92 (France) and the US Navy. They are all recognised by their national authorities and widely used in commercial air and SurDO$_2$ diving. When compared with respect to TDT and oxygen breathing time, the USN and DCD tables are more conservative than DCIEM and MT92 tables. However, detailed safety records from experimental or field diving are not available for any of these tables. It is thus not possible to claim that they have been satisfactorily validated. The probabilistic model of the USN suggests that their air/O$_2$ decompression table should perform with a DCS incidence comparable to air diving.

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Hyperbaric oxygen treatment in children: experience in 329 patients

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Keywords
Barotrauma; Carbon monoxide; Hearing loss sudden; Safety; Side effects

Abstract
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Introduction: Paediatric patients, like adults, may undergo hyperbaric oxygen treatment (HBOT) in both life-threatening situations and chronic diseases. There are particular challenges associated with managing paediatric patients for HBOT. This paper documents the indications, results, complications, and difficulties that occur during HBOT for a large cohort of paediatric patients and compares them with adult data in the literature. Methods used to reduce these difficulties and complications in children are also discussed.
Methods: This was a 15-year retrospective review of paediatric patients treated with HBOT at two hyperbaric centres. Between January 2006 and June 2021, patients under the age of 18 who received at least one session of HBOT were included.
Results: Three hundred and twenty-nine paediatric patients underwent a total of 3,164 HBOT exposures. Two-hundred and fifty-four patients (77.2%) completed treatment as planned and 218 (66.5%) achieved treatment goals without complications. Two patients treated for carbon monoxide poisoning exhibited neurological sequelae. Amputation was performed in one patient with limb ischaemia. Middle ear barotrauma events occurred in five treatments. No central nervous system oxygen toxicity was recorded during the treatments.
Conclusions: This patient series indicates that HBOT can be safely performed in pediatric patients with low complication rates by taking appropriate precautions. The cooperation of hyperbaric medicine physicians and other physicians related to paediatric healthcare is important in order for more patients to benefit from this treatment. When managing intubated patients an anaesthesiologist may need to participate in the treatment in order to perform necessary interventions.

Introduction

According to the Undersea and Hyperbaric Medical Society (UHMS), hyperbaric oxygen treatment (HBOT) is a medical treatment based on breathing 100% oxygen in hyperbaric chambers at pressures higher than 101.3 kPa (1 atmosphere absolute pressure [atm abs]) although the inspired pressure of oxygen should be at least 142 kPa (1.4 atm abs) or more for clinical effect.[1] Treatment is performed in monoplace or multiplace hyperbaric chambers at pressures (in most indications) between 203 to 284 kPa (2.0 to 2.8 atm abs) for 90–120 minutes. Oxygen is inhaled through a mask, hood or endotracheal tube or from ambient air if the chamber is pressurised with 100% oxygen. The physical, physiological and biochemical effects of hyperbaric oxygen are described elsewhere.[2–6] Indications for HBOT are determined by the UHMS and the European Committee of Hyperbaric Medicine (ECHM), and consensus reports with evidence levels are published.[1,7] Hyperbaric oxygen treatment in Turkey is administered for indications determined by the Ministry of Health in association with these reports. These indications have been included in the reimbursement list by the Social Security Institution.[8] While HBOT is the primary treatment for decompression sickness and arterial gas embolism, it is used as adjunctive treatment in carbon monoxide (CO) and cyanide poisoning, necrotizing soft tissue infections, necrotising fasciitis, clostridial myonecrosis (gas gangrene), crush injury, compartment syndrome and other acute traumatic ischaemias, diabetic and non-diabetic chronic ulcers, thermal burns, radiation injuries, chronic refractory osteomyelitis, sudden visual and hearing loss, femoral head necrosis and some type of intracranial abscesses.[1,7–9] The number of daily sessions is determined according to the disease. The most common side effects observed in treatment are barotraumas due to raised pressure.[10]

Currently, there are a total of 54 centres providing HBOT in general hospitals and independent centres in Turkey (53 multiplace facilities, and one monoplace chamber facility). However, there are no hyperbaric chambers in any paediatric hospitals. Due to the unique needs of the paediatric age group and the potential high risk of inter-hospital transfers, especially for intensive care patients, there may be delays in treatment. There is a lack of studies in the literature on the problems encountered during HBOT in children.

This study aimed to document the indications for HBOT among paediatric patients in Turkey, and to appraise treatment results, problems (such as ear equalisation problems and anxiety), and other adverse events. A further goal was to discuss strategies to minimise difficulties and adverse events in children.

Methods

Approval for this retrospective study was obtained from the Ethics Committee of İzmir Tepecik Education and Training Hospital (2021/05-15). Files and system records were searched for paediatric patients (0 to 18 years of age) who received HBOT between 1 January 2006 and 15 May 2021. Demographic data, indications, HBOT protocol and number of sessions, problems encountered during the treatments, complications, and treatment outcomes were extracted.

Hyperbaric oxygen treatments were performed in multiplace hyperbaric chambers (Barotech, Turkey) and in sessions of 90–120 min at 203–304 kPa (2–3 atm abs). Informed consent was obtained from the parents before the treatment. HBO staff accompanied all children throughout the treatments. In elective cases, we showed the children the hyperbaric chamber before treatment, introduced them to the staff and explained all aspects of the treatment. Children under the age of four underwent treatment together with family member inside the chamber. In addition, we allowed children with anxiety symptoms (who did not want to enter the hyperbaric chamber alone, were afraid, cried and refused treatment) to attend the first session with a family member. Family members who were taken to the hyperbaric chamber with the patient where necessary, were also examined before hyperbaric exposure and chest X-rays were taken. However, X-rays were not ordered in life-threatening emergencies.

Since CO poisoning usually affects other family members many of our paediatric patients were treated together with their families. We allowed them to take toys that do not pose a fire hazard as a distraction during compression. There are two monitors in the hyperbaric chambers, and patients can watch movies on these monitors during treatment. In order to make the treatment more enjoyable for paediatric patients, we turned on their favorite, age-appropriate animations or cartoons. We taught the children ear equalisation manoeuvres before the treatment, with those who failed to perform such manoeuvres to stretch or chew gum. For younger children we allowed them to suck a bottle. Patients who could not adapt to an oxygen delivery mask, very young children and infants breathed oxygen through a hood. Intubated patients were ventilated using a pneumatic ventilator. The endotracheal tube cuff was filled with liquid instead of air to prevent changes in cuff volume during changes in ambient pressure.

During the treatments, patients were monitored through portholes and cameras, and verbal communication was provided via intercom. We used the chamber medical lock for drugs and small equipment required during the treatment and the entry lock of the hyperbaric chamber for larger medical equipment, patient or physician entrances and exits. Our hyperbaric chambers have a fire extinguishing system against the risk of fire. In addition, while no electrical devices were taken into the chamber, the oxygen level inside was closely monitored throughout the treatments and the chamber was ventilated as needed. During the treatment, the temperature was controlled with a specially designed air conditioning system.

Data were entered into a Microsoft Excel (Microsoft®, US) spreadsheet under the categories of: complete healing, minor morbidity (partial improvement in hearing loss, minor amputation, skin grafting or surgical debridement), major morbidity (no improvement in hearing loss, major debridement/amputation and neurologic sequelae), death, and complications related to HBOT. These data were subject to statistical analysis with Microsoft 365 Excel 2021.

Results

PATIENTS AND CONDITIONS TREATED

During a 15-year period, we treated a total of 329 patients aged 0–18 years with eight indications in two HBOT centres (Table 1). We performed a total of 3,164 patient sessions in 329 patients (mean treatments per patient 9.6, standard deviation [SD] 12.6, 95% confidence interval [CI] 4.2–15.1). Two hundred and fifty four patients (77.2%) completed their treatment as planned. Forty-eight patients (14.6%) terminated their treatment voluntarily. In three patients (0.9%), treatment was discontinued due to a different treatment plan of the responsible physician. We could not obtain information about the treatment outcome of 24 patients (7.3%). No patient discontinued treatment due to claustrophobia.

The mean age was 12 years (range 0.75–18). The first session in treatment of carbon monoxide poisoning and necrotising anaerobic soft tissue infections lasted for 90 min at 284 kPa (2.8 atm abs) using a protocol incorporating three 25-minute oxygen periods separated by five-minute air breaks, and for other indications lasted 120 min at 243 kPa (2.4 atm abs) using a protocol incorporating three 30-minute oxygen periods again separated by five-minute air breaks. Ninety-six patients (29.2%) underwent treatment with family members; 36 were treated together because they were younger than four years old, and 60 patients because their families also had CO poisoning. Thirty-seven patients breathed oxygen through a hood. Six patients were treated while still receiving ventilation support.

We treated a total of 234 (71.1%) paediatric patients with CO poisoning aged between nine months and 18 years (30.3% of total sessions). The source of CO was gas leaking from a stove (wood or coal) in 176 patients (75.2%) and a water heater in the bathroom in 46 patients (19.7%). Four patients (1.7%) were poisoned by trying to warm up with a barbecue

Table 1
Indication, patient demographics and number of sessions for 329 children treated with HBOT; F – female; M – male; SD – standard deviation

Indication	*n*	Mean (SD) age	Sex (F:M)	Mean (SD) sessions
Carbon monoxide poisoning	234	12 (4.7)	135:99	4.1
Sudden hearing loss	37	16 (2.7)	19:18	15.6
Delayed wound healing	23	11 (4.3)	12:11	28.3
Chronic refractory osteomyelitis	17	15 (4.4)	10:7	29.4
Crush injury, compartment syndrome	8	14 (4.3)	2:6	20.5
Femoral head necrosis	4	12 (2.5)	2:2	40.3
Soft tissue radionecrosis	4	14.5 (5.9)	3:1	28.5
Central retinal artery occlusion	2	12 (2.8)	1:1	20.0
Total	329	12 (4.7)	184:145	9.6 (12.6)

Table 2
Treatment outcomes by indication in 329 children treated with HBOT; CO – carbon monoxide

Indication	*n*	Complete recovery	Recovery with minor morbidity	Recovery with major morbidity	No recovery	Complication	Withdrawal from treatment
CO poisoning	234	189	2	2	1	1	39
Sudden hearing loss	37	4	8	0	13	1	11
Delayed wound healing	23	13	0	0	2	0	8
Chronic refractory osteomyelitis	17	5	0	0	4	1	7
Crush injury, compartment syndrome	8	4	1	0	0	1	2
Femoral head necrosis	4	2	1	0	1	0	0
Soft tissue radionecrosis	4	1	0	0	1	1	1
Central retinal artery occlusion	2	0	2	0	0	0	0
n (%)	329	218 (66.3)	14 (4.2)	2 (0.6)	22 (6.7)	5 (1.5)	68 (20.7)

indoors, two patients (0.9%) by smoke inhalation in a fire, two patients (0.9%) by a liquefied petroleum gas (LPG) stove in the kitchen, two patients (0.9%) by hookah smoking, one patient (0.4%) by natural gas and one patient (0.4%) by LPG as a result of suicide attempt. In all cases, poisoning occurred at home. The most common symptom was change in consciousness and transient loss of consciousness (81.2%) followed by headache (63.7%). Nausea and vomiting were present in 44% and balance disturbance in 21.8%. Carboxyhaemoglobin (COHb) levels were measured in 185 patients at presentation and 151 had COHb levels > 20%. However, we did not observe any clinical correlation between COHb level and severity of symptoms. One hundred and eighteen patients were poisoned together with family members (35.9%). The total number of HBOT sessions administered among the 234 CO poisoning patients was 959.

We treated 37 patients (11.3%) with sudden hearing loss. In six the hearing loss was total, and in two cases there was bilateral involvement. We administered an average of 15.6

Table 3
Adverse events by indication in 329 children treated with HBOT

Indication	Ear barotrauma	Anxiety
Carbon monoxide poisoning	1	49
Sudden hearing loss	1	0
Delayed wound healing	0	5
Chronic refractory osteomyelitis	1	3
Crush injury, compartment syndrome	1	1
Soft tissue radionecrosis	1	1
Total	5	59

HBOT sessions to each patient; 18.2% of sessions were administered for this indication.

We treated a total of 23 patients (7.2%) for delayed wound healing (surgical wounds, chronic ulcers of the skin, ulcers on deformed feet due to meningomyelocele and necrotising soft tissue infections). The average number of sessions was 28.3; 20.5% of all sessions were administered for this condition.

Seventeen patients were treated with the diagnosis of chronic refractory osteomyelitis and the average number of sessions was 29.3 (15.8% of all sessions). Nine patients had femur osteomyelitis, seven patients had tibia-fibula osteomyelitis and one patient had humeral osteomyelitis. Eleven developed after traffic accidents. Four developed as a result of other trauma, and two developed as a result of septic arthritis.

We treated eight patients with the diagnosis of crush injury and compartment syndrome (2.4%). We administered a mean number of 20.5 sessions of HBOT (5.2% of all sessions). We applied 5.3% of all sessions to four patients (1.2%) whom we treated for femoral head necrosis. Four patients (1.2%) were treated with the diagnosis of soft tissue radionecrosis. Three of the patients had radiation cystitis and one had radiation myelitis (3.6% of all sessions).

We treated two patients (ages 12 and 14) with unilateral central retinal artery occlusion and administered 20 sessions of HBOT each (1.3% of all sessions).

TREATMENT RESULTS

Treatment results are shown by indication for HBOT in Table 2. We achieved complete recovery in 66.3% of patients (n = 218). As a result of CO intoxication, two patients recovered with major morbidity (neurological sequelae) (0.6%) and 14 patients recovered with minor morbidity (4.3%). In 22 patients (6.7%), there was no improvement.

OUTCOMES AND ADVERSE EVENTS

A total of 64 adverse events were observed in 3,164 treatment sessions (2% of the 3,164 patient treatments) (Table 3). Fifty-nine of these events (1.9% of patient treatments) were anxiety experienced by children in the hyperbaric chamber. There were no patients who refused treatment due to claustrophobia. Only five of the 329 patients (1.5%) had a single episode of ear barotrauma, which is only 0.2% of the 3,164 treatment sessions. There was no resulting morbidity or disability in these patients. An otolaryngologist performed a prophylactic myringotomy before treatment in one patient presenting with a middle ear ventilation disorder. We also did not encounter any central nervous system (CNS) or pulmonary oxygen toxicity in either children or family members accompanying them during treatment. Six patients (1.9%) were treated while still receiving ventilation support in the hyperbaric chamber and were accompanied by an anaesthesiologist. These patients did not develop any complications requiring intervention during the treatments. A total of 241 patients including six intubated patients were transported to our clinics via ambulance. We did not record any complications during patient transport to the HBOT centre.

Discussion

The indications for HBOT have been determined by international organisations, with the indications and levels of evidence updated via consensus meetings.[1,7] All patients we treated presented with indications accepted in the consensus reports published by the UHMS. Since there are no randomised controlled trials of HBOT in any of its accepted indications in paediatric patients, the same indications as for adults are also used for children.[11–13] The only study published in Turkey on HBOT in children is a thesis study in which paediatric patients treated at Istanbul University over 30 years were evaluated.[14]

In the present study, the most common disease treated was CO poisoning (71.1%) which was similar to the 79% of CO poisoning reported in another series of 139 paediatric patients.[12] Carbon monoxide is one of the leading fatal toxins globally typically occurring as a result of incomplete combustion of carbon-containing materials; it is not uncommon in children.[15–17] Complete resolution occurred with a single HBO session in 87 patients and the treatment was terminated. One hundred forty-seven patients required more than one treatment. Two patients who recovered with neurologic sequelae were also intubated before HBOT. The mean number of HBO sessions given was 4.1 and none of our patients presented with late sequelae. Similarly, no late neuropsychiatric sequelae were reported in another series of 111 patients.[12]

Sudden sensorineural hearing loss occurs with a mean loss of 30 dB or more in at least three consecutive frequencies. It

is a rare condition in the paediatric age group and there are very few relevant studies in the literature.[18] In adults, HBOT is recommended to be used in combination with medical treatment.[7,19] We did not find any published cases of sudden hearing loss treated with HBOT in children. However, it was the second most treated indication in the present. This may be explained, at least locally, by the increasing use of HBOT in sudden hearing loss and the awareness of ENT physicians about HBOT.[18,19]

The 23 patients treated for delayed wound healing accounted for 20.5% of the sessions performed (7.2% of the patients). In this group, adherence to treatment was quite high and the rate of patient discontinuation was low. This can be explained by adequate wound care in our clinics. In contrast to other studies,[20,21] necrotising soft tissue infections were very rare in our cohort. We did not have any paediatric patients presenting with necrotising fasciitis or gas gangrene. Relevant presentations were mostly from orthopedic clinics and patients with wound healing problems at the operation site. Three children with necrotising soft tissue infections were given HBOT after surgical intervention for anaerobic crepitant cellulitis and parenteral antibiotics. We treated the patients with two sessions per day at 284 (2.8 atm abs) on the first day and one session per day at 243 kPa (2.4 atm abs) thereafter.

Hyperbaric oxygen reduces tissue hypoxia and necrosis with its anti-hypoxic effect in crush injuries, compartment syndrome and other acute traumatic ischemias;[22] it also reduces tissue oedema with its vasoconstrictive effect. Hyperoxia increases the phagocytosis and bacterial killing ability of leukocytes.[23] In this study, minor amputation was performed in one patient with crush injury. Various levels of amputation were reported in other paediatric patient series.[12,13,20]

Adverse events that occur in HBOT are typically pressure-related events (ear barotrauma, sinus barotrauma), and hyperoxia-related events (visual refractive changes, pulmonary oxygen toxicity, central nervous system toxicity) and claustrophobia. The overall rate of adverse events was quite low in the present series compared with with other paediatric patient series in the literature. For example, one study reported a 5.3% adverse event rate,[13] and another reported a total of 47 adverse events including hypotension, bronchospasm, haemotympanum, and hypoxemia in a series of 32 critically ill patients.[21] The most common adverse event in HBOT is middle ear barotrauma. In adult patients, the incidence of barotrauma is reported at very variable rates in different series.[24–30] One study reported 1.9% ear barotrauma in 11,142 patient sessions,[25] while in another the rate of barotrauma was 3.05% and symptoms occurred mostly in the first three sessions.[27] In a third adult series the overall adverse event rate was 17.4% and the main complication was middle ear barotrauma which occurred in 9.2% of patients and 0.04% of sessions.[31] In this paediatric series, ear barotrauma was detected in 1.5% of patients and 0.15% of sessions. There are several reasons why ear barotrauma seemed rare. First, we examined the patients before accepting them for treatment, and assessed their risk of barotrauma. We also evaluated the patients before each session and interrupted the treatment in cases where there was an elevated risk of barotrauma, such as during inflammatory diseases of the upper respiratory tract. The presence of HBOT staff in the sessions and the fact that children under the age of four were taken into the hyperbaric chamber with their parents in the first session may also have contributed to the low incidence of adverse events in general. In addition, we kept the compression rate in the range of 10 to 14 kPa·min^{-1} because of the higher risk of barotrauma in the first sessions.[24] However, we paid attention to the compression rate because very slow compression can also lead to barotraumas.[30] During the compression phase, HBO staff maintained eye contact with the children, which allowed us to intervene early in ear equalisation difficulties. Another potential reason for the low rate of barotrauma is that we taught patients different methods for ear equalisation before treatment and told children to choose whichever method provided easy equalisation. Children can learn the Valsalva and Frenzel manoeuvres or other Eustachian tube stretching movements such as yawning, swallowing, and chewing gum only at the age of 4–6 years. In younger children, sucking a bottle can open the Eustachian tube with a manoeuvre like the Frenzel. Nevertheless, myringotomy may be necessary before entering the hyperbaric chamber. Distractions during compression (such as safe toys for the hyperbaric chamber or TV) also facilitated ear equalisation. Children under four years of age attending their first session with a family member also helped to reduce complications.

It must also be acknowledged that signs of tympanic barotrauma were not prospectively evaluated using otoscopy among our patients, and had this been done it would almost certainly have resulted in a higher reported incidence of barotrauma.

Sinus barotrauma is the second most common barotrauma encountered in HBOT. The reported incidence in adults ranges from 1.2% to one in 10,000 treatments.[25,28] The incidence of pulmonary barotrauma is extremely low in routine HBO treatment sessions. The risk is reduced by performing chest radiography before treatment and evaluating patients in terms of contraindications and taking necessary precautions. There were no cases of sinus barotrauma or pulmonary barotrauma in our paediatric series.

A potentially serious side effects of high oxygen pressure is CNS oxygen toxicity. Its incidence is given at different rates in the literature (one in 2,000–10,000 patient sessions).[28,32] Pulmonary oxygen toxicity is usually not observed in routine HBO sessions. However, it may occur in a longer treatment such as the US Navy Treatment Table 6 used for

decompression illness.[28] There are no controlled studies suggesting that oxygen toxicity is more frequent in children than in adults. Oxygen toxicity has been reported at low rates in other paediatric patient series.[13,20] One study reported two cases of oxygen toxicity, one pulmonary and one CNS, but the incidence could not be determined because the total number of sessions was not reported.[12]

Hyperoxic myopia is a very common side effect in HBO treatments. The incidence has been reported between 25–100% in different adult patient series.[28,33] Interestingly, it is more pronounced when a hood oxygen delivery system is used compared to an oronasal mask.[34] Vision typically returns to normal 4–6 weeks after the end of treatment. Cataract is not expected in limited duration treatments. However, HBOT has occasionally been associated with faster progression of existing cataracts.[28,33] No ocular adverse events were observed in paediatric patients in the present study.

Claustrophobia is a condition with a rate of 2% in the general patient population and may cause confinement anxiety even in multiplace hyperbaric chambers.[28] Although confinement anxiety has been reported as 8 per 10,000 in different studies, it is actually thought to be higher.[24,33] In one study the rate was reported as 4.3%.[25] In paediatric patients, the rate of anxiety has been reported as 2–3.2%.[13,20] Confinement anxiety was the most common adverse event observed in the present study. Some HBOT centers, particularly in the USA, do not allow family members to go inside the chamber during the treatment. However, it is anxiety-inducing for a child to be in a closed room for two hours breathing oxygen through a mask, which they have never seen in their life. Therefore, we showed the children the hyperbaric chamber before the first treatment and explained what they would experience there. We introduced them to the hyperbaric personnel and allowed patients with anxiety symptoms to enter the hyperbaric chamber with their families. We also asked them to bring their toys that did not pose a fire risk in the hyperbaric chamber. We asked them about their favorite animated movies and cartoons and showed them those movies during the treatment. We allowed babies to suck a bottle in the chamber. Thanks to all these actions, confinement anxiety in children was minimised and adaptation to treatment increased.

Different sizes of laryngoscope sets, masks, and hoods should be available in the clinic. It is important that the HBO personnel who will accompany the patient are especially knowledgeable about critical patient management. Intubated patients should be accompanied by an anaesthesiologist during treatment. In addition, the mechanical ventilator should be set at appropriate values to avoid pulmonary barotrauma in paediatric patients.

It is acknowledged that one important limitation of this study is missing data regarding patients who terminated their treatment plans early.

Conclusions

Hyperbaric oxygen treatment can be life or limb saving in children.

Barotraumas, which are the most common adverse events among adults undergoing HBOT, can be reduced by teaching children different manoeuvres beforehand and through HBOT staff support in the hyperbaric chamber. Anxiety of confinement can be minimised by allowing family members to enter the hyperbaric chamber and allowing children to get to know the hyperbaric chamber and take appropriate toys with them before treatment. According to these observational data, HBOT can be used as a safe treatment with a low risk of adverse events in paediatric patients. Paediatric and intensive care physicians who follow the patient should be informed about the safety of HBOT. Critically ill patients can be safely transported in the presence of anaesthesiologists. In this way, more critically ill paediatric patients with appropriate indications can benefit from this treatment.

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Snorkelling and breath-hold diving fatalities in Australian waters, 2014 to 2018

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Abstract
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Introduction: This study investigated snorkelling and breath-hold diving deaths in Australia from 2014–2018 and compared these to those from 2001–2013 to identify ongoing problems and assess the effectiveness of countermeasures.
Methods: Media reports and the National Coronial Information System were searched to identify snorkelling/breath-hold diving deaths for 2014–2018, inclusive. Data were extracted from witness and police reports, medical histories, and autopsies. An Excel® database was created and a chain of events analysis conducted. Comparisons were made with the earlier report.
Results: Ninety-one fatalities (78 males, 13 females, median age 48 years [range 16–80]) were identified with one third likely doing some breath-hold diving. Fifty-two of 77 with known body mass index were overweight or obese. Approximately two thirds were inexperienced snorkellers and 64 were alone. Fifty-one were tourists. Planning shortcomings, such as solo diving and diving in adverse conditions, as well as pre-existing health conditions and inexperience predisposed to many incidents. Primary drowning was the likely disabling condition in 39% of cases with drowning recorded as the cause of death (COD) in two thirds. Cardiac events were the likely disabling conditions in 31% although recorded as the COD in 21% of cases.
Conclusions: Increasing age, obesity and associated cardiac disease have become increasingly prevalent in snorkelling deaths and there is a need for improved health surveillance and risk management. Closer supervision of inexperienced snorkellers is indicated. Apnoeic hypoxia from extended breath-holding and poor supervision remain a problem. The increased risk of harvesting seafood in areas frequented by large marine predators needs to be appreciated and managed appropriately.

Introduction

Snorkelling involves a person using a mask and snorkel and often fins while swimming to enable them to observe the underwater environment. In addition to swimming on the surface, snorkelling may also involve breath-hold diving underwater. Snorkelling is a popular recreational activity in Australia, but despite some estimates based on relatively small samples, there are no reliable data on the number of participants.[1–3]

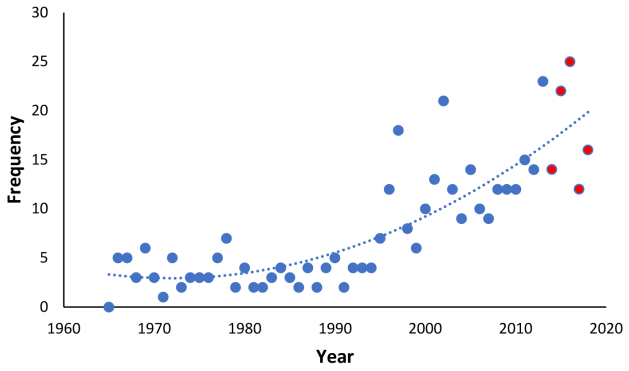
Immersion counters the effect of gravity and encourages redistribution of venous blood into the thorax, so increasing the cardiac workload.[4] It can also be strenuous and anxiety provoking and aspiration of water through the snorkel is common in novices. Breath-holding diving elicits the diving reflex which involves peripheral vasoconstriction, hypertension and bradycardia.[5] The physiological changes, physical challenges, other environmental factors and pre-existing medical conditions in snorkelling participants has an associated incidence of fatalities which has shown a marked increase over time with various peaks and troughs, largely but certainly not solely associated with levels of activity (Figure 1). Earlier Australian epidemiological studies have reviewed snorkelling-related deaths from 1987 to 1996, 1994 to 2006, and 2001 to 2013.[6–8] The last of these highlighted increasing age and prevalence of pre-existing medical conditions of the victims as increasingly common risk factors, in addition to the traditional issues such as inexperience, environmental challenges, planning deficiencies, and unsafe practices.

This current report investigates snorkelling and breath-hold fatalities in Australian waters from 2014 to 2018 inclusive. Causation and outcome data are compared to that of the previous reporting period of 2001–2013 to determine ongoing problems and assess countermeasures.

Methods

Ethics approvals for the collection and reporting of these data was received from the Victorian Justice Human Research Ethics Committee to access the National Coronial Information System (NCIS Approval number CF/21/18434).[9]

Figure 1
Annual snorkelling and breath-hold diving fatalities from 1965 to 2018. The red dots signify the study period



This represents a complete, or near-complete, case series of snorkelling and breath-hold diving deaths in Australia from 1 January 2014 to 31 December 2018. For inclusion, the diver generally must have been reported to have been wearing at least a snorkelling mask. However, an exception to this is a person who partakes in breath-hold diving and who is specifically practicing extending their breath-holding in water to increase their diving limits.

SEARCH

A comprehensive keyword search was made of the NCIS for snorkelling and breath-hold-related deaths throughout Australia for the period 1 January 2014 to 31 December 2018. Keywords included snork*, spear fish*, free diving, underwater fishing. Data obtained from the NCIS was matched with that listed on the Australasian Diving Safety Foundation (ADSF) fatality database[10] obtained via media or word of mouth.

REVIEW PROCEDURE AND OUTCOME MEASURES

The investigator reviewed all datasets, a range of outcome measures were extracted for each case and entered into a specially created anonymised and protected Microsoft® Excel spreadsheet. Where available, these data included demographics, health factors, experience, origin of victims, dive location and conditions, buddy circumstances and oversight, dive purpose and setting, equipment worn and rescue and resuscitation factors.

ANALYSIS

A chain of events analysis (CEA) was performed for each case using existing templates with modifications to better tailor these to snorkelling incidents.[11] Categories were defined as follows:

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

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

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

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

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

Descriptive analyses based on means and standard deviations or medians and ranges, and *t*-tests, $\chi 2$ and Mann-Whitney U tests for comparisons of age or body mass index (BMI), as appropriate, were conducted using SPSS Version 29.0.0.0 (IBM Armonk, NY; 2022). The level of statistical significance assumed was $P < 0.05$.

Results

Between 2014 and 2018 there were 91 identified fatalities in snorkellers and breath-hold divers (hereafter generally combined with snorkellers) throughout Australia. The annual counts were 14, 22, 25, 13 and 16 deaths for the consecutive years from 2014 to 2018, respectively. Based on their reported experience and activity at the time, it is likely that around 55 of the victims were predominantly 'surface snorkelling' and 33 were breath-hold diving. Brief summaries of individual cases can be found at Appendix 1.

DEMOGRAPHICS

There were 78 males and 13 female victims. The median (interquartile range [IQR]) age of the victims was 48 (32, 64) years with a range of 16 to 80 years. There was no significant difference in ages between males and females ($P = 0.65$). The surface snorkellers were older than the breath-hold divers with median (IQR) ages of 55 (33, 70) and 38 (31, 50) years, respectively ($P = 0.003$).

Body mass index was available for 77 of the snorkellers (mean [SD] 28.4 [7.4] $kg.m^{-2}$) and, although higher for the females (30.6 vs 28.1 $kg.m^{-2}$), this was non-significant ($P = 0.70$). Twenty-nine of the victims (25 men and

Footnote: * Appendix 1 is available on DHM Journal's website: https://www.dhmjournal.com/index.php/journals?id=319

Table 1
Location of the incident and origin of the snorkelling victims; NSW – New South Wales; QLD – Queensland; SA – South Australia; VIC – Victoria; WA – Western Australia

State	Deaths	Local	Interstate tourist	Overseas tourist
QLD	48	9	6	33
WA	16	5	3	8
NSW	14	11	1	2
VIC	8	8	0	0
SA	5	3	0	2

four women) were classified as overweight (BMI 25–29.9 $kg.m^{-2}$), and 23 (19 men and four women) were obese (BMI ≥ 30 $kg.m^{-2}$). The highest BMI was 66.4 $kg.m^{-2}$ in a male victim. There was no difference in the BMI for the surface snorkellers and breath-hold divers ($P = 0.71$).

Although higher, the mean (SD) BMI for Australian victims (29.8 [9.2] $kg.m^{-2}$) was not significantly different than for overseas visitors (27.3 [5.3] $kg.m^{-2}$) ($P = 0.06$).

TRAINING AND EXPERIENCE

There was some indication of perceived swimming ability in 53 cases. At least 38 of the victims were reported to have been 'good' swimmers, but at least 16 were reportedly 'weak' or 'non-swimmers'.

Of the 58 incidents where there was some indication of the victim's snorkelling experience, 22 (37%) were reported to have been experienced snorkellers. Seven were certified scuba divers, two being instructors. The remainder were novices, with at least five first-time snorkellers. Breath-hold divers were reportedly more experienced than the surface snorkellers (67% vs 13%).

ORIGIN, LOCATION AND SETTING

Of the 91 snorkel victims, 46 (51%) were Australians or Australian residents, 35 of whom were snorkelling locally and 10 were snorkelling interstate. Forty-one (45%) of the victims were overseas tourists, three overseas workers, and one was a student from overseas. The overseas visitors were from Asia (25), North America (7), the United Kingdom (7), and Europe (6). The locations of the incidents are shown in Table 1.

Sixty-three (69%) of the victims were snorkelling privately, while 28 (31%) were with a commercial operator, all but six of these in Queensland. Thirty-nine of the 48 victims in Queensland (81%) were tourists as were 11 of the 16 (69%) of those in WA. Forty-two (88%) of deaths in Queensland occurred on the Great Barrier Reef (GBR). Surface snorkellers were less likely to dive locally than the breath-hold divers (16% vs 78%).

BUDDY SITUATION AND SUPERVISION

At least 42 (46%) of the snorkellers had set off without a buddy, including 10 who were solo but within a large group under some sort of supervision. Only 26 (29%) of the victims were with a buddy when the incident occurred. Forty-two (47%) were under some supervision, whether from shore, a pontoon, or a boat. Only 11 of the 42 who set out solo were known to have been reasonably experienced snorkellers.

DEPTH OF INCIDENT

At least 70 (77%) of the incidents occurred on the water's surface, similar to the earlier period (71%). Seven occurred underwater, whilst two victims collapsed after boarding the boat or pontoon. The maximum reported water depth for a breath-hold dive was 21.5 metres of seawater (msw).

EQUIPMENT WORN

At least 46 (51%) victims were wearing a mask, snorkel and fins, with 14 of these also wearing a wetsuit, and another 10 wore a stinger suit. At least 17 victims were not wearing fins, and this was unclear in another 19 cases. At least 12 snorkellers were using floatation devices such as a lifejacket, vest, ring or noodles. Sixty-four were not using additional floatation and the remainder were unreported. Only three of the 11 victims who were known to have set off wearing a weight belt had ditched their weights when found. Only one victim was confirmed to have been wearing a full-face snorkel mask but there was an unconfirmed report that another may have been.

ACTIVITY

Fifty-seven snorkellers (63%) were sightseeing, including one each during organised whale shark and sea lion encounters. Twenty-six (29%) died while hunting or gathering seafood, and four died while practising breath-holding – one from a boat with scuba divers nearby, two in pools with others nearby and one was diving solo close to a crowded beach in 4.5 msw depth. One diver died while trying to retrieve a tender in a strong current, and one while doing research.

Seventeen of the victims were relatively new Australian residents, half of Asian origin. All but one of the latter were hunting seafood at the time of their demise, with six looking for abalone in Victoria. Three of the non-Asian Australian residents were known to have been hunting seafood.

RESCUE AND RESUSCITATION

A rescue attempt appears to have been made with 65 (71%) of the victims and all but four of the bodies, or in some cases the remains of the other snorkellers, were later recovered after extended submersion periods of up to one week. In-

Table 2
Predisposing factors associated with 89 snorkelling deaths from 2014 to 2018; some incidents involved multiple predisposing factors and no factors were identified in three deaths; IHD – ischaemic heart disease

61 (68%) – Planning
Solo or poor buddy system
Deep breath-hold diving
Failure to check weather forecast
48 (53%) – Health
Significant medical history
Obesity
Undiagnosed IHD
33 (37%) – Experience/skills
Inexperience
Poor skills
15 (17%) – Equipment-related
Lack of fins and/or buoyancy aids
Wearing weights but no fins
Full-face mask
11 (12%) – Activity
Extended apnoea (some including hyperventilation)
Obviously unsuitable conditions
Spearfishing
10 (11%) – Poor supervision
Lookout failure
Inappropriate ratios
4 (4%) – Organisational
Poor participant skills screening
Poor participant medical screening
Inadequate supervision ratio planning
2 (2%) – Communication
Participant failure to declare medical condition
Failure to inform boat staff of medical condition

water rescue breathing (IWRB) was attempted in at least five incidents, including in-water chest compressions in one.

Cardiopulmonary resuscitation (CPR) was attempted in 72 (79%) cases. It was not performed in other cases due to the delays in body recoveries and the absence or condition of the bodies. Twenty-six of the reports mentioned that blood, froth, water and/or stomach contents were cleared from the airway, two indicated that this was not a factor, and no relevant information was available in 47 cases.

An automated external defibrillator (AED) was available at the site and attached to 39 of the victims, 32 of whom were in Queensland. There was little or no information about the timing of application, but it appears likely that it was less than 10 minutes in only two cases. No shock was advised in at least 20 cases, shocks (1–7) were delivered to 10 victims, and there was no information for the other nine cases.

No relevant information about oxygen (O_2) administration was available for 34 incidents. Oxygen equipment was unavailable in at least 18 cases and O_2 administration was not applicable in 14 cases due to the absence of a body or delays to recovery. Supplemental O_2 was reported to have been available and administered to 25 (28%) of the victims. Nine of these were in a private setting, and 16 in a commercial setting, all but two of these in Queensland. The O_2 delivery method was only reported in three cases, which included two manually-triggered ventilators and one bag-valve-mask.

CHAIN OF EVENTS ANALYSIS

Predisposing factors

One hundred and eighty-five possible or likely predisposing factors were identified in 87 of the incidents as shown in Table 2. There were no PFs identified in three incidents. The most common PF were planning-related, most often a decision by the victim to snorkel without a buddy and/or set off or continue to snorkel in unsuitable conditions.

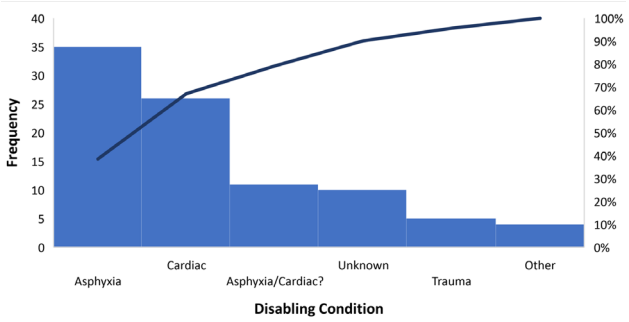
Health-related predisposing factors were also common and generally involved victims with diagnosed or occult moderate to severe ischaemic heart disease (IHD, 27), obesity (22), hypertension (16), diabetes (13), cardiomegaly (17) and left ventricular hypertrophy (LVH, 21), the latter two only identified at autopsy. Five victims had a history of seizures. In five cases, toxicology revealed the presence of drugs at intoxicating and/or supratherapeutic levels.

Inexperience and the consequent lack of skills were identified as a contributory factor in many cases, a common theme in snorkelling incident investigations. Lack of appropriate equipment was identified in 15 (17%) of cases but may well have been a factor in more.

Some activities carried a higher likelihood of an incident. These included five examples of spearfishing in areas frequented by large sharks or crocodiles, and six extended apnoea events with or without hyperventilation. Supervision shortcomings arising from insufficient lookouts and/or lookouts failing to identify persons requiring rescue were identified.

Organisational failures included inadequate screening of participants' medical status and/or skills to enable the implementation of appropriate countermeasures, as well as planning for adequate supervision for the expected number of participants.

Figure 2
Disabling conditions associated with 91 snorkelling deaths from 2014 to 2018



Poor communication predisposed to at least two deaths, one involving the withholding of relevant and required medical information by the victim. In the other, although the victim declared relevant medical conditions to office staff (albeit possibly downplaying their impact), this information was not communicated to the dive supervisor who might have re-considered site suitability and/or countermeasures to reduce the risk of an incident.

Triggers

Seventy-four likely or possible triggers were identified in 49 of the incidents with many incidents involving multiple likely triggers. The most common triggers were environment-related involving adverse sea conditions (17), and the direct effects of immersion often combined with exertion (14). Attacks by sharks (4) or a crocodile (1) led to five deaths. Water aspiration was witnessed in seven incidents but was likely associated with many more. Extended apnoea led to at least six deaths. Diver error was the trigger in one incident in which the breath-hold diver's arm became trapped in a rock crevice while trying to catch a crayfish.

Disabling agents

Likely or possible disabling agents were identified in 76 incidents. The most common disabling agent (in up to 38 incidents) was pre-existing cardiac pathology. Two victims with a history of epilepsy and snorkelling solo were likely disabled by seizures. Environmental disabling agents were apparent in at least 14 cases and were mainly adverse conditions, but also including the shark and crocodile attacks and entrapment. Apnoeic hypoxia was highly likely to have disabled six, and possibly seven victims, buoyancy problems were identified as the likely disabling agent in five incidents but may have been an issue in more cases, and immersion pulmonary oedema (IPO) was a possible disabling agent in up to three cases although the evidence was relatively weak.

Disabling conditions

Asphyxia was the likely disabling condition in 35 (38%) cases although IPO was an alternative for one of these.

Table 3
Comparison of some snorkelling fatality victim characteristics between the 2001–13 and 2014–18 periods; there were no statistically significant differences between the two time periods

Characteristic	2001–13	2014–18
Age (median, years)	51	48
Male sex (%)	90	86
Experienced (%)	50	37
Solo (%)	27	34
Commercial setting (%)	37	31
Tourists (%)	50	60
Disabling condition		
Cardiac (%)	34	31
Asphyxia (%)	41	38
Body mass index classification		
Overweight or obese (%)	66	68
Obese (%)	24	30

Cardiac causes were identified as the likely disabling condition in 28 (31%) incidents although, again, IPO was a possibility in two (Figure 2). In another 11 (12%) of cases it was too difficult to determine whether a cardiac event or asphyxia was the probable disabling condition. The disabling condition could not be determined in ten cases; three as the victims' bodies could not be retrieved. The trauma deaths were associated with shark and crocodile attacks. The remaining disabling conditions were likely seizures (2), drug toxicity (1) and neck dislocation from trauma in one diver.

There was a higher frequency of asphyxia as a disabling condition in breath-hold divers than in surface snorkellers. This occurred with 18/33 (55%) of breath-hold divers and 18/54 (33%) of surface snorkellers (OR = 2.40, 95% CI = 0.99 to 5.84, $P = 0.0735$). There was a higher frequency of cardiac disabling conditions in surface snorkellers, occurring in 26/54 (48%) compared to 2/34 (6%) of breath-hold divers (OR = 14.39, 95% CI = 3.13 to 66.23, $P = 0.0006$).

Cause of death

The reported causes of death were: drowning (60, 67%), cardiac (19, 21%), trauma (4, 4%), olanzapine toxicity (1, 1%) and unascertained (7, 8%).

COMPARISONS WITH PREVIOUS PERIOD

When comparing various characteristics from this current period to those of the period 2001–13 or part thereof, although there were some apparent differences, they were not significant. These included the 33% increase in deaths from the previous five-year period and the 50% lower percentage of experienced victims and higher proportion of tourists during the 2001–13 period (Table 3).

Discussion

The victims of these snorkelling incidents were predominantly middle-aged males, many of whom had pre-existing medical conditions which predisposed them to an incident while snorkelling. Almost one half were overseas tourists, most of whom were inexperienced and unfamiliar with the snorkelling sites. Two thirds of the victims were snorkelling privately, and many were snorkelling without a buddy or supervision which caused delays to rescue. Most were sightseeing, but more than a quarter were hunting or harvesting seafood. Rescue and resuscitation attempts were made in most cases although the commonly associated delays greatly reduced the likelihood of survival.

The finding that half of the victims had pre-existing health conditions that may have contributed to their deaths is consistent with the age and prevalence of obesity of the cohort – predominantly middle-aged males with two thirds being overweight or obese. This is similar to the 67% reported in the general Australian population,[12] whilst epidemiologically there are known links between significant health conditions, especially cardiovascular disease, and both obesity and increasing age.[13,14] The finding in this cohort at autopsy of significant IHD, cardiomegaly and LVH increases the risk of a cardiac event which is much more likely to prove fatal in the aquatic environment.

This prevalence of cardiomegaly and LVH reflects the high level of hypertension in the community, reported to affect one in three Australian adults.[15] Although only 18% of these victims were known to have hypertension, the autopsy findings suggest missed diagnoses and a need for improved health vigilance. Left ventricular hypertrophy is associated with an increased risk of cardiac arrhythmias, and, when combined with moderate to severe IHD, likely explains the high incidence of cardiac-related deaths in both snorkellers and scuba divers.[16] The effects of immersion, exertion, anxiety, aspiration, the diving reflex, cold and increased respiratory resistance, provide a potent mix of precipitants to a cardiac event in a susceptible individual.[4,7]

At least half of the victims in both periods were tourists, predominantly from overseas, who would have been unfamiliar with the location and were often inexperienced snorkellers. Lack of experience and unfamiliarity with the site and conditions (e.g., currents, other tidal effects and local marine life) is a potent mix for increasing risk. The majority of all the victims had little or no experience snorkelling. Inexperienced snorkellers are more likely to be anxious, to aspirate water through their snorkel or mask, and less able to manage the effects of chop, swell and current. They are less familiar with the equipment and more likely to have difficulty with it. In addition, about two-thirds of the victims over both periods were snorkelling in a private setting, often without supervision. This is generally associated with more substantial delays to recognition of a problem and to rescue with a consequent reduced likelihood of survival.

Concern has been raised about the disproportionate incidence of drowning deaths in Victoria of people born overseas.[17] This is reflected in this study by the apparently disproportionate number of overseas-born snorkellers (mainly of Asian origin) who died collecting abalone in Victorian waters, where conditions can often be more challenging than in tropical regions, especially for those less familiar with such sites.

Consistent with the previous period and as reported elsewhere,[18] the majority of victims were alone at the time of their incident, whether they had set off solo or become separated from their buddy or group beforehand. Despite repeated advice about the benefits of an effective buddy system, which may enable more rapid recognition of a problem and subsequent assistance, sadly this is often ignored, and the serious consequences persist. Snorkelling without a designated buddy as part of a large group often provides a false sense of security, even in the presence of supervision.[19]

Some snorkelling activities carry a higher risk. Yet again, this series includes fatalities from apnoeic hypoxia in individuals who were pushing their breath-hold limits. Some were distracted while spearfishing, others were practising extending their breath-hold time. Two of these occurred in pools, with other swimmers and bystanders very nearby albeit distracted, highlighting how easily such an incident can occur. Some of these incidents involved pre-dive hyperventilation, a practice that is still relatively common despite its known risks. Many breath-hold divers still fail to appreciate that such blackouts occur rapidly, generally without warning and, unless a vigilant buddy is present and accessible, drowning is the likely outcome.

In addition to the risk of apnoeic hypoxia, spearfishing (and other seafood collection) can attract the unwanted attention of marine predators. As well as the four fatal shark attacks in this series, there were around 20 documented non-fatal shark attacks of snorkellers during the same period, at least half of these while spearfishing.[20] This is likely an underestimate as all such injuries may not be recorded centrally. Of the 13 known fatal shark attacks on snorkellers in Australia from 1960 to 2018, at least 11 were hunting or harvesting seafood, nine spearfishing.[21] Seafood hunters and harvesters should be familiar with the marine life likely to frequent their potential dive sites and, where possible, keep their catch distant from themselves.

In this series of 91 fatalities, only up to three cases were identified as possibly due to IPO. Diagnosis of IPO in the Australian fatalities is appropriately conservative and relies heavily on witness accounts and the victims' medical and snorkelling history.[22] Dyspnoea with coughing, often with frothy, blood-stained expectoration are used as primary indicators of severe IPO. In the absence of these indications, other potential disabling conditions such as primary drowning, or cardiac arrhythmias with or without secondary

drowning, are prioritised depending on supporting evidence, which can sometimes be rather speculative in the absence of definitive tests.

Hawaii is another popular snorkelling destination with frequent fatalities. From 2009–2018 there were 206 snorkelling deaths in Hawaiian waters, the victims being predominantly males with an average age of 59 years and most of whom were tourists (very similar demographics of the Queensland cohort).[23,19] Many victims had been wearing full-face snorkel masks (FFSMs), raising concerns about the safety of such devices. By comparison, FFSMs are not commonly used in Australia with only one victim confirmed to have been wearing such a mask in this series.

A recent report from Hawaii argues that what it terms rapid onset pulmonary edema (ROPE) is a very common cause of what it describes as 'silent' snorkel drownings.[24,25] The report describes the results of a study which included mainly survivor accounts (37% had worn FFSMs), some autopsy reviews, and basic testing of the breathing characteristics of a wide array of snorkels including FFSMs. It was reported that while all snorkels tested showed some (variable) degrees of resistance to inhalation, only some were increased to serious levels requiring very elevated negative transthoracic pressures believed likely to have been one of the factors precipitating ROPE. (Foti P, personal communication, 2023 February 26). It concluded that: "*rapid onset pulmonary edema-induced hypoxia is a mechanism leading to some, possibly most, fatal and non-fatal snorkel-related drownings*."

However, the above claim that possibly most of these deaths may be due to IPO/ROPE is not supported by the Australian fatality data. The main symptoms reported by the Hawaiian respondents were dyspnoea, fatigue, and limb weakness, which arguably could result from exertion and breath-holding alone, or comparatively mild IPO. There was no specific question about coughing or expectoration, likely indications of a more severe event. In addition, the survey respondents were younger and evenly gender balanced, and may not represent the cohort of deceased snorkellers in Hawaii. It is also interesting to note that some of the serious IPO incidents and deaths are not necessarily 'silent', often showing significant signs of distress.[22]

Concern about FFSMs inspired a Duke University study in which the breathing characteristics and inhaled and exhaled gas composition of an array of FFSMs were tested. It concluded that: "*while this testing yielded no conclusive 'smoking gun' to explain the snorkeller deaths, some of the mask models showed patterns of increased breathing resistance with water intrusion … and this increased resistance could potentially create elevated levels of respiratory distress in snorkellers during real world use*".[26] A potentially relevant example from the present series is case 74 (Appendix 1).

While there is no doubt about its existence, the potential severity, and the very probable underreporting of IPO in snorkellers (as well as scuba divers and swimmers), long-term Australian data does not support an assertion that it is possibly associated with most snorkelling deaths and cardiac factors are often more convincing.

LIMITATIONS

Even using multiple sources, it is possible that some fatalities were not recorded due to limitations in recording and NCIS searches. As with any uncontrolled case series, the collection and analysis of the fatality data are subject to inevitable limitations and uncertainties associated with the investigations. Witness reports varied in their likely reliability. Police reports varied in their content, often related to the expertise of the investigators. Given that many incidents were unwitnessed, some of the assertions in the reports are speculative. Many data items were not available which rendered the study data incomplete, thus limiting the conclusions that can be drawn. The CEA attempts to identify the predominant features of each case, but there always remains an element of uncertainty.

Conclusions

As with the preceding review of Australian snorkelling fatalities, and in common with their scuba counterparts, advancing age, obesity, and cardiac comorbidities were prevalent in this cohort, many of whom were disabled by a cardiac event. Almost forty percent of incidents were identified as likely primary drowning events, although secondary drowning occurred in others with drowning reported as the cause of death in two thirds of cases. Inexperience, unfamiliarity with the site, poor conditions, and the absence of, or an ineffective buddy system, were once again substantial contributors to the incident itself or delays to rescue. These need to be better addressed through improved health surveillance, on-going education, and better pre-dive briefing, screening, and supervision.

Drowning following apnoeic hypoxia persists and the inherent dangers of hyperventilation and pushing breath-hold limits needs to be continually reinforced, as does the need for close buddy scrutiny during extended apnoea, even in a pool. Risks can arise through environmental and human factors and need to be managed by careful planning and execution snorkelling activities.

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Self-reported vitality and health status are higher in Dutch submariners than in the general population

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Abstract
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Introduction: Living aboard submarines has a potential negative effect on health. Although studies have evaluated specific health hazards and short-term outcomes, long-term health effects have not been investigated in this population.
Methods: Veteran submariners were contacted through the veterans' society and administered a World Health Organisation validated questionnaire (SF-36) assessing their physical, emotional, and social functioning. Scores were compared with those of the general (reference) population and scores in veteran submariners were differentiated by rank, time at sea and time in service. Statistical analyses were performed using the Wilcoxon signed rank and Kruskal-Wallis tests.
Results: Of the 1,025 submariners approached in December 2019, 742 (72.4%) completed and returned the questionnaire before July 2020. All 742 were men, of median age 68 (interquartile range [IQR] 59–76) years (range 34–99 years). Of these subjects, 10.3% were current smokers, 64.4% were former smokers and 23.7% had never smoked. Submariners scored significantly better ($P < 0.001$) than the general population on all eight domains of the SF-36. Except for 'pain' and 'change in health status over the last year', scores for all domains decreased with age. Scores were not significantly affected by smoking status, rank, service, and time at sea.
Conclusions: Dutch veteran submariners have better self-reported vitality and health status than the general Dutch population. Rank, service, and time at sea did not significantly affect scores of Dutch submariners.

Introduction

The military value of sub-maritime warfare, in particular the submarine services, cannot be understated. Because of their stealth and espionage capabilities, along with their substantial firepower, submarines are commonly deployed as part of covert maritime operations. Depending on the size and type of submarine, these vessels can stay submerged for days to months, although specifics are rarely published due to the covert nature of submarine warfare.

Living in a confined area for long periods of time can have negative effects on submariners.[1–3] Occupational health specialists must balance the thin line between operational military necessity and the possibilities of preventive medicine.[1,4] Many specific factors have a negative effect on health. For example, recycling the air when submerged reduces air quality, which could contribute to the development of occupational asthma or lung disease. In addition, the restrictions in available space limit the ability to meet the World Health Organization (WHO) criteria for daily exercise, which could contribute to obesity and cardiovascular disease.[5] Moreover, disturbances in circadian rhythm can cause diabetes, cardiovascular disease and cancer.[6] However, surveys in submariners have shown no adverse effects on markers associated with cardiovascular disease while being submerged.[7] Lastly, the submariner community in the Royal Netherlands Navy seem to believe their long-term health is negatively affected due to their service aboard submarines.

Many studies to date have analysed health hazards in submarines of the Royal Netherlands Navy. Due to the covert nature of submarine warfare, however, these reports are rarely published in peer reviewed scientific literature. Additionally, many of these reports have focused on specific health hazards, most often on their short-term effects,[8] making it difficult to determine whether sailing on a submarine affects submariners' quality of life, especially in the long term. Moreover, several publicly available studies have evaluated mortality alone and have not included a more modern perspective on health.[9,10]

A survey assessing the quality of life of veteran submariners may provide greater understanding of the long-term health effects (LTHE) of sailing on a submarine. This study hypothesised that veteran submariners would have a poorer quality of life than members of the general population, with this reduction perhaps due to the perceived health hazards aboard submarines.

Methods

The Medical Ethics Committee affiliated with the Amsterdam University Medical Centre approved the methods used in this study for handling personal details and privacy and concluded that they were in accordance with the guidelines of the Association of Universities in the Netherlands and the Declaration of Helsinki (document reference: W18-337). The study was also approved by the group commander of the Dutch submarine services and the surgeon general of the Netherlands Armed Forces (document reference: DGO120818027).

CONTEXT

The Royal Netherlands Navy has several diesel-powered submarines of the "*Walrus*" class. Submariners are military personnel who are selected and screened for certain qualities, including medical and psychological parameters. These screenings are identical to those of all members of the Netherlands Armed Forces. Individuals who enter the submarine service must undergo additional dive medical screening, similar to military divers and in compliance with ANEP/MNEP-86.[11] Personnel deemed medically fit for the submarine service undergo medical assessments every five years, and yearly after age 40 years. Additionally, submariners scheduled to participate in pressurised submarine escape tower (PSET) training undergo a dive medical examination in accordance with the aforementioned standards. Until 2019, only men were allowed to sail in Dutch submarines; currently both sexes are allowed.

DATA COLLECTION

Health status can be assessed by both qualitative (e.g., interviews) and quantitative (e.g., questionnaires) methods. Although a qualitative approach may yield more detailed results, a quantitative approach was adopted to be able to include as many submariners as possible. General health status can be evaluated using many validated questionnaires. In the present study, health status was evaluated using the Dutch translation of the short form 36 health survey (SF-36) questionnaire.[12–14] The SF-36 assesses many aspects of health status, such as physical, emotional and social functioning, generating a score on eight health domains with multi-item scales (35 items) and an additional single item assessing change in perceived health during the previous year (12 months). Each score varies from 0 (lowest) to 100 (highest), with 50 being the average of the general population of the United States of America and validated in the general population of the Netherlands. Additionally, several baseline characteristics were recorded, including age, rank, and time at sea with the submarine service, as well as smoking status. It was estimated that it would take between 15 to 20 minutes to complete the questionnaire.

Subjects were approached through the Dutch submarine veterans' society, with the opportunity to opt-out of the study. Subsequently, a paper questionnaire was sent to each veteran at his or her home address, and the veteran was asked to complete the form and return it by mail to the Royal Netherlands Navy Diving and Submarine Medical Centre. Participation was voluntary, with no consequences for veterans who did not fill out the questionnaires. To emphasize, only Dutch submariners were included in this study, the reference data are based on the aforementioned studies.[12–14]

DATA ANALYSIS

Returned questionnaires were included in the database if the subject had sailed aboard a Dutch submarine, with no restriction on length of submarine service. Because no data associated with an individual respondent were recorded, any missing data could not be checked. Subjects were divided into three age groups: < 60 years, 60 to 75 years and > 75 years.

All data were entered in a database, allowing calculations of scores on the domains of the SF-36. As the results were not normally distributed, they were compared using the Wilcoxon signed-rank tests and Kruskal-Wallis tests for hypothesis testing. Statistical analyses were performed using SPSS Statistics for Windows software (IBM Corp; Armonk, NY: 2020, version 27.0), with $P < 0.05$ defined as statistically significant.

Results

Questionnaires were sent to 1,025 veteran submariners in December 2019. Eight respondents (0.01%) died during the time between the initial approach through the veterans' society and distribution of the questionnaires. After July 2020, no further questionnaires were received. A total of 742 questionnaires (72.4%) were returned. All respondents were men, of median (IQR) age 68 (59–76) years (range, 34–99). By rank, 50.1% of respondents were enlisted men or corporals, 19.4% were (chief) petty officers and 28.4% were officers. The majority (48.7%) served in the technical services, 32.7% in the operational services, 10.6% in the logistic service and 1.3% in the weapons division. Of these subjects, 10.3% were current smokers, 23.7% had never smoked and 64.4% were former smokers. Evaluation by age showed that 186 respondents were aged < 60 years, 226 were aged > 75 years and 325 were aged 60 to 74 years.

Table 1

Relevant characteristics of the study subjects; due to missing data, not all numbers or percentages add up to 100%; (C)PO – petty officer or chief petty officer; IQR – interquartile range

Parameters		Total	< 60 years	60–75 years	> 75 years
n		742	186	325	226
Age, median (IQR)		68 (59–76)	55 (51–57)	68 (64–72)	80 (78–83.5)
Rank *n* (%)	Enlisted/corporal	372 (50.1%)	95 (51.1%)	176 (54.2%)	100 (44.2%)
	(C)PO	144 (19.4%)	36 (19.4%)	63 (19.4%)	44 (19.5%)
	Officer	221 (28.4%)	50 (26.9%)	86 (26.5%)	75 (33.2%)
	Missing data	15 (2.0%)	5 (2.7%)	0 (0%)	7 (3.1%)
Service *n* (%)	Technical	361 (48.7%)	88 (47.3%)	165 (50.8%)	107 (47.3%)
	Operational	243 (32.7%)	65 (34.9%)	108 (33.2%)	70 (31.0%)
	Logistics	79 (10.6%)	19 (10.2%)	31 (9.5%)	29 (12.8%)
	Weapons	10 (1.3%)	3 (1.6%)	2 (0.6%)	4 (1.8%)
	Missing data	49 (6.6%)	11 (5.9%)	19 (5.8%)	16 (7.1%)
Smoking *n* (%)	Never	176 (23.7%)	65 (34.9%)	70 (21.5%)	40 (17.7%)
	Discontinued	478 (64.4%)	98 (52.7%)	216 (66.5%)	163 (72.1%)
	Yes	77 (10.3%)	21 (11.3%)	38 (11.7%)	18 (8.0%)
	Missing data	11 (1.5%)	2 (1.1%)	1 (0.3%)	5 (2.2%)

Table 2

SF-36 scores of subjects in the three age groups; the domains marked with an asterisk (*) varied significantly among the three age groups on Kruskal-Wallis tests

Domain	Total	< 60 years	60–75 years	> 75 years
1. Physical performance *	92 (80–100)	90 (75–95)	95 (82.25–100)	85 (65–95)
2. Limitation due to physical complaints *	100 (75–100)	100 (75–100)	100 (100–100)	100 (25–100)
3. Limitations due to emotional complaints *	100 (100–100)	100 (100–100)	100 (100–100)	100 (67–100)
4. Vitality *	75 (65–90)	80 (65–90)	80 (65–90)	75 (60–85)
5. Mental health *	92 (80–96)	92 (80–96)	92 (84–96)	88 (74–96)
6. Social function *	100 (75–100)	100 (75–100)	100 (75–100)	88 (75–100)
7. Pain	90 (78–100)	90 (78–100)	90 (78–100)	90 (68–100)
8. General perception of health *	70 (55–80)	70 (55–80)	70 (55–85)	65 (50–75)
9. Change in health status	50 (50–50)	50 (50–50)	50 (50–50)	50 (50–50)

Table 1 illustrates the baseline characteristics of these subjects.

Analysis of the results of the SF-36 questionnaires showed that the scores for the first eight domains were significantly higher than 50 on Wilcoxon signed rank tests ($P < 0.000$) (Table 2), which means the submariners scored better than the reference population. The score for the last domain (change in health status over the previous year) was significantly lower than 50, indicating the submariners experienced less change in health status than the general population, with a *P*-value of 0.030. Additionally, except for domains 7 (pain, $P = 0.251$) and 9 (change in health status during the previous year, $P = 0.074$) scores of all domains varied significantly among age groups on Kruskal-Wallis tests.

Evaluation by rank showed statistically significant differences on domains 2 (limitation due to physical complaints), 6 (social functioning), 7 (pain) and 9 (change in health status) (Table 3). Evaluation by service (or branch as they are known in some navies) showed a statistically significant difference on domain 2 (limitation due to physical complaints), but this effect was not observed when analysing by age groups.

Table 3

SF-36 scores by rank and service / branch; the domains marked with an asterisk (*) varied significantly between ranks on Kruskal–Wallis tests; domains marked with a dagger (†) varied significantly among the different services

Domain	Enlisted (n = 372)	Petty officers (n = 144)	Officers (n = 211)	Technical (n = 361)	Operational (n = 243)	Logistics (n = 79)	Weapons (n = 10)
1. Physical performance	95 (80–100)	90 (75–95)	95 (85–100)	95 (85–100)	95 (80–100)	94 (85–100)	90 (65–100)
2. Limitation due to physical complaints * †	100 (75–100)	100 (50–100)	1000 (100–100)	100 (62.5–100)	100 (75–100)	100 (100–100)	100 (25–100)
3. Limitations due to emotional complaints	100 (100–100)	100 (100–100)	100 (100–100)	100 (100–100)	100 (100–100)	100 (100–100)	100 (100–100)
4. Vitality	75 (65–85)	75 (60–90)	80 (70–90)	85 (75–91.25)	75 (65–85)	75 (65–90)	75 (60–85)
5. Mental health	92 (80–96)	92 (80–96)	92 (84–96)	90 (83–92)	92 (82–96)	92 (84–96)	92 (72–96)
6. Social function *	100 (75–100)	100 (75–100)	100 (88–100)	100 (100–100)	100 (75–100)	100 (75–100)	100 (63–100)
7. Pain *	90 (78–100)	90 (68–100)	100 (78–100)	100 (84.5–100)	90 (69–100)	90 (78–100)	90 (68–100)
8. General perception of health	70 (55–80)	67.5 (55–80)	70 (60–85)	65 (60–77.5)	70 (55–85)	70 (60–80)	65 (50–80)
9. Change in health status *	50 (50–50)	50 (50–50)	50 (50–50)	50 (50–56.25)	50 (50–50)	50 (50–50)	50 (50–50)

Time at sea was longer for enlisted men (median, 96 months; IQR, 56–142 months) than for petty officers (median, 79.5 months; IQR, 48–121.5 months) and officers (median, 42 months; IQR, 26.3–60 months). Evaluation of the relationship between rank/time at sea and scores on the domains of the SF-36 showed that, except for domains 8 (general perception of health), and 9 (change in health status), higher SF-36 scores were significantly associated with lower rank/longer time at sea (= < 0.05 by Wilcoxon signed rank tests). For officers, time at sea was significantly associated with scores on all nine domains of the SF-36; for petty officers, time at sea was significantly associated with scores on domains 1, 4, 8, and 9; and for the enlisted men, time at sea was associated with scores on all domains except for domains 3 and 6. SF-36 scores did not differ significantly among the services.

Smoking did not have a statistically significant effect on SF-36 scores, as determined by Wilcoxon signed-rank tests, with *P*-values between 0.166 (physical performance) and 0.914 (change in health status). These findings were observed in all age groups, ranks, services, and times at sea, as determined using Kruskal-Wallis tests.

Discussion

To our knowledge, this is the first study to report the long-term health status of submariners. These individuals scored significantly higher than the general population on all eight domains of the WHO quality of life questionnaire, independent of age, rank, and time at sea. These findings suggest that sailing on a submarine might not have significant negative effects on submariners' health.

These results may be due to a 'healthy worker effect', in that submariners, and military personnel in general, may be healthier than members of the general population due to the higher fitness requirements of the former.[15] Additionally, having medicals once every five years up until 40 and then yearly thereafter may also have a positive impact on the long-term health effects although we cannot predict to what extent this might be true. Individuals who can no longer meet the military requirements, due for example to illness or an accident, are discharged from the armed forces. Although these results are highly applicable to submariners, health hazards that cause a substantial decrease in function may have been masked in this study, i.e., individual cases where exposure could have led to disease, such as asthma or lung cancer after polluted air exposure, have little effect on the group as a whole. However, this is unlikely as the population was not selected based on time at sea.

The SF-36 questionnaire is a self-reported assessment of vitality and health. Although this introduces bias, quality of life may not be analogous to objective markers.[9,16] Individuals usually compare their quality of life with that of peers in the same age group.[17] Although objective measures show that elderly subjects have more limitations due to

ailments than younger populations, their perceived quality of life may not differ. The advantage of the method utilised in this study was that, in the absence of chronic disease, the perceived quality of life remains generally unchanged with increasing age. Therefore, this method effectively screens for disease and 'normal ageing', without complex data permutations.

Interestingly, scores among submariners were well above average long after these subjects left military service compared to the general population (Table 2). Because a substantial number of respondents were aged > 75 years, it was reasonable to assume that some health hazards, as mentioned earlier the development of occupational asthma, lung disease, obesity, cardiovascular disease, diabetes, or cancer from sailing on a submarine may show a delayed onset, equivalent to mesothelioma resulting from exposure to asbestos.[17] Although rank is often regarded as a surrogate marker for socioeconomic status, the perceived good health among submariners was minimally affected by access to healthcare or other facilities.[18]

The present study also evaluated the smoking status of respondents. In the Netherlands, smoking has the greatest negative effect on health, with 19.4% of the total population being cigarette smokers.[19] Almost two-thirds of the submariners surveyed had previously smoked, with an average time from quitting smoking until survey completion being 24.6 years. These individuals can perhaps be regarded as non-smokers, with only 11% of respondents being current cigarette smokers. This difference in cigarette smoking rates between submariners and the general population may further explain the higher score of the former on the study questionnaires. In addition, smoking has been prohibited aboard Dutch submarines since 2004, which may have encouraged submariners to quit smoking.

STRENGTHS AND LIMITATIONS

Submitting these data to peer review and releasing them for publication may encourage an academic discussion on occupational medicine associated with submarines. Increases in health and safety standards aboard submarines, along with increases in their strategic deployment, may help in setting naval health policy for this specific niche population.

The present study had several limitations. First, the study population consisted of male veterans of the submarine service who were affiliated with the veterans' society. Therefore, extrapolation of these results to other populations should be done carefully. Additionally, members of the submariner community are highly active, with many submariners showing lifelong engagement in the veterans' society. This is illustrated by the response rate of the study population to the questionnaire, which was > 70%. Some veteran submariners, however, may not have received the questionnaire, but there is no indication that inclusion of these subjects would have affected the results of this study. Second, although the response rate was substantially higher than might be expected from questionnaires, data were unavailable for non-responders and for submariners who died. Therefore, mortality associated with working on a submarine would have been undetected, which may have affected the study results. Finally, the control group in this study consisted of the general population, not the veteran military population. Although no data are currently available for the latter group, similar studies are planned with veterans of the naval fleet and marine corps.

Conclusions

Publication of data evaluating the long-term health status of (veteran) submariners may contribute to the scientific advancement of the submarine community. The Dutch veteran submariner community self-reported vitality and health status were significantly higher than those of the general population. Neither rank, a surrogate marker for socioeconomic status, nor service significantly affected these results. The positive relationship between time at sea and perceived health and the evaluation of a population that includes individuals up to a high age indicate that a submarine is a safe working environment, although modern technologies may have introduced new health hazards that were not considered in this survey. Future research could focus on the self-reported vitality and health status of naval fleet personnel or marines to further substantiate the safety of a submarine as a working environment. Similar studies in navies of other countries may validate these findings.

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Outcomes of hyperbaric oxygen treatment for central and branch retinal artery occlusion at a major Australian referral hospital

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Blindness; Circulation; Ophthalmology; Retinal artery occlusion; Vision

Abstract
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Introduction: This study analysed the treatment outcomes of patients that received hyperbaric oxygen treatment (HBOT) for retinal artery occlusion (RAO) at the Royal Brisbane and Women's Hospital in Brisbane, Australia between 2015 and 2021.
Methods: Retrospective study from patient records including 22 eyes from 22 patients that received HBOT for either central RAO (17 patients) or branch RAO (five patients). Patients received the Royal Brisbane and Women's Hospital RAO protocol for their HBOT. Analysis included best corrected visual acuity pre- and post-treatment, subjective improvements, side effects and patient risk factors were also recorded.
Results: Improvement in best corrected visual acuity was LogMAR -0.2 for central RAO on average with 8/17 (47%) experiencing objective improvement, 5/17 (29%) experienced no change and 4/22 (24%) experienced a reduction in best corrected visual acuity. Subjective improvement (colour perception or visual fields) was reported in an additional 4/17 patients, resulting in 12/17 (71%) reporting improvement either in visual acuity or subjectively. There was no improvement in the best corrected visual acuity of any of the five patients suffering from branch RAO. Cardiovascular risk factors present in the cohort included hypertension, hypercholesterolaemia, previous cardiovascular events, cardiac disease and smoking. Limited side effects were experienced by this patient cohort with no recorded irreversible side effects.
Conclusions: Hyperbaric oxygen treatment appears a safe, beneficial treatment for central RAO. No benefit was demonstrated in branch RAO although numbers were small. Increased awareness of HBOT for RAO resulting in streamlined referrals and transfers and greater uptake of this intervention may further improve patient outcomes.

Introduction

The retina has an increased sensitivity to hypoxic states due to its high oxygen demand.[1,2] Central retinal artery occlusion (CRAO) or branch retinal artery occlusion (BRAO) can lead to profound and irreversible visual loss by hypoxic injury to the inner retina – usually within 4–6 hours.[3] In the acute phase, there are currently limited treatment options in both trying to resolve the occlusion and/or minimise the degree or duration of retinal hypoxia/ischaemia.

Hyperbaric oxygen treatment (HBOT) works by inhalation of 100% oxygen at pressures greater than atmospheric, which markedly increases dissolved oxygen tension in plasma. This mechanism is used to attempt to increase the oxygen delivered to the inner retina via the choroidal circulation while the central/branch retinal artery is compromised. The treatment may be required multiple times for an extended period, until the retinal artery or branch recanalises which is typically in the first 72 hours.[4]

This study reports on the outcomes of patients with acute CRAO and BRAO receiving HBOT at The Royal Brisbane and Women's Hospital (RBWH), a large tertiary referral centre in Queensland, Australia.

Methods

Ethics approval exemption was granted by the Human Research Ethics Committee of the Royal Brisbane and Women's Hospital. (Reference. EX/2022/QRBW/84263). The study was performed in line with the principles of the Declaration of Helsinki. Informed consent was obtained from all individual participants included in the study.

Figure 1

Pathway in the Royal Brisbane and Womens Hospital (RBWH) central retinal artery occlusion (CRAO) hyperbaric oxygen treatment (HBOT) protocol; BCVA – best corrected visual acuity; FiO_2 – fraction of inspired oxygen; VA – visual acuity

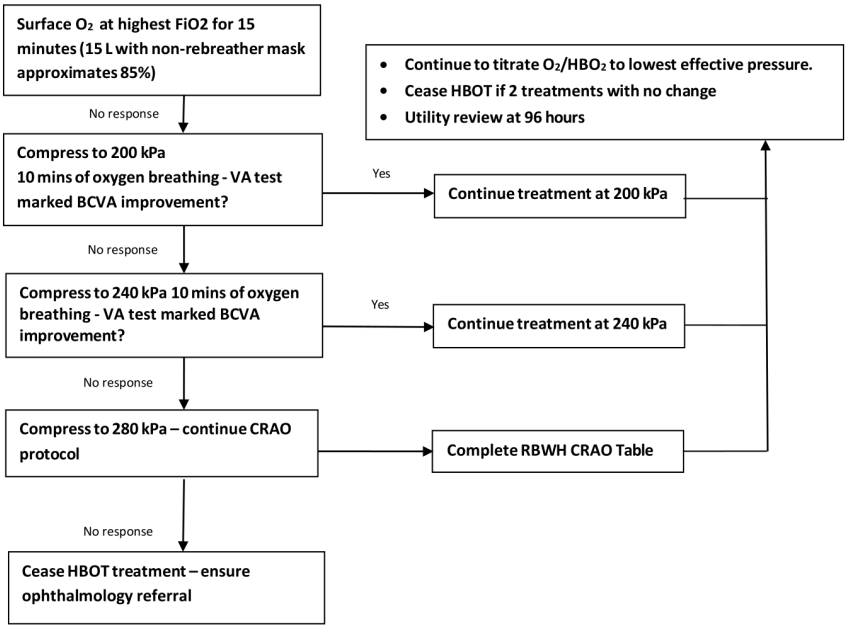
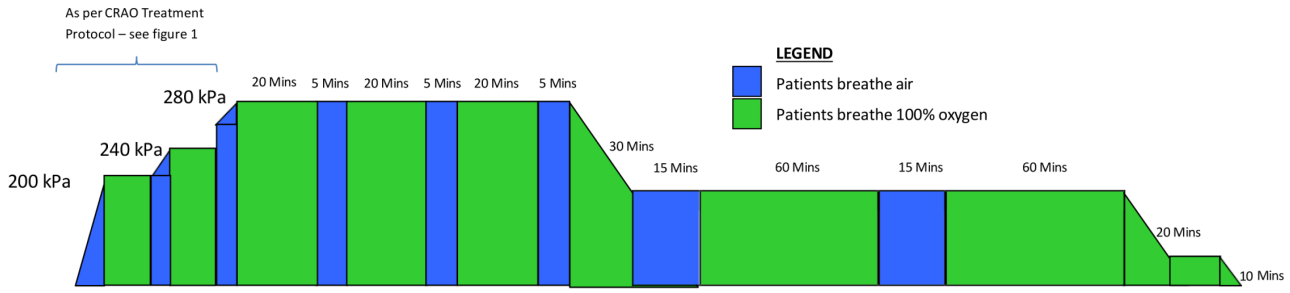


Figure 2

Hyperbaric oxygen treatment protocol for central retinal artery occlusion (CRAO) (see also Figure 1) designed to titrate the minimum treatment pressure to attain a 'marked improvement' in the patient's visual acuity



CLINICAL DATA COLLECTION

All patients presenting to the Royal Brisbane and Women's Hospital with CRAO and BRAO that received HBOT between 2015 and 2021 were included. Data points collected were patient risk factors (including age, sex, hypertension, smoking history, hyperlipidaemia, atrial fibrillation, and diabetes), best corrected visual acuity (BCVA) prior to treatment and after treatment, subjective improvement in visual acuity as reported by the patient, time to intervention from beginning of symptoms, maximum compression pressure, number of HBOT treatments and side effects of treatment. Visual acuity measured with Snellen charts was converted to the logarithm of the minimum angle of resolution (logMAR) using standard conversion charts for statistical analysis. Change in logMAR visual outcome was calculated by subtracting the initial logMAR BCVA from the final logMAR BCVA. Hyperbaric oxygen treatment was utilised as monotherapy in all included patients during the acute phase of their management.

HYPERBARIC OXYGEN TREATMENT AND MANAGEMENT

Ophthalmology registrars and consultants examine patients with suspected CRAO/BRAO in the emergency department and hyperbaric medicine consultants assess and instigate HBOT after the diagnosis is confirmed. Once confirmed, patients will then receive their first round of HBOT while still an emergency department patient where possible.

The HBOT given utilises the 'RBWH CRAO protocol' (Figure 1, pathway in the RBWH CRAO protocol and Figure 2, hyperbaric oxygen treatment protocol). This protocol is adapted from the Undersea and Hyperbaric Medical Society (UHMS) HBO_2 indications book

(14th edition).[5,6] As the aim of the therapy is to protect the retina from hypoxic injury while the retinal artery occlusion recannalises, the protocol utilises HBOT at the lowest pressure at which the patient has a marked improvement in their BCVA. Patients are maintained at lower pressures if marked improvement in BCVA is made prior to getting to the maximum 280 kPa absolute compression pressure specified in the RBWH CRAO protocol. The flow chart in Figure 1 demonstrates the HBOT pathway available to the hyperbaric medical team.

After initial HBOT treatment, patients are then admitted under a medical team with hourly checks of their BCVA. Whilst patients are on the medical ward, they receive 15 minutes of oxygen at15 $L \cdot min^{-1}$ via a nonrebreather mask every hour and breathe room air for 45 minutes in the hour. If any loss of BCVA is detected during the hourly observations or detected after any changes noted by the patient, further HBOT is considered immediately. In the absence of deterioration, the same protocol is then followed for further HBOT treatment the following day. Once no further improvement is realised, HBOT is ceased, the total number of cycles that were used was then recorded. The medical team also assesses the patients for cerebrovascular risk factors, investigates and where appropriate commences secondary prevention of cerebrovascular disease.

STATISTICAL ANALYSIS

Summary statistics were presented as mean and standard deviation for continuous variables and as number and percentage for categorical variables. Multiple linear regression was used to investigate the effect, on the dependent variable of change in logMAR BCVA, of the independent variables of age, delay to initiation of HBOT, number of HBOT cycles and maximum compression pressure reached. Stata IC version 16.1 for Mac was used for regression analysis and to generate figures.

Results

Twenty-two eyes of 22 patients were included in the study. Patients comprised 15 males and seven females with a mean age of 64 years. Seventeen of the 22 patients had CRAO and five of the 22 patients had BRAO. Baseline characteristics are summarised in Table 1.

CRAO PATIENTS

Pre-HBOT BCVA of CRAO patients ranged from perception of light only to 6/60 (LogMAR 2.7 – LogMAR 1.0). After treatment there was an average improvement of LogMAR -0.2 ranging from a final BCVA of no perception of light to 6/12 (LogMar 3.0 – LogMAR 0.3).

Of the 17 patients with CRAO, 8/17 (47%) had an objective improvement in their BCVA, 5/17 (29%) had no change and 4/17 (24%) had a reduction in their BCVA. Four of the five patients that did not have an objective improvement in their BCVA, did report subjective improvement in their vision such as brighter colours, or subjective reduction in visual field defect.

Table 1
Baseline and hyperbaric oxygen treatment (HBOT) characteristics of central (CRAO) and branched retinal artery occlusion (BRAO) patients; SD – standard deviation

Retinal artery occlusion type	CRAO (*n* = 17)	BRAO (*n* = 5)
Age, years mean (SD)	67.4 (14.6)	51.0 (21.9)
Sex, *n* (%)		
Male	11 (65)	4 (80)
Female	6 (35)	1 (20)
Risk factors, *n* (%)		
Hypertension	11 (65)	3 (60)
Hyperlipidaemia	8 (47)	1 (20)
Former smoker	5 (29)	1 (20)
Current smoker	3 (18)	0 (0)
Atrial fibrillation	1 (6)	1 (20)
Diabetes	1 (6)	0 (0)
HBOT factors, mean (SD)		
Delay to HBOT, hours	12.4 (4.9)	15.5 (6.4)
Number of cycles	6.5 (5.1)	4.0 (3.7)

Mean delay to treatment time in CRAO patients was 12 hours, range 3–24 hours. Maximum compression pressures were 280 kPa for 16 out of 17 patients and 1 patient received treatment to a maximum of 240 kPa. Patients received on average 6.5 compression cycles, range of 1–18 (Table 1).

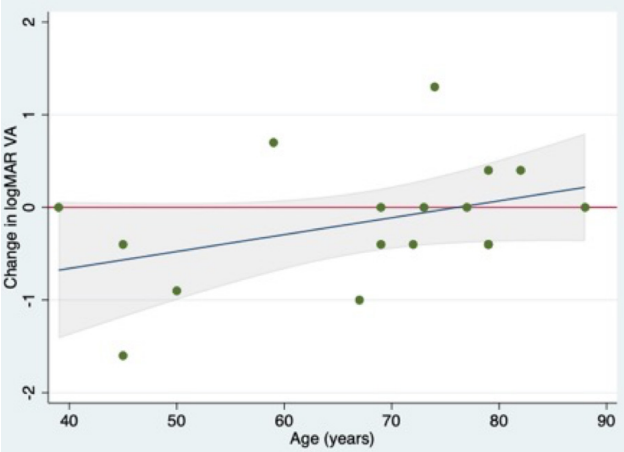
For CRAO patients, multiple linear regression analysis failed to predict the dependent variable of change in logMAR visual acuity with the independent variables of age, delay to HBOT or number of HBOT cycles, $F(3,12) = 1.46$, $P = 0.28$, adjusted $R^2 = 0.08$. Table 2 breaks down the results of regression analysis for each variable. Maximum HBOT pressure could not be used in the linear regression analysis due to collinearity as all values for CRAO patients, except one, were 280 kPa.

Given the low sample size ($n = 17$) and the relatively low P-value for age ($P = 0.08$), graphical analysis and simple linear regression were performed with only age as the independent variable and change in logMAR VA as the dependent variable to investigate this relationship in more detail. Figure 3 shows the results of this regression analysis. This model, again, did not statistically significantly predict the change in logMAR visual acuity, $F(1,15) = 2.79$, $P = 0.12$, adjusted $R^2 = 0.10$, with a slightly smaller effect

Table 2
Multiple linear regression for change in logarithm of the minimum angle of resolution (logMAR) visual acuity for central retinal artery occlusion patients; SE – standard error

Variable	Coefficient	SE	*t*	*P*-value	95% confidence interval
Age	0.0207	0.0110	1.88	0.08	-0.0033 to 0.0447
Delay	-0.0343	0.0387	-0.88	0.39	-0.1187 to 0.0502
Number of cycles	-0.0444	0.0371	-1.20	0.25	-0.1252 to 0.03636
Intercept	-0.8011	0.8854	-0.90	0.38	-2.7302 to 1.1281

Figure 3
Dependent variable change in logarithm of the minimum angle of resolution (logMAR) visual acuity by age for patients with CRAO



size for age (coefficient = 0.0182, SE = 0.0109, t = 1.67, P = 0.12).

Simple linear regression analysis was performed and is illustrated in Figure 3: dependent variable change in logMAR VA by age for patients with CRAO. The blue line represents the multiple least squares regression line. Grey shading represents the 95% confidence interval for the regression line. The red line is at y = 0, representing no change in VA.

Recorded side effects in the CRAO patients were limited to haemotympanum (three patients) and anxiety (two patients).

BRAO PATIENTS

Pre-HBOT BCVA ranged from no perception of light to 6/5 (with central scotoma) (LogMar 3.0 – LogMar -0.1). After treatment the post-HBOT BCVA ranged from no perception of light to 6/5 (with central scotoma) (LogMar 3.0 – LogMar -0.1). One patient described a decreased scotoma subjectively. However, of the five BRAO patients, none had any change in their BCVA.

Mean delay to treatment time in BRAO patients was 16 hours. Maximum compression pressures were 280 kPa for four patients and 240 kPa for one patient. Patients received on average four compression cycles, with a range of 1–10 (Table 1).

For BRAO patients, multiple linear regression could not be used as all patients had no change in logMAR BCVA, making all variables multicollinear. Side effects in the BRAO patients were limited to one of the five patients who needed frequent stops for anxiety associated with the apparatus.

A patient summary including occlusion type (branch or central), presenting BCVA, intraocular pressure, and BCVA post treatment is presented in Table 3.

Discussion

This study reviews the experience at the RBWH with hyperbaric oxygen therapy for the treatment of CRAO and BRAO. The use of HBOT for retinal artery occlusion is controversial among ophthalmologists as its potential benefit is variable and this is shown in our results where any visual acuity recovery was variable.

As previously reported, the rate of objective improvement in BCVA in CRAO patients receiving HBOT varies widely. Previous studies have shown objective improvement in BCVA in 29–59% of patients, with a mean logMAR improvement ranging from -0.05 to -0.53.[7–9] Conversely, a recent meta-analysis concluded that there was no significant change in BCVA post HBOT.[10] The authors did however suggest that subgroups of patients receiving early HBOT did show improvement in several of the studies.[10]

One major contributor to this variability may be the average time to initial HBOT treatment from onset of symptoms. In previous reports with early HBOT initiation this delay has ranged from 5.3 to 8.4 hours. Although other studies have shown a potential benefit of early HBOT,[11,12] only four of the 17 CRAO patients in our study received HBOT within eight hours of the onset of symptoms and of these patients only one showed improvement in BCVA. Our analysis confirmed that

Table 3
Presenting best corrected visual acuity (BCVA), intraocular pressure (IOP) (measured with either iCare IC100 or iCare IC200 handheld tonometer) and final BCVA; CF –count fingers; F – female; HM – hand movements; M – male; NPL – no perception of light; PL – perception of light

Patient ID	Age	Sex	*n* HBOT	CRAO/ BRAO	Initial IOP (mmHg)	Initial BCVA	Final VA	Subjective improvement noted (if applicable)
1353	74	M	8	CRAO	22	6/60	HM	Visual field
1719	79	M	3	CRAO	23	HM	CF	Visual field
1757	67		12	CRAO	11	CF	6/48	Visual field
1818	79	F	18	CRAO	21	CF	HM	Visual field
1859	77	F	3	CRAO	13	PL	PL	Colour perception, brighter light
1871	73	M	3	CRAO	12	HM	HM	Visual field, luminance
1946	79	F	8	CRAO	14	PL	HM	Visual field, brightness
1991	51	F	5	BRAO	14	6/6, inferior quadrantanopia	6/6, inferior quadrantanopia	
2153	18	M	1	BRAO	14	6/5, central scotoma	6/5, central scotoma	
2171	45	M	16	CRAO	14	CF	6/12	Visual field
2230	50	F	3	CRAO	13	CF	6/60	Luminance
2247	57	M	3	BRAO	20	6/7.5, scotoma	6/7.5, scotoma	Visual field
2251	69	M	1	CRAO	10	HM	HM	
2260	50	M	1	BRAO	23	6/6, temporal quadrantanopia	6/6, temporal quadrantanopia	
2280	72	M	11	CRAO	16	HM	CF	
2333	82	M	8	CRAO	12	HM	PL	
2365	69	F	8	CRAO	16	PL	HM	Visual field
2397	45	M	6	CRAO	18	HM	CF	
2414	39	F	1	CRAO	12	HM	HM	
2441	88	M	2	CRAO	13	HM	HM	
2457	59	M	1	CRAO	9	HM	NPL	
2573	79	M	10	BRAO	10	NPL	NPL	

there was no statistically significant relationship between shorter delay to initial HBOT and improvement in logMAR BCVA in our cohort.

Among all our CRAO patients, the average time to initial HBOT treatment was longer at 13 hours with a range of 3–24 hours. This can be partly explained by the large geographical catchment area of the RBWH and the increased time it can take patients to present to hospital. Despite this delay, in patients with CRAO who received HBOT, 47% of patients had an objective improvement in their BCVA and 24% reported subjective improvement including decrease in visual field defects, brighter perception of light or greater perception of colours. In terms of trying to identify those patients who may improve, unfortunately our CRAO results also could not identify a factor that was a significant predictor of BCVA improvement after HBOT e.g., age or pre-HBOT BCVA. A post-hoc power analysis showed that the regression analysis investigating the relationship between age and change in logMAR BCVA was underpowered at around 0.09, therefore, despite a visible graphical relationship and close *P*-value for the relationship between age and outcome, this analysis could not detect a correlation beyond chance. We suspect this may not be the case with a larger sample.

Although other studies have shown significant BCVA improvement in 75% of patients post-HBOT,[2] our patients did not show any change in BCVA after treatment albeit in a small five patient group.

In terms of side effects of HBOT, these were limited to five patients in the cohort, three experienced haemotympanum and another two patients had several breaks during treatment due to anxiety. The rare side effects discussed in the literature such as severe barotrauma or generalised seizures were not seen.[10]

Future studies may benefit from utilising a protocolised diagnostic workup for patients presenting with CRAO. Consistent recording of the presence or absence of a cherry red spot which has been shown to be an important prognosticator and may be used to guide therapy is warranted.[7] Fundus fluorescein angiography also provides important diagnostic and prognostic information, for CRAO allowing further classification into subtypes including non-arteritic CRAO (NA-CRAO), NA-CRAO cilioretinal artery sparing, transient NA-CRAO and Arteritic CRAO as described by others.[13] Visual field testing at time of diagnosis and after treatment would also allow the clinician to separate true improvement in BCVA from eccentric fixation that may confound changes in BCVA after treatment.[13]

Conclusions

In summary, objective logMAR BCVA improvement was seen in 47% of CRAO patients but no improvement was seen in any BRAO patient in our cohort. No patient factor was identified which might predict an improvement in BCVA with HBOT. Although our cohort had minimal side effects from HBOT, it is clear from the current limited evidence that larger randomised studies are required to better understand the efficacy and safety of HBOT in treatment of RAO.

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Contemporary practices of blood glucose management in diabetic patients: a survey of hyperbaric medicine units in Australia and New Zealand

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Keywords
Blood sugar level; Diabetes; Hyperbaric oxygen treatment; Protocol; Questionnaire

Abstract
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Introduction: Blood glucose levels may be influenced by hyperbaric oxygen treatment (HBOT). Patients with diabetes mellitus commonly receive HBOT but there is a lack of standardised blood glucose management guidelines. We documented relevant contemporary practices applied for patients with diabetes treated in hyperbaric medicine units.
Methods: A survey was administered in 2022 to the directors of all 13 accredited hyperbaric units in Australia and New Zealand to identify policies and practices related to management of patients with diabetes receiving HBOT.
Results: Twelve of the 13 units routinely managed patients with diabetes. Three-quarters (9/12) used < 4 mmol·l^{-1} as their definition of hypoglycaemia, whereas the other three used < 5, < 3.6, and < 3 mmol·l^{-1}. Units reported 26% (range 13–66%) of their patients have a diagnosis of diabetes of which 93% are type 2. Ten (83%) units reported specific written protocols for managing blood glucose. Protocols were more likely to be followed by nursing (73%) than medical staff (45%). Ten (83%) units routinely tested blood glucose levels on all patients with diabetes. Preferred pre-treatment values for treatments in both multiplace and monoplace chambers ranged from ≥ 4 to ≥ 8 mmol·l^{-1}. Seven (58%) units reported continuation of routine testing throughout a treatment course with five (42%) units having criteria-based rules for discontinuing testing for stable patients over multiple treatments. Two-thirds of units were satisfied with their current policy.
Conclusions: This survey highlights the burden of diabetes on patients treated with HBOT and identifies considerable variability in practices which may benefit from further study to optimise management of these patients.

Introduction

Approximately 5.3% of Australians and 5.7% of New Zealanders have diabetes (type 1 and type 2) making it common in the general population.[1,2] In addition, non-healing diabetic ulcers are one of the approved indications for treatment with hyperbaric oxygen.[3] As such, diabetes is a frequent co-morbidity in hyperbaric medicine patients.

Hyperbaric oxygen treatment (HBOT) has been shown to affect blood glucose levels in patients with diabetes by a postulated mechanism of increasing peripheral insulin sensitivity.[4,5] Its specific effect on individual patients, however, has been inconsistent among studies. Within eight papers examining blood glucose fluctuations with HBOT, five show an overall decline in blood glucose levels,[6–10] one shows an increase[11] and two suggest no change.[12,13] As well, individual changes may vary markedly with one study showing a range from +13.3 mmol·l^{-1} to -20.0 mmol·l^{-1} for blood glucose responses during a single treatment.[13] Although the type of diabetes, insulin usage, and control of blood glucose prior to treatment have been investigated, no variable has demonstrated a consistent ability to predict an individuals' blood glucose response.[6,7,11,13]

Safety for patients and staff in a closed, pressurised hyperbaric chamber is paramount. Symptomatic hypoglycaemia, particularly hypoglycaemia-associated seizures, represent a major safety concern during HBOT. Event rates of symptomatic hypoglycaemia have been reported from 0.19% to 4.6% of treatments depending on patient population studied and definitions of hypoglycaemia applied.[6,11] Seizures due to hypoglycaemia are extremely infrequent during HBOT[14] but represent a medical emergency that is

challenging to manage particularly in monoplace chambers. To prevent these complications, different suggestions have been proposed for testing and targeting specific pre-HBOT glucose levels. Based on a limited body of evidence, recommendations have consistently advised relatively elevated pre-treatment levels[6,8,10,13,15,16] leading to changes in both patient and physician management of diabetes during HBOT.[17]

Although diabetes is among the most common comorbid illnesses observed among patients treated with HBOT and this treatment may result in adverse effects on their glucose management, there is a paucity of information surrounding actual practices. The objective of this study was therefore to conduct a survey of accredited hyperbaric units in Australia and New Zealand to describe contemporary practices of glucose management among patients with diabetes undergoing HBOT.

Methods

This project was submitted to the Royal Brisbane and Women's Hospital Human Research Ethics Committee and was found to be exempt from full ethics review as it was considered negligible risk research (Ref: EX/2022/QRBW/83562).

STUDY DESIGN

The survey utilised a mixed semi-quantitative, semi-qualitative design. A pilot survey was created with questions based on practice principles utilised in the hyperbaric unit at Royal Brisbane and Women's Hospital. It was tested on clinicians at two hyperbaric units within Brisbane, Queensland for the relevance of questions and ease of administration and modified iteratively to form the final version. Respondents were asked to complete the survey within the context of a typical month of treatments. Questions were grouped into themes: determining the proportion of patients with diabetes, definitions of hypoglycaemia, presence of written protocols, and practices surrounding monitoring and management of glucose before, during, and after HBOT.

PARTICIPANTS

The survey was offered to the medical directors of each of the 13 accredited hyperbaric units within Australia and New Zealand. Consent to participate was demonstrated by participation. All survey responses were kept confidential. Respondents were offered the option to be contacted for further detailed discussion of their responses.

DATA ANALYSIS

Data obtained within the survey were collated with unit and director identifiers anonymised. Analysis was descriptive. Categorical values were reported as proportions (%). continuous variables were reported as means with standard deviations or medians with ranges. The post-survey interview responses were grouped into themes.

Results

All 13 eligible units responded to the survey. One unit did not routinely treat patients with diabetes and did not participate further, leaving 12 units in the analysis. All twelve units reported having a multiplace chamber. Five units also had monoplace chambers, with four reporting treating patients with diabetes in that chamber.

DEFINITIONS

Definitions for hypoglycaemia varied between units. Three-quarters (9/12) used < 4 $mmol \cdot l^{-1}$ as their definition, whereas the other three units each used definitions of < 5, < 3.6, and < 3 $mmol \cdot l^{-1}$. One half of the units (6/12) agreed that symptomatic hypoglycaemia was defined by the specific number they had chosen for hypoglycaemia (i.e., < 4 $mmol \cdot l^{-1}$) along with the addition of symptoms. The other one half of units defined symptomatic hypoglycaemia to be subjective or objective symptoms at any blood glucose level.

PREVALENCE OF DIABETES

A total of 210 patients had been treated amongst all units in the month surveyed of which 55 (26%) were diabetic (Figure 1). The median total number of patients treated per unit was 12 (range 3–38), of which a median of 5 (range 1–9) were diabetic. The prevalence of diabetes among the units ranged from 13–66% (Figure 1). Of the 55 patients with diabetes, 5 (7%) and 51 (93%) had types 1 and 2 diabetes, respectively, representing 2% and 24% of patients overall.

PROTOCOLS

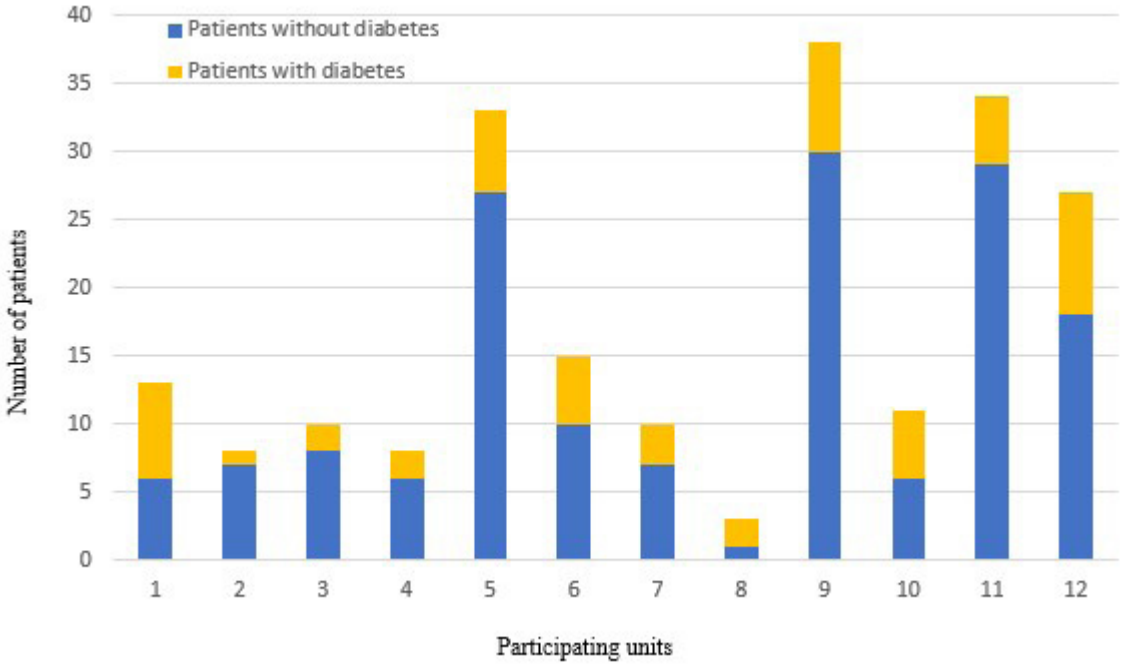
Ten (83%) of the units reported a specific written protocol for management of patients with diabetes in the hyperbaric chamber. Among the other two, one had a strict set of verbally agreed upon guidelines and one reported having no defined protocol. Of the 11 units with protocols, 8 (73%) reported that nursing staff were compliant with that protocol, whereas 5 (45%) responded that medical staff were likely to be compliant.

MULTIPLACE CHAMBER RESPONSES

Pre-chamber testing

Ten (83%) units routinely tested blood sugar levels on all patients with a diagnosis of diabetes regardless of type (1 or 2) or treatment (diet, oral tablets, or insulin). In the other two units testing was not initiated on those who were diet controlled. One of these also did not test those taking metformin as a sole oral agent.

Figure 1
Number of patients with and without diabetes treated in the month surveyed per hyperbaric medicine unit (numbered 1–12)



Seven units (58%) continued testing on all patients with diabetes prior to every treatment, whereas five (42%) discontinued testing under certain criteria. Two units ended if values were considered stable for diet-controlled patients. One unit stopped testing on all patients with diabetes after multiple treatments if blood glucose levels had been stable over an undefined time period. One unit discontinued testing on all patients with diabetes once blood glucose levels had been stable for one week. One unit ended testing if patients were considered stable, hypoglycaemia aware, and not on insulin.

Pre-treatment values

Five units (42%) target pre-treatment blood glucose was ≥ 8 mmol·l^{-1}. For four units (33%) this value was ≥ 6 mmol·l^{-1}. Two units (17%) targeted ≥ 5 mmol·l^{-1}. One unit described a range of acceptable values between of 4–10 mmol·l^{-1} (Figure 2). The pre-treatment levels were an absolute requirement for two units with the other 10 relying on clinical judgement if values were: close to the desired level, trending upward, if patients had recently eaten, or if no insulin had been given.

Patients were considered not suitable for treatment on a given day for several reasons. Four units deferred treatment if a patient's blood glucose level was less than the desired pre-treatment level. Two units did not treat patients if their blood glucose levels were unstable or trending downward. Six units used criteria to determine eligibility of patients when pre-test values were lower than the desired initial level. These criteria included: symptoms, blood glucose not rising after the patient was given a carbohydrate, fasting patients with type 1 diabetes, blood glucose trending downward, and recent dose of short acting insulin. One unit also deferred treatment for elevated blood glucose levels > 25 mmol·l^{-1} with no insulin given.

In-chamber testing

In-chamber glucose checks were able to be performed in all twelve units. One half of the units tested blood within the chamber with the other half transferring blood out for testing. There was significant variability in the protocols for testing during treatments. Three (25%) units tested all patients with diabetes. One unit stopped routine testing after three stable treatments. The remainder (67%) tested based on criteria that included symptoms, staff concerns, lower than usual pre-test levels, and whether insulin had been given prior to entering the chamber.

Post-treatment testing

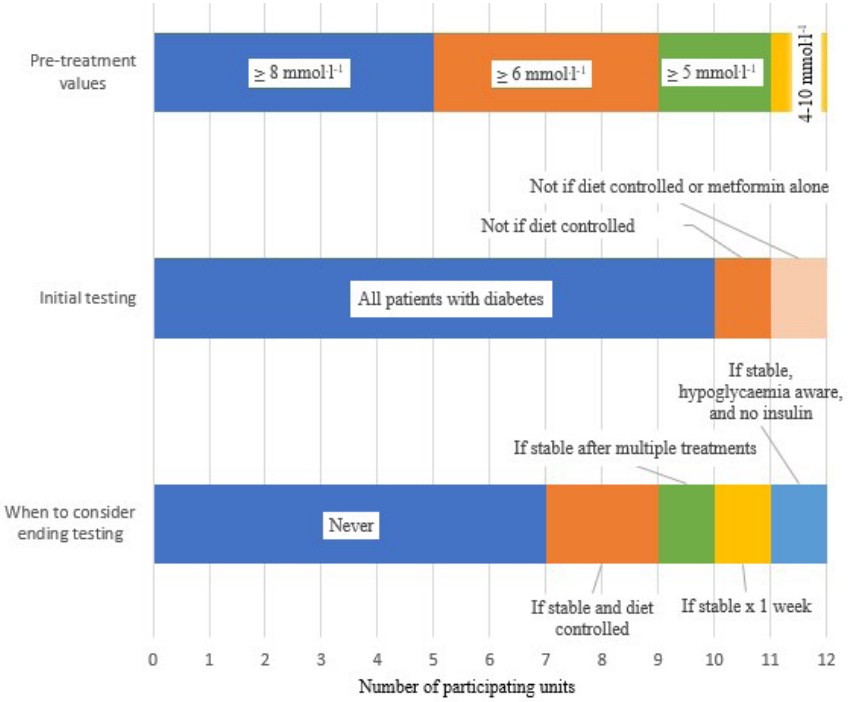
Post-treatment testing was reported as routine by two (17%) units while three (25%) reported no prescribed testing. Criteria-based testing was employed in the remaining seven (58%) units with indications including in-chamber events, stability of blood glucose during previous runs, type 1 diabetics, and hospital inpatients.

Carbohydrate usage

Prior to treatment, eleven (92%) units gave carbohydrate if blood glucose levels were lower than their pre-treatment

Figure 2

Multiplace chamber responses to survey questions (12 hyperbaric units); 'pre-treatment values' refers to target blood glucose prior to HBOT; 'intial testing' refers to conducting blood glucose measurement prior to each HBOT treatment



desired level. One unit gave carbohydrate regardless of level if patients had not eaten or were symptomatic.

Once a carbohydrate had been given, all units reported conditional treatment if glucose was rising on subsequent testing. If a carbohydrate had been given and blood sugars were stable, four (33%) units allowed treatment, two (17%) would not, and six (50%) used criteria such as absolute level of blood glucose or other factors (no recent insulin, recently eaten, previously stable blood glucose level in chamber) to determine eligibility for treatment. Carbohydrates were given during treatment in the multiplace chamber by all twelve (100%) units with reasons for this being directed by in-chamber blood glucose levels in one half and symptoms in the other half of units.

MONOPLACE CHAMBER RESPONSES

Among the four units that treated diabetic patients in their monoplace chambers, pre-treatment blood glucose requirements were ≥ 8 mmol·l^{-1} for three units and a range of 4–10 mmol·l^{-1} for one (Figure 3). Units reported a tendency for stricter adherence to specific blood glucose thresholds with monoplace as compared to multiplace treatments. If a patient's initial blood glucose level was below the pre-treatment threshold in three of the four units, treatment was conditional on a carbohydrate being given and subsequent blood glucose levels being higher than the pre-treatment threshold. One unit required three stable multiplace treatments to be eligible for monoplace treatment. Two units specified that monoplace patients had sugary drinks available within the chamber.

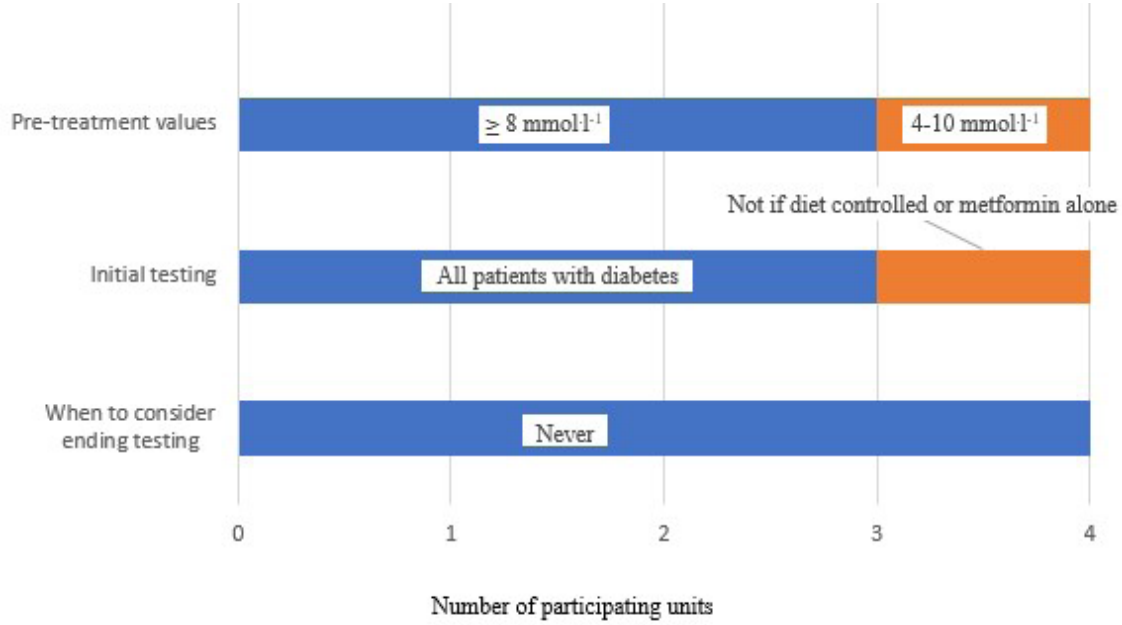
Post-treatment testing was performed routinely by two of the four units, and all four maintained regular testing on patients using the monoplace chamber even if stable over ongoing treatments (Figure 3).

If patients experienced a hypoglycaemic event in the monoplace chamber, three units transferred that patient back to the multiplace chamber for the remainder of their treatments, and one unit allowed ongoing monoplace treatments but with a higher pre-treatment blood glucose requirement.

SATISFACTION

Overall, eight (67%) units indicated they were satisfied with their current policy. The two units without written policies each mentioned they would like to formalise a policy. Potential changes suggested for improvement among existing protocols included: more detail regarding reasons to test pre-treatment and mid-treatment, specifying dose of carbohydrate to be used, having different criteria for patients with type 1 and 2 diabetes, and expanding opportunity for patients with type 2 diabetes to treat in a monoplace chamber.

Figure 3
Monoplace chamber responses to survey questions (4 units); 'pre-treatment values' refers to target blood glucose prior to HBOT; 'intial testing' refers to conducting blood glucose measurement prior to each HBOT treatment



Discussion

This study identifies that patients with diabetes are commonly treated with HBOT, they are treated in both monoplace and multiplace chambers, and most units have a protocol to help guide management of blood glucose levels. A majority of units reported routine and ongoing blood glucose testing of patients both prior to and during HBOT. There was marked heterogeneity in practice related to defining safe pre-chamber glucose levels and how these were subsequently managed, including indications for deferring HBOT. This survey highlights the burden of diabetes on patients treated with HBOT and identifies variability in practices which may benefit from further study to optimise management of these patients.

Diabetic protocols centre around optimal pre-treatment levels of blood glucose. On one hand, pre-HBOT levels that are too low may be exacerbated by treatment with a risk for hypoglycaemic symptoms and seizures. On the other hand, requirement of levels that are too high may lead to cancellation of treatments or exacerbation of diabetic complications associated with poor control. Glycaemic control, and in particular a haemoglobin A1c of < 8% has been shown to improve wound healing during the treatment of diabetic foot ulcers and to decrease amputation rates.[18,19] If criteria for HBOT require elevated glucose levels to enter treatments, then patients may be encouraged to maintain higher levels than normal so as not to be excluded from treatment.[17] In addition, the multiple glucose checks often required during a treatment may augment this concern without improving management.[15]

Units in our study targeted pre-HBOT levels from ≥ 4 mmol·l^{-1} to ≥ 8 mmol·l^{-1}. The marked heterogeneity and lack of consensus between units mirrors the literature which offers pre-treatment suggestions from > 6 mmol·l^{-1} to ≥ 9.4 mmol·l^{-1}, with multiple values in-between.[8,10] Interestingly, 3/12 of units in our survey use numbers lower than these. These units each expressed satisfaction with their protocols suggesting few adverse events. It could be postulated that use of lower numbers would encourage glycaemic control and decrease exclusions without significantly increasing risk compared to higher entry criteria, but further evidence is required.

Comments and criteria included in the survey indicate a focus on insulin use to determine initial and ongoing testing, with stability of blood glucose being used to determine termination of testing. While two studies conclude that any patients with diabetes using insulin are at higher risk for hypoglycaemic events,[6,20] other authors found that that this risk is related to those patients with type 1 diabetes and not those on insulin therapy *per se*.[11] This is further supported by another group who observed that patients with type 2 diabetes had more treatments with a drop in blood glucose than those with type 1, but of patients with lower post treatment levels (< 5 mmol·l^{-1}), 70% were on insulin alone.[13] Interestingly, one study found that when patients had a standardised meal and medications prior to treatment, non-insulin dependent patients had a significant decrease in their blood glucose level, but insulin-dependent patients did not.[7] When considering the postulated mechanism of HBOT increasing peripheral insulin sensitivity,[4,5] it would make most sense that patients with type 2 diabetes should

have a more consistent, but predictable decrease in their blood glucose levels during treatments. Patients requiring insulin, would have more variable drops that would be more dependent on their diabetic control overall. This is further supported by the observations that patients with good diabetic control over their course of treatments, evidenced by a change of < 2.8 mmol·l^{-1} in all of their blood glucose readings, experienced no hypoglycaemia.[13]

Only two studies have prospectively examined interventions to minimise hypoglycaemia during hyperbaric treatments. One group created a protocol based on blood glucose changes from 3,136 HBOT sessions in their hyperbaric unit.[15] They examined outcomes before and after introduction of this protocol which excluded patients on an intravenous insulin infusion and who could not communicate hypoglycaemia. Utilising the protocol criteria of not continuing to test patients with a pre-chamber blood glucose of > 8.3 mmol·l^{-1} and specifically defining testing and carbohydrate dosage for those with blood glucose between 3.9 and 8.2 mmol·l^{-1}, they noted the incidence of hypoglycaemia decreased from 1.5% to 0 in the short time frame studied and the number of finger prick tests done decreased by 33%. A second group examined a scoring system incorporating pre-treatment teaching, a risk analysis profile (diabetic control and complications), and pre-treatment glucose.[21] This score was modified daily based on timing of food and medications. Their incidence of hypoglycaemic events using this system decreased from 1.3/100 diabetic patients to 0.16/100. Interestingly, none of the units in our survey utilised either protocol. All units, however, used criteria closer to the second of the above approaches to routinely modify their own protocols. This multifactorial approach, with more specific criteria on who to test, and when to end testing, could be incorporated into a more comprehensive protocol to maintain glycaemic control and maximise patients included in treatment.

In our survey, approaches to glucose management in monoplace chambers tended to be more conservative than the literature. In a series of 1,825 monoplace HBOT treatments in 77 patients the authors required a pre-treatment level of > 6.7 mmol·l^{-1} and gave glucose to those with lower numbers, with an incidence of hypoglycaemia of 0.2%.[13] Another group[10] examined 700 HBOT sessions in a monoplace chamber, administering glucose to those with a blood glucose level of < 5.5 mmol·l^{-1} before the session and having an incidence of symptomatic hypoglycaemia of 0.29%.[10] Neither series reported any serious adverse outcomes. Of those units using the monoplace chamber in our survey, three of four required a pre-treatment blood glucose level of ≥ 8 mmol·l^{-1} (including following the administration of carbohydrate) to allow treatment. It may be that our survey respondents were more conservative than the literature because a multiplace chamber is available at each site meaning there is little need to incur any risk of hypoglycaemia within a monoplace unit.

Although it provides insight into contemporary practices related to glucose management in hyperbaric units in Australia and New Zealand, our study does have some limitations that merit discussion. As a survey we were only able to obtain reported practice which may differ from actual practice. We only surveyed one individual at each centre, and it is possible that responses may not be fully reflective of all staff at those centres. As patient data included in the survey was only collected over one month there may be bias in terms of numbers of diabetic patients treated due to natural fluctuation. Additionally, carbohydrate was not specifically defined in the questionnaire. Those responding units who contributed their protocols generally utilised 15 g of carbohydrate, however this may not be true of all units. As well, the use of rapid versus longer acting carbohydrate may have impacted decisions on further patient testing. Finally, our study is limited in that we did not ask for outcome data in terms of frequency of hypoglycaemic events, hypoglycaemic seizures, or numbers of cancelled treatments, as our aim was to keep the survey brief to encourage full participation by all sites. Given the significant variability in reported practice demonstrated by this survey, the potential influence of that variability on these outcomes would be an interesting area for further study.

Conclusions

Patients with diabetes are common in hyperbaric medicine units in Australia and New Zealand, accounting for 26% of all patients treated during the month surveyed. Survey responses indicate that blood glucose management protocols utilise similar principles to the satisfaction of most units. There is considerable variability in reported practice however, suggesting opportunities exist to enhance glycaemic control and facilitate patient treatment.

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A retrospective review of the utility of Chest X-rays in diving and submarine medical examinations

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Abstract
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Introduction: Performance of routine chest X-rays (CXRs) in asymptomatic individuals to assess hyperbaric exposure risk is controversial. The radiation risk may overshadow the low yield in many settings. However, the yield may be higher in certain settings, such as tuberculosis-endemic countries. We evaluated the utility of routine CXR in diving and submarine medical examinations in South Africa.
Methods: Records of 2,777 CXRs during 3,568 fitness examinations of 894 divers and submariners spanning 31 years were reviewed to determine the incidence of CXR abnormality. Associated factors were evaluated using odds ratios and a binomial logistic regression model, with a Kaplan-Meier plot to describe the duration of service until first abnormal CXR.
Results: An abnormal CXR was reported in 1.1% per person year of service, yielding a cumulative incidence of 6.5% (58/894) of the study participants. Only four individuals had a clinical indication for the CXR in their medical history. A range of potential pathologies were seen, of which 15.5% were declared disqualifying and the rest (84.5%) were treated, or further investigation showed that the person could be declared fit.
Conclusions: In South Africa, a routine CXR has a role to play in detecting abnormalities that are incompatible with pressure exposures. The highest number of abnormalities were found during the initial examinations and in individuals with long service records. Only four individuals had a clinical indication for their CXR during the 31-year span of our study. Similar studies should be performed to make recommendations in other countries and settings.

Introduction

Fitness-for-work evaluations comprise three components, namely (1) the ability to perform the inherent job requirements, (2) endurance to work the full shift and (3) absence of undue risk to self and others.[1] There is a considerable emphasis on the cardiorespiratory system during diving and submarine medical evaluations and chest x-rays (CXRs) are usually performed to exclude conditions that represent undue risk.[2] Some countries mandate annual CXR in divers and submariners while others (e.g., the Health and Safety Executive in the United Kingdom) recommend "*only upon clinical indication*".[3]

Exposure to ionising radiation carries obvious risk, described elsewhere.[4–6] International standards and guidelines for radiation safety are also available.[7–9] Many of these guidelines are now mandated through regulation in most countries, including South Africa.[10] Routine imaging studies may also uncover coincidental findings with unclear clinical significance, potentially leading to unnecessary disqualification from diving.[2] Conversely, coincidental findings may be valuable in isolated cases.[11]

Despite the risks, there is still a need to use radiological imaging on patients as part of diagnostic workup and treatment. There is however a paucity of information regarding the utility of routine radiological imaging performed on asymptomatic individuals as part of an occupational medical (fitness-for-work) programme in divers and submariners.[2,12] This is of particular importance in the South African context, where endemic diseases such as tuberculosis could lead to structural lung changes, considered incompatible with exposure to increased environmental pressure.[13] The main aim of this study was to evaluate the utility of routine CXR as a component of the fitness evaluation of persons in South Africa who would be exposed to increased environmental pressure as part of

their work. The study objectives included: to determine the incidence of abnormal CXR reports and describing the range thereof, to describe the incidence of false positive results and the subsequent response, and to determine whether the medical history and clinical examination indicated CXR imaging.

Methods

The study protocol was independently reviewed by two ethics committees, namely the Health Research Ethics Committee (HREC) of Stellenbosch University (Reference U18/04/011), and the Research Ethics Committee of the South African Military Health Services (SAMHS) (Reference 1MH/302/6/02.01.2018). Consent was thereafter obtained from the appropriate authority in SAMHS to access the study data.

The researchers retrospectively reviewed the clinical records of all individuals who underwent a diving and / or submarine medical examination employed in the South African National Defence Force (SANDF). All these examinations are performed by qualified diving medical officers and subsequently reviewed by senior medical officers at the Institute for Maritime Medicine. The employees are allocated a specific category based on their mustering. Divers have a '*D*' category and submariners an '*S*' category captured in the records. A fitness certificate has a 12-month maximum duration. Chest x-rays are usually performed with each medical, but some examiners opt out of requesting these, mainly due to radiation risk concerns. Apart from the routine fitness evaluations, all SANDF personnel and their families receive comprehensive medical services via the SAMHS at no cost. This includes all relevant paramedical and multidisciplinary services, and it is therefore exceptionally unlikely for personnel to have medical consultations outside of the SAMHS while in service. The medical records of each healthcare encounter (including clinical examinations, medical notes, special investigations, specialist reports, laboratory results, etc.) are kept indefinitely in an electronic format and can be retrieved for many years back.

The researchers reviewed all CXR reports of everyone with a '*D*' or '*S*' classification, denoting everyone who ever had a diving or submarine medical examination during the study period June 1987 to April 2018. Whenever an abnormality was reported on a CXR, all medical records of the individual for the preceding year were reviewed to determine whether there was a medical indication for the CXR, or whether an asymptomatic abnormality was detected by routine screening.

In order to minimise information bias, the researchers made use of all available resources, including hard copies of the fitness evaluation files, which are kept at the Institute for Maritime Medicine. The identification of study participants was based on the '*D*' and '*S*' categories, including those deemed permanently unfit during their initial medical – potentially as a result of CXR abnormalities. This ensured that everyone who ever had such a medical examination was included in the study, thus eliminating the healthy worker effect.

To determine the frequency of CXR abnormalities, the researchers calculated the incidence of having a first abnormal CXR report (to avoid multiple counts of the same individual), using the number of person-years in the study as denominator. Potential predictive factors available for analysis included the specific service branch, the sex and average age of participants while in diving or submarine service and their total duration of such service. The odds ratio (OR) of contingency tables (with 95% confidence intervals [CIs]) was calculated for individual categorical variables. Statistical significance of the associations was determined using the Chi-squared or Fisher's exact test. In addition, a binomial logistic regression model was used to determine variables associated with CXR abnormality. A significance level of 0.05 was used for all hypothesis testing. A Kaplan-Meier survival analysis was used to estimate the employment duration until first abnormal CXR.

Results

The study sample comprised every CXR performed on SANDF divers and submariners who had their first medical examination for diving or submarine service between June 1987 and April 2018. This included new recruits, as well as individuals transferring from other service units during this period. A total of 894 individuals were included in this study, of which 47% (n = 422) were divers, 51% (n = 451) were submariners and 2% (n = 21) were qualified for both. The participants' median age while in service was 27.6 (IQR = 23.7–33.8) years and they had a short median service duration of 3.3 (IQR 1–7.5) years (range 0 to 28.4) and similar between divers and submariners (P = 0.13). Only 6.7% (60/894) had service records exceeding 20 years, of which most (n = 43) were submariners. The study participants contributed a total of 5,281.4 person-years of service.

A total of 3,562 fitness-for-work examinations were performed on the study participants, including a total of 2,777 CXRs. There were thus 22% (785/3,562) examinations where the examining doctor did not request a CXR. Good source data existed for all study participants, with only 0.2% (5/2,777) CXRs not having a radiology report.

Seventy CXR abnormalities were reported in 6.5% (58/894) of the study participants (Table 1). The range of abnormal findings on the routine CXRs was similar between divers and submariners (P > 0.05).

Most (n = 50) had only one abnormal CXR reported, six individuals had two, one had three, and one individual had a series of six abnormal CXR reports (without fitness contraindication). Subsequent presentations of the same

Table 1

Number of particular chest X-ray (CXR) abnormalities reported for the first time among all study participants; ǂ – an individual with an abnormal CXR may present with more than one abnormality. Seventy distinct abnormalities were reported in the CXRs of the 58 individuals. The same abnormality reported in the same individual in subsequent examinations was not counted again

Chest X-ray abnormalities	Found in 58 participants with abnormal CXRs ǂ
Scarring/Fibrosis	16
Pleural thickening	6
Opacities	8
Bronchial wall thickening	8
Nodular opacities	6
Pulmonary cyst	4
Granulomas	4
Calcification	2
Pulmonary infection	3
Bronchiectasis	2
Possible active TB	1
Other	7
'Abnormal' (no detail)	3

abnormality in the same individual were not counted again and are not included in the results. The incidence of first abnormal CXR reports was thus 10.98 per 1,000 person years (or 1.1% per person year). Many abnormal CXRs (n = 34) were seen during the first diving or submarine medical examinations of study participants (Figure 1). This means that 58.6% (34/58) of all persons with abnormal CXRs were identified during their first medical (when they first joined as new recruits or during their first transfer from other service units).

However, when the total number of individuals who had CXRs performed at a specific career duration is included as denominator, the percentage of first presentation of abnormal CXRs at each career point, yields a different picture. Eleven percent of all study participants had an abnormal CXR at the time of their first medical examination, and high percentages were thereafter also seen during the fourth year (9.1%), ninth year (11.1%), twelfth year (13.3%), fourteenth year (20%), sixteenth year (20%), 18th year (40%), twentieth year (50%), 22nd year (16.7%) and 23rd year (10%) of service (Figure 2).

When considering the full cohort of divers and submariners, the survival analysis indicates that almost 20% of individuals would have an abnormal CXR after approximately 30 years of service (Figure 3).

Twelve of the 58 individuals presenting with an abnormal CXR were declared as unfit for work, of which 9 were because of the CXR findings (Table 2). This constitutes a predictive value (for being declared unfit because of an abnormal CXR) of 15.5%, including one case of active TB disease detected by routine examination. For the rest of the cases (84.5%) with abnormal CXRs, the findings during

Figure 1

Number of divers and submariners with a chest x-ray abnormality reported for the first time (n = 58) by length of service (no denominator)

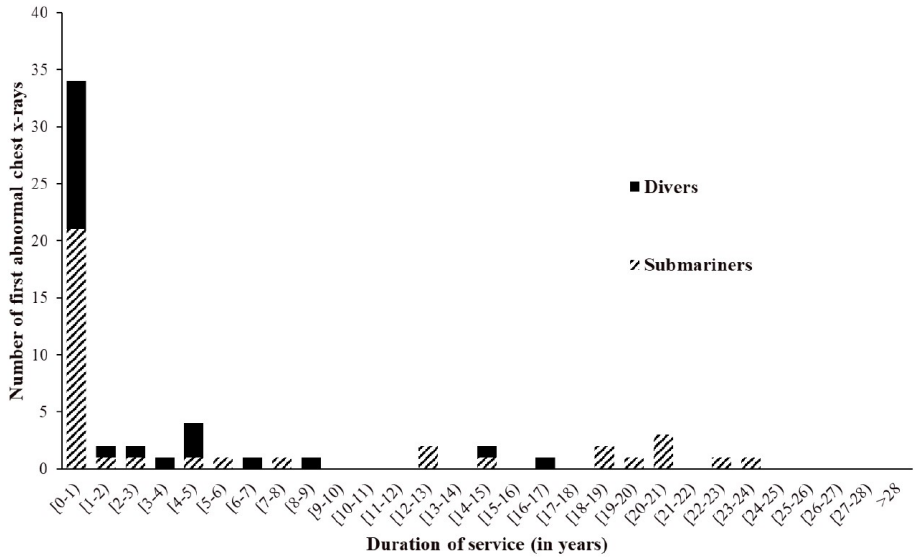

Figure 2
Percentage of divers and submariners with a chest X-ray abnormality reported for the first time (n = 58) by length of service of all study participants (n = 894)

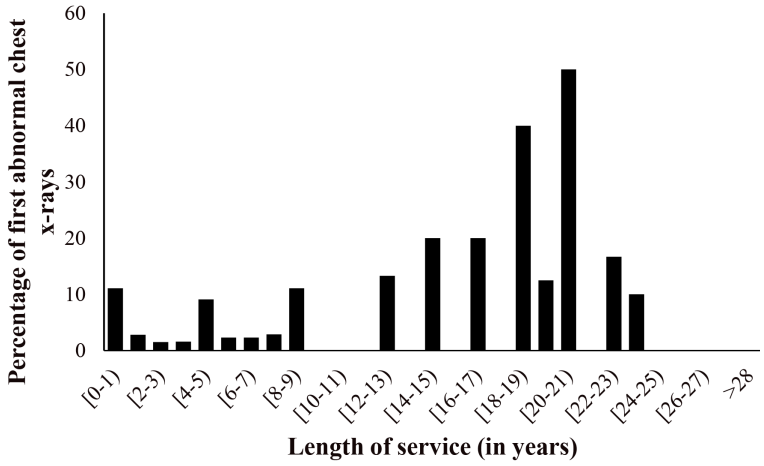

Figure 3
Survival analysis indicating duration of service until first report of an abnormal chest X-ray

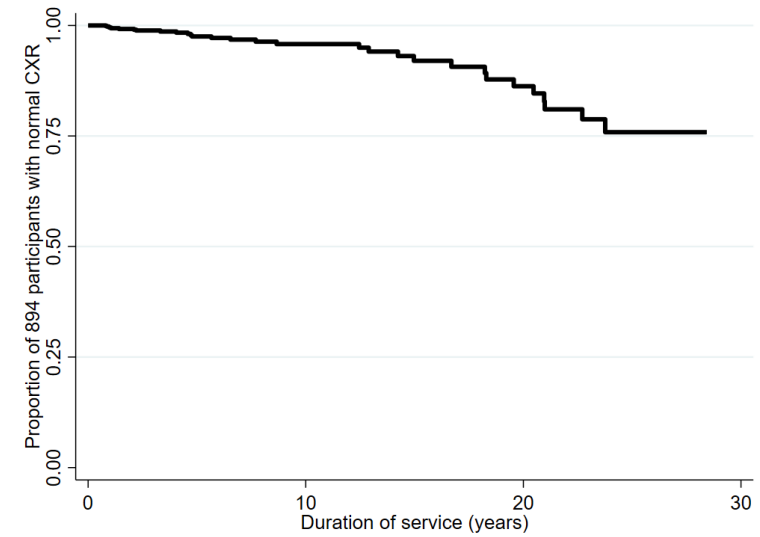

Table 2
Radiological follow-up of the 58 individuals with an abnormal CXR and impact on final fitness; § – one individual had both CXR and CT; CXR – chest X-ray; CT – computerised tomography; N/A – not applicable

Follow-up	Confirmed abnormal	Declared unfit
No follow-up (n = 31)	N/A	5
Follow-up CXR (n = 13)§	5	3
Follow-up CT (n = 15)§	4	4

follow-up evaluations denoted acceptable risk, and they were eventually declared fit for work.

When reviewing the full medical records of the preceding year for individuals presenting with an abnormal CXR, only 6.9% (4/58) had a possible clinical indication noted in their files, including one submariner who presented with respiratory symptoms and a diagnosis of active tuberculosis which was confirmed with CXR and sputum microscopy.

Thirteen percent of the study participants (n = 118) were female. However, sex did not constitute an independent risk factor (OR = 1.4, 95% CI = 0.7–3.2; P = 0.37). Submariners were more likely to present with CXR abnormalities than divers (OR = 2.2, 95% CI = 1.3–3.2; P < 0.01). The binary logistic regression model did not provide an adequate fit to the data (deviance = 242.6). Apart from the length of

service ($P < 0.05$), none of the variables could adequately predict CXR abnormality (including average age while in these service branches).

Discussion

The use of routine CXR is rightfully discouraged by several authors, particularly in low-risk settings.[2,14–19] In high-risk settings, routine CXRs are still advised.[20] Our study found a relatively high (6.5%) cumulative incidence of abnormal routine CXRs, and a very high proportion (up to 50%) were found to be abnormal in individuals with long service records. The Kaplan-Meier survival curve indicates that approximately one in five individuals with long service duration would be declared unfit for hyperbaric exposures. This curve should however be interpreted with caution, given the relatively short service duration of most study participants (median of 3.3 years).

Despite the high incidence of CXR abnormalities, only four individuals over the span of 31 years of our study had a clinical indication for CXR. A possible explanation is that divers and submariners would fail to report symptoms of illness for fear of losing their fitness certification. This is problematic for approaches using symptom-based screening (as recommended by others).[2] This problem is also recognised internationally among commercial divers and addressed in some detail in a publication of the International Marine Contractors Association (IMCA D 061).[21] This IMCA document provides examples of divers with severe illness who chose not to disclose this during medical examinations and in some cases, it resulted in fatality.

Individuals with longer service records have a higher probability of presenting with abnormal CXRs (irrespective of average age in service). The lack of association between length of service and age in our study is likely due to us using average age of individuals while serving in diving or submarine branches, rather than the average age over their full career. Some individuals have transferred from other service branches (e.g., surface vessels) and were only employed for very short diving or submarine service periods, while others spent their full career in these branches.

The association of abnormal CXR reports with service duration may indicate work-related pathology as a possible cause. Scarring, fibrosis, nodular changes and pleural thickening may be caused by specific occupational exposures (and included asbestos-related disease in some of our study participants). Apart from clinical (cardiopulmonary and other symptom) indications it is therefore also important to consider other risks, such as workplace exposures and a high prevalence of infectious diseases in the community, as indications for a CXR. For instance, nearly one quarter of tuberculosis cases among HIV-infected adults in South Africa were subclinical.[22] In a high prevalence setting, it is therefore conceivable that lung pathology can be present without the individual having symptoms to indicate the need for CXR. Furthermore, while numerous international policies propose screening based on symptoms as clinical indication, it is difficult to implement such a policy in a setting where there is a real incentive for employees to conceal their symptoms for fear of losing their fitness certification and jobs.[2] Computed tomography seems preferable in symptomatic individuals, but may result in unnecessary exclusions from work.[2] Similar dilemmas have been reported with routine CXRs in other settings.[23] Despite this concern, some countries still implement routine low-dose chest CT as part of military diver screening.[12] A checklist and clinical decision-making tool was recently developed to guide targeted baseline thoracic imaging in persons undergoing hyperbaric oxygen therapy.[24]

The apparent regular interval 'peaks' in our data for reporting a CXR abnormality (Figure 2) should be interpreted with caution, since CXRs were not requested in 22% of examinations and this examination was therefore not done with every annual fitness examination. These 'peaks' may thus represent information bias artifacts based on the frequency with which CXRs were requested.

Several variables that could potentially predict abnormal CXRs were not evaluated in our study. Some variables are prohibited by local legislation (e.g., HIV status, denoting increased risk for tuberculosis scarring, may only be tested in employees if sanctioned by the Labour Court).[25] Other variables (e.g., using race as proxy) were specifically excluded due to ethical concerns.[26] Future studies should consider a wider range of variables that may better predict the need for CXR and further refine indications for routine CXR, particularly including workplace risk assessments.

Conclusions

Our results indicate that there is a role for routine CXR in asymptomatic South African divers and submariners. The highest number of cases were found during the baseline (first medical) examination and in individuals with a long service history, suggesting that occupational exposures may play a role. While CXRs may be indicated based on the medical history of the individual, this was rarely the case in our study population, with only four cases thus detected in the 31-year span of our study.

We advise that similar studies be performed in other settings and other countries, to provide an evidence-based approach to the routine use of CXRs in occupational health fitness screening programmes.

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A retrospective review of divers treated for inner ear decompression sickness at Fiona Stanley Hospital hyperbaric medicine unit 2014–2020

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Diving medicine; Diving research; ENT; Hyperbaric oxygen; Persistent (patent) foramen ovale (PFO); Right-to-left shunt; Vertigo

Abstract
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Introduction: Inner ear decompression sickness (IEDCS) is increasingly recognised in recreational diving, with the inner ear particularly vulnerable to decompression sickness in divers with a right-to-left shunt, such as is possible through a persistent (patent) foramen ovale (PFO). A review of patients treated for IEDCS at Fiona Stanley Hospital Hyperbaric Medicine Unit (FSH HMU) in Western Australia was performed to examine the epidemiology, risk factors for developing this condition, the treatment administered and the outcomes of this patient population.

Methods: A retrospective review of all divers treated for IEDCS from the opening of the FSH HMU on 17 November 2014 to 31 December 2020 was performed. Patients were included if presenting with vestibular or cochlear dysfunction within 24 hours of surfacing from a dive, and excluded if demonstrating features of inner ear barotrauma.

Results: There were a total of 23 IEDCS patients and 24 cases of IEDCS included for analysis, with 88% experiencing vestibular manifestations and 38% cochlear. Median dive time was 40 minutes and median maximum depth was 24.5 metres. The median time from surfacing to hyperbaric oxygen treatment (HBOT) was 22 hours. Vestibulocochlear symptoms fully resolved in 67% and complete symptom recovery was achieved in 58%. A PFO was found in 6 of 10 patients who subsequently underwent investigation with bubble contrast echocardiography upon follow-up.

Conclusions: IEDCS occurred predominantly after non-technical repetitive air dives and ongoing symptoms and signs were often observed after HBOT. Appropriate follow-up is required given the high prevalence of PFO in these patients.

Introduction

Inner ear decompression sickness (IEDCS) has traditionally been associated with deep, mixed gas diving.[1] Bubble formation in the vestibulocochlear system was partly attributed to gas counter-diffusion, where bubbles formed as a result of transient super-saturation when the breathing gas was switched from helium to nitrogen during ascent.[2] It is however becoming increasingly recognised that IEDCS may also occur in non-technical air diving, demonstrating that there are other mechanisms by which the inner ear structures are susceptible to damage from bubble formation.[3–6] Previous research has suggested the association of a right-to-left shunt as a predisposing factor, whereby arterialised gas emboli can potentially grow due to slower inert gas washout from tissues of the inner ear.[6,7]

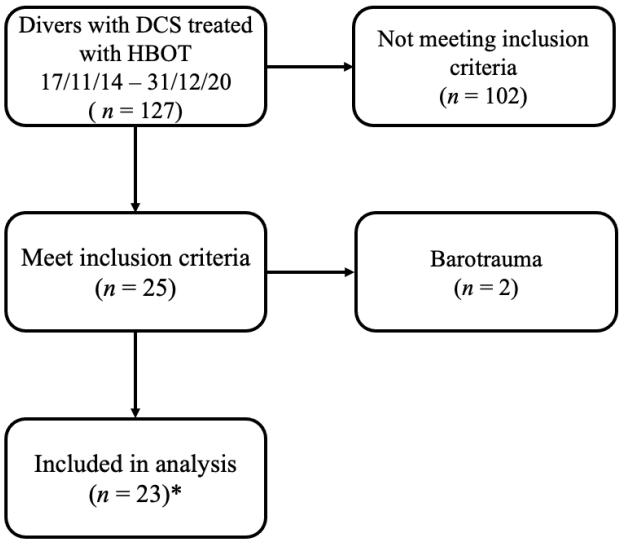
Inner ear decompression sickness is characterised by bubble mediated injury to the vestibulocochlear system. Vestibular manifestations including vertigo, nystagmus, nausea and vomiting have been found to present more commonly than those of the cochlear system, encompassing hearing loss and tinnitus. The propensity for vestibular symptoms to predominate is thought to be due to lower perfusion and hence slower inert gas washout of the vestibular system compared with the cochlea.[3]

A universally accepted definition of IEDCS is lacking and studies to date vary in their inclusion criteria. Other dive related injuries such as cerebellar decompression sickness (DCS) or inner ear barotrauma (IEBt) can display symptoms similar to IEDCS and the diver with symptoms of vestibulocochlear dysfunction can present a diagnostic challenge to the clinician.[8] A review has recently described the most useful variables in differentiating IEBt from IEDCS, including dive type (breath-hold versus scuba), dive gas (compressed air versus mixed gas), dive profile (mean depth 13 versus 43 metres), symptom onset (upon descent

versus ascent or after surfacing), symptom distribution (vestibular versus cochlear) and absence or presence of other DCS symptoms.[9] It is important to perform a thorough assessment to attempt to differentiate IEDCS from IEBt, although case data has shown that a trial of hyperbaric oxygen treatment (HBOT) may not worsen IEBt if it cannot be excluded, provided the diver can equalise their ears.[10]

The aim of this study was to investigate the epidemiology of patients with IEDCS treated at Fiona Stanley Hospital Hyperbaric Medicine Unit (FSH HMU) in Western Australia (WA) and provide a description of risk factors for developing the condition, in addition to investigating treatment outcomes for this population. The FSH HMU is the WA State Referral Service for diving and hyperbaric medicine and takes referrals from throughout the state, as well as Australia's Indian Ocean territories (Cocos-Keeling and Christmas Islands). Western Australia has the longest coastline of any Australian state, providing a vast area for dive activities and potential challenges in the retrieval of injured divers.[11] The time to HBOT was compared with other case series from around the world to determine if this unique geography may affect treatment times and outcomes.

Figure 1
Flow diagram of data acquisition; *one diver was treated twice during the study period giving a total 24 episodes of IEDCS; DCS – decompression sickness; HBOT – hyperbaric oxygen treatment



Methods

Written approval was obtained for data review and extraction by Governance, Evidence, Knowledge and Outcomes (GEKO) at FSH (Approval Number 39297), and by the Human Research Ethics Committee of Curtin University (Approval Number HRE2021-0029).

PATIENT SELECTION

A retrospective review of the electronic medical record of all patients with a diagnosis of DCS presenting to FSH HMU from its opening on 17 November 2014 to 31 December 2020 was performed. The medical records were reviewed to identify those with symptoms suggestive of IEDCS. Patients were included if presenting with vestibular dysfunction (vertigo, nystagmus) and/or cochlear dysfunction (hearing loss, tinnitus) occurring within 24 hours (h) of surfacing from a dive. Patients with symptoms or signs of aural barotrauma (history of ear clearing difficulty, tympanic membrane erythema or haemorrhage), suggesting the alternative diagnosis of IEBt, were excluded.

DATA COLLECTION

Data were collected in an electronic spreadsheet and the following parameters were recorded: patient demographics including age and sex, diving experience, risk factors for DCS including dive time, maximum depth, rapid ascent, omitted decompression obligation, dehydration, thermal comfort, exertion during the dive, and repetitive diving within 24 h. Also recorded were breathing gas and delivery system used, plus DCS symptomatology including time from surfacing to onset of symptoms, vestibulocochlear symptoms reported, and associated DCS symptoms. Data regarding treatment and outcomes including time to first HBOT, initial treatment table used, total number HBOT and outcome following treatment including residual symptoms at discharge and follow-up were collected. Data were also collected regarding recommendations made for investigation of a right-to-left shunt, with results of these tests documented where available.

STATISTICAL ANALYSIS

Data were compiled from a manual review of FSH HMU electronic patient records, stored in MS Excel® and analysed using SAS version 9.4 (SAS, Cary NC, USA). One patient was treated twice for IEDCS, two years apart, and was counted separately (weighting per case = 1) when describing number of treatments received and dive conditions, but counted just once (given a weighting of 0.5 per IEDCS) when describing age, proportion male, and other demographic factors. Frequency counts are reported with percentages. Weighted age and unweighted dive time in minutes (min), maximum depth in metres (m), time to symptom onset from surfacing (min) and delay to recompression (h) are each reported as medians with interquartile ranges (IQR).

Results

There were 23 IEDCS patients, comprising 18% of patients treated with HBOT for DCS over the study period, and 24

cases of IEDCS included for analysis. A flow diagram of patient record identification is shown in Figure 1. Of the 23 patients, 21 (91%) were male, with median age 44 years (IQR 15).

Seven (30%) divers reported having less than 100 dives experience, another seven (30%) between 100–500 dives, and nine (39%) > 500 dives. Three (13%) divers reported previous DCS (one musculoskeletal treated with HBOT with details of the other two not documented). During the 24 dives that resulted in IEDCS, 20 (83%) divers were breathing air, three (13%) enriched air nitrox (32% oxygen (O_2)) and one was using a rebreather. There were 22 (92%) divers using scuba and two (8%) were using surface supply. Median dive time was 40 min (IQR 11) and median maximum depth was 24.5 m (IQR 10).

Substantial exertion underwater and being underweighted were reported by two (8%) divers, whilst being cold during the dive was reported by one (4%). One diver self-reported dehydration and drinking alcohol to excess on the evening prior to the incident dive. Nineteen (79%) were making repetitive dives within 24 h. Four (17%) reported a rapid ascent and four (17%) reported omitting a decompression obligation. Half the divers reported making a safety stop and half did not. Five (21%) divers reported substantial exertion after the dive.

Median delay to symptom onset after surfacing was 20 min (IQR 41) and median delay between surfacing and treatment with HBOT was 22 h (IQR 59.5). Eleven (46%) cases were treated initially at our HMU, while 50% of patients were given first-aid at another hospital prior to transfer, and one (4%) patient had received prior initial recompression in Bali, Indonesia.

Of the 24 IEDCS cases, 21 (88%) showed vestibular involvement and nine (38%) cochlear involvement, with six (25%) having both vestibular and cochlear symptoms. Other manifestations of DCS were present in 18 (75%), which included constitutional (n = 13, 54%), musculoskeletal (n = 9, 38%), neurological (n = 6, 25%), cutaneous (n = 5, 21%), and spinal (n = 1, 4%). Hearing tests were documented for 11 (46%) patients and three (13%) were prescribed corticosteroids for hearing loss. The initial HBOT table used was the United States Navy Treatment Table 6 (USN TT6) for 23 (96%) patients, with one (4%) receiving a 18:90:60 treatment table (284 kPa / 2.8 atmospheres absolute for 90 min with a 60 min decompression). The total number of HBOT received ranged from 1–15 (median 6, IQR 6.5), with 10 (42%) cases reporting residual symptoms after their final treatment. Adjunctive therapy with intravenous (IV) fluids was administered in 17 (71%) cases. Of the 23 patients, 13 (57%) were advised to undergo bubble contrast transthoracic echocardiography to investigate for a right-to-left shunt, with 10 (77%) having this performed. This confirmed the presence of a persistent foramen ovale (PFO) and hence an intracardiac shunt in six (60%) of the 10 tested patients. No intrapulmonary shunts were identified. Table 1 shows patient and incident dive demographics, symptoms, treatment administered and outcomes.

Discussion

The aims of this study were to describe the epidemiology and risk factors for IEDCS cases treated at FSH HMU and to describe their treatment outcomes. Patients with IEDCS comprised 18% of divers treated for DCS during the study period. This is similar to other case series, which have reported the incidence of IEDCS to be between 16% and 24% of treated DCS cases.[4,5] A study from Malta has shown that the incidence of IEDCS had increased between 1987–2017, with the cause for this hypothesised to be due to changes in the practice of recreational diving, with more divers diving deeper, repetitively, and with reverse profiles.[12]

The median maximum depth of 24.5 m for the incident dive is shallower than previously reported case series, yet the median dive time of 40 min is similar.[4] This may reflect a difference in recreational diving practices or the coastal topography in Western Australia. Two patients had symptom onset after 10 m incident dives, without a history of rapid ascent to suggest cerebral artery gas embolism. One made a repetitive dive with significant physical exertion at depth and multiple freedives during the preceeding surface interval, with subsequent bubble contrast echocardiography negative for a PFO. Another presented with unilateral sensorineural hearing loss with tinnitus, without clinical evidence of peri-lymph fistula upon otolaryngologist review. Right-to-left shunt testing was not performed in this patient. The vast majority of divers were breathing scuba air, with three using nitrox and one using a rebreather. This supports the hypothesis that IEDCS occurs via mechanisms other than supersaturation caused by gas switching, as occurs in deep technical diving.[1]

The most common risk factor for IEDCS in this study was repetitive diving within 24 h, with 19 (79%) of divers reporting this. This is consistent with recent case series where predisposing factors related to inert gas uptake, or on-gassing, were found to be the predominant risk factors for IEDCS.[5,12]

The kinetics of inert gas in the inner ear has been described through the use of a three compartment model.[2] The inner ear comprises the membranous labyrinth, the perilymph and the endolymph. Only the membranous labyrinth has a vascular supply, and hence is the location for inert gas washout. It has been described that there is a prolonged period of supersaturation of inert gas in the membranous labyrinth immediately after surfacing from a dive, when compared with brain tissue.[13] This suggests how bubbles,

Table 1

Patient demographics, incident dive characteristics, symptomology, treatment and outcomes; *one diver was treated twice during the study period; C – cochlear; DCS – decompression sickness; F – female; HBOT – hyperbaric oxygen treatment; M – male; NP – not performed; PFO – persistent (patent) foramen ovale; V – vestibular; VC – vestibulocochlear

Age	Sex	Dive time (min)	Maximum depth (m)	Symptom onset from surfacing (min)	Vestibulocochlear symptoms	Other DCS symptoms	Delay to recompression (h)	Total number HBOT	Residual symptoms at HBOT completion	Result of transthoracic bubble contrast echocardiography
25	F	60	18	30	V	Yes	88	2	Nil	NP
27	M	44	30	15	V	Yes	20	7	Ataxia	PFO
27	M	40	12	240	V	Yes	24	3	Nil	NP
30	M	46	32	5	V	Yes	5	8	Nil	NP
34	M	42	15	120	C	Yes	28	2	Nil	NP
36	M	60	10	0	V	Yes	216	2	Nil	Normal
39	M	20	28	0	V	Yes	7	3	Musculoskeletal	NP
41	M	43	10	1140	C	No	90	10	Nil	NP
41	M	29	37	3	VC	Yes	11	15	Tinnitus	NP
42	M	125	18	90	V	No	9	10	Ataxia	PFO
43	M	37	28	60	V	No	9	8	Nil	PFO
44	M	58	19	0	V	Yes	24	2	Nil	NP
44	M	35	30	15	VC	No	8	6	SNHL	NP
45	M	36	20	20	V	Yes	20	2	Nil	PFO
47	M	15	24	15	VC	No	164	7	SNHL	NP
48	M	60	28	30	V	Yes	4	6	Nil	PFO
50	F	35	18	240	V	Yes	48	2	Nil	NP
51	M	40	25	30	V	Yes	5	7	Ataxia	Normal
52	M	?	62	0	VC	Yes	115	9	Nil	Normal
55	M	40	15	0	C	Yes	192	1	Headache	NP
55	M	40	20	20	V	No	20	10	Nil	Normal
70 *	M	25	25	10	V	Yes	30	3	Nil	PFO
72 *	M	25	25	30	VC	Yes	6	6	Ataxia, tinnitus	PFO
72	M	45	25	20	VC	Yes	30	9	Ataxia	NP

entering the arterial circulation through a PFO, can grow in the membranous labyrinth due to the localised increased partial pressure of gas. This is postulated to be a mechanism by which isolated IEDCS can occur.

The second most common risk factor for IEDCS was exertion and heavy lifting after the dive, present in five (21%) divers, with three of those being recommended to have bubble contrast echocardiography. Of those, two were tested, confirming the presence of a PFO in both. Increased right atrial pressure, as occurs after the release phase of the Valsalva manoeuvre, or after heavy lifting with a closed glottis, may shunt blood from the venous to the arterial circulation in those with a PFO.[14] It has been shown that bubbles may be detected in the venous system after surfacing from a dive, even when diving within no-decompression limits and they are predominantly filtered by the lungs.[15] When the bubbles become arterialised through a PFO, divers are at increased risk of certain subtypes of DCS, including cerebral, spinal, cutaneous and inner ear.[16,17]

A safety stop was not documented in half of the cases reviewed, which may be a contributory risk factor for developing DCS, however there could be limitations in documentation. Nevertheless, the performance of a safety stop when diving is recommended for reducing the risk of DCS and this may reflect the need for improving diver education. More pertinent to the risk of developing DCS was the omission of a decompression obligation. This was present in four (17%) cases, increasing the risk of bubble formation in this cohort. The time from surfacing to onset of symptoms varied from zero to 1,140 min, with a median of 20 min (IQR 41). This is similar to previously reported onset times for IEDCS.[4] Symptoms developed within two hours of surfacing in 88% of divers, with two divers developing symptoms at four hours and one patient developing isolated severe unilateral sensorineural hearing loss with tinnitus at 19 h. This outlier made a complete recovery with normal hearing thresholds on audiometry after 10 HBOT sessions with adjunctive oral corticosteroids.

The majority (88%) of patients had vestibular symptoms, with fewer (38%) showing cochlear involvement, which is a consistent finding in other reports.[4,5] The predisposition for vestibular symptoms is thought to be due to the vestibular apparatus having a quarter of the blood flow than that of the cochlea with an increased tissue volume, causing slower gas washout and prolonged supersaturation and therefore propensity to bubble growth within the vestibule.[7] Isolated IEDCS was present in 25% of patients, while the majority (75%) had other systems involved. This trend is similar to findings from a recent report.[5] The predominant associated DCS symptoms were constitutional (54%), followed by musculoskeletal (38%), neurological (25%), cutaneous (21%), and spinal (4%).

The time to first HBOT varied from four to 216 h (median 22, IQR 59.5). The median time to recompression was longer than in previous studies of IEDCS.[4] This could be due to the vastness of WA and distances involved in transporting injured divers for treatment in Perth, where our HMU has the only hyperbaric chambers in WA available for civilian use. Twelve patients were recompressed within 24 h, with seven (58%) of those having ongoing symptoms at completion of HBOT. Twelve patients had a delay to recompression of greater than or equal to 24 h, however only three (25%) of these had ongoing symptoms at discharge. Patients who presented earlier could have been sicker, which could account for the increase in residual symptoms seen in this cohort. However, our numbers are small and a recent study found that patients with decompression illness that present with tinnitus or hearing loss have worse outcomes with increased residual symptoms when recompressed beyond 48 h.[18]

Thirteen patients initially presented to healthcare facilities other than our own. Two of these were outside of Australia; in Bali Indonesia where HBOT was administered to one patient, and in Fiji where normobaric O_2 was given, with both of these patients flying on standard commercial flights to Perth where they were subsequently reviewed. Of the 11 patients who presented to other facilities within WA, all were given normobaric O_2 and IV fluids at the initial receiving facility. Five of these arrived to our HMU by private vehicle, with distances driven ranging from 160 km (Bunbury, WA) to 1,200 km (Exmouth, WA). Four were transferred by road ambulance with ongoing normobaric O_2 and IV fluids, of which three were from local metropolitan hospitals and one was from Bunbury, WA. Two patients were transported by fixed wing air ambulance, by the Royal Flying Doctor Service, in cabins pressurised to sea level, with face mask O_2 and IV fluids during transfer. Decisions regarding the transport method to our unit depend on clinical stability, response to first aid, distance involved and the capacity of retrieval services.

The United States Navy Table 6 was the initial treatment table for all but one patient, who received treatment table 18:90:60. This patient had been diving in Fiji three days prior to presentation to our HMU and developed a rash 30 min after surfacing, with associated limb pain and headache, which was treated there as a presumed allergic reaction with corticosteroids and antihistamines. A few hours later they subsequently developed worsening musculoskeletal pain with vestibular symptoms, which fully resolved after two HBOT sessions upon return to Perth. They were advised to have investigation for a PFO, however they had not had this performed upon follow-up.

Residual symptoms were present in 10 (42%) patients at discharge, with the majority (58%) being symptom free post-HBOT. Ongoing symptoms were isolated vestibular for

four patients, isolated cochlear for three, vestibulocochlear for one and other DCS manifestations for two (neurological and musculoskeletal). This demonstrates that IEDCS may be refractory to treatment and symptoms may persist despite treatment. Of the 10 with ongoing symptoms after completion of HBOT, documentation of subsequent follow-up was available for four patients. Two reported persistent tinnitus, one had ongoing severe sensorineural hearing loss, and one patient had complete resolution of shoulder pain. The long-term consequences of IEDCS could not be fully explored due to limitations in the data, however we have shown that a select group of patients had ongoing deficits to both the vestibular and cochlear systems.

A previous study on IEDCS reported ongoing vestibulocochlear deficits upon follow-up in 91% of patients.[19] Of eight patients with ongoing vestibular deficits, only one (12.5%) was symptomatic. This could be explained by the more extensive otoneurologic assessment used in that study, which included electronystagmography, alternate bi-thermal caloric testing, sinusoidal harmonic acceleration testing and computerised dynamic posturography. It is important to consider the implications regarding return to diving for patients with a history of vestibular IEDCS, as a high proportion may have ongoing underlying vestibular dysfunction despite apparent resolution of symptoms. An unfamiliar underwater environment could overwhelm the brain's compensatory mechanisms that develop after an insult to the vestibular system with potentially serious consequences.[19] A more recent study found 69% of patients recovered completely after treatment for IEDCS, which is more reflective of our cohort.[5] A thorough assessment and follow-up is recommended for all patients presenting with IEDCS, as ongoing deficits can be subtle, with audiometry a minimum for testing on discharge.

Investigation for a right-to-left shunt with bubble contrast echocardiography was advised for 13 patients, of which 10 had the investigation, with six (60%) of these being positive. This supports the hypothesis that IEDCS is associated with a right-to-left shunt as the prevalence of a PFO in the general population is approximately 25%.[20] The reason for not investigating the remaining 10 patients with bubble contrast echocardiography was not documented. Once bubbles pass into the arterial circulation, they can cause damage to the inner ear through a number of mechanisms. The vestibular vascular supply is an end-artery, thus occlusion or endothelial bubble stripping can cause localised inflammation and ischaemia.[21] Bubbles can increase in size due to localised supersaturation of the inner ear, causing selective vulnerability of the inner ear to DCS in those with a right-to-left shunt.[13] Due to the increased risk of certain subtypes of DCS in those with a PFO, it is advised to consider PFO testing with trans-thoracic bubble contrast echocardiography, including provocative testing with Valsalva and sniffing, for patients presenting with IEDCS, as per the South Pacific Underwater Medicine Society and United Kingdom Sports Diving Medical Committee joint position statement.[22]

A disproportionate number (91%) of the patients in this IEDCS study were male, although our dataset is too small to infer an association between gender and IEDCS. Historically, there have been three surveys of recreational divers in WA; the first in 2000 ($n = 540$) found 72% of divers were male, the second in 2006 ($n = 499$) 75%, and the third in 2014 ($n = 139$) 73%.[23–25] Of 24 WA recreational diving fatalities described in 2009, 16 (75%) were male.[26] Of the 83 divers treated for decompression illness at our HMU reported in 2020, 80% were male.[27] Much larger numbers would be needed to determine whether any gender related pre-disposition exists.

Two patients were excluded from the study due to signs of middle ear barotrauma (MEBt), suggesting a likely alternate diagnosis of IEBt, however no patients reported difficulty equalising. A case series of 50 patients with IEBt found 38% had no evidence of MEBt at otoscopy, hence the absence of erythema of the tympanic membrane does not exclude IEBt.[28] Features suggestive of IEBt include vestibulocochlear symptom onset during ear clearing manoeuvres or straining, a previous history of aural barotrauma and co-existent MEBt. IEDCS is increasingly likely in divers with a provocative dive profile, history of rapid ascent, omitted decompression obligation, coexistent symptoms of DCS or when symptoms occur after surfacing. Several investigations have been used to investigate suspected cases of IEBt including pure tone audiometry (+/- positional testing), the fistula test, high resolution temporal bone computed tomography scanning, electronystagmography, caloric testing, the Tulio phenomenon and otoacoustic emission testing.[10] These tests may be useful in helping differentiate the cause of vestibulocochlear symptoms in divers who present a diagnostic dilemma to the clinician.

LIMITATIONS

The study design is retrospective and reliant upon data collected from the medical records. Some data were missing and the diagnosis of IEDCS relied on assessments that had been documented for the purpose of treatment, not research. Also, dive profile computer downloads were not available so maximum tissue super-saturation pressures could not be factored in, nor ascent rates. Information on vestibular testing and hearing tests were limited. Long-term outcomes were difficult to assess given follow-up was documented for a minority (43%) of patients. Ten patients were not documented to have been recommended a PFO test, although guidelines suggest that all patients with IEDCS should have this considered, or else modify their diving practices.[22] This could be due to limitations in the documentation, however a new assessment form for divers is being developed in

our HMU to more accurately record details of clinical assessment, dive demographics, risk factors for DCS and follow-up.

Conclusions

This study presents data for all IEDCS cases treated at FSH HMU from 2014 to 2020. Divers were predominantly using scuba equipment breathing air, with a median maximum depth of 24.5 m, supporting previous literature that this disease is not isolated to deep technical diving. The main risk factor was repetitive diving and the vestibular system was found to be more vulnerable to DCS than the cochlear system. It is important to perform a comprehensive otoneurologic assessment for patients presenting with potential IEDCS, and counselling and follow-up is important given the high prevalence in this cohort of a PFO and right-to-left shunt, with implications for future diving practices and safety.

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Selecting optimal air diving gradient factors for Belgian military divers: more conservative settings are not necessarily safer

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Abstract
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Introduction: In 2018, the Belgian Defence introduced a commercial off-the-shelf dive computer (Shearwater Perdix™) for use by its military divers. There were operational constraints when using its default gradient factors (GF). We aimed to provide guidelines for optimal GF selection.
Methods: The Defence and Civil Institute of Environmental Medicine (DCIEM) dive tables and the United States Navy (USN) air decompression tables are considered acceptably safe by the Belgian Navy Diving Unit. The decompression model used in the Shearwater Perdix (Bühlmann ZH-L16C algorithm with GF) was programmed in Python. Using a sequential search of the parameter space, the GF settings were optimised to produce decompression schedules as close as possible to those prescribed by the USN and DCIEM tables.
Results: All reference profiles are approached when GF_{LO} is kept equal to 100 and only GF_{HI} is reduced to a minimum of 75 to prolong shallower stop times. Using the Perdix default settings (GF_{LO} = 30 and GF_{HI} = 70) yields deeper initial stops, leading to increased supersaturation of the 'slower' tissues, which potentially leads to an increased DCS risk. However, Perdix software does not currently allow for the selection of our calculated optimal settings (by convention $GF_{LO} < GF_{HI}$). A sub-optimal solution would be a symmetrical GF setting between 75/75 and 95/95.
Conclusions: For non-repetitive air dives, the optimal GF setting is GF_{LO} 100, with only the GF_{HI} parameter lowered to increase safety. No evidence was found that using the default GF setting (30/70) would lead to a safer decompression for air dives as deep as 60 metres of seawater; rather the opposite. Belgian Navy divers have been advised against using the default GF settings of the Shearwater Perdix dive computer and instead adopt symmetrical GF settings which is currently the optimal achievable approach considering the software constraints.

Introduction

Breathing compressed air at depth during a dive leads to the diffusion of the inert gas in the breathing gas, nitrogen, into the different tissues of the body. This process is driven by the 'inert gas pressure gradient'; the difference between the pressure of the inspired inert gas in the lungs and the inert gas tension in blood and tissues. The process is reversed when the diver ascends; inert gas will be washed out from the tissues as the inert gas tension in the tissues and blood now exceeds the inert gas pressure in the lungs. When the ambient pressure is reduced, the sum of all metabolic and inert gas tensions in the tissues can be larger than the ambient pressure and the tissues become supersaturated. This supersaturation is relieved either by inert gas elimination through diffusion from the tissues into the blood and subsequently to the alveoli to be expired, or, if the decompression is sufficiently large and rapid, by the formation and growth of inert gas bubbles in tissues and/or blood, which may lead to decompression Sickness (DCS).[1] The reduction in ambient pressure, and the resulting inert gas supersaturation, must be carefully controlled to allow sufficient washout of inert gas and to minimise the formation of bubbles. Computing a safe reduction in ambient pressure and a safe ascent level is the main purpose of a dive computer.

In 2018, the Belgian Defence introduced a commercial dive computer, the Shearwater Perdix™, for use by its military divers, replacing the end-of-life Cochran EMC-20H™ dive computer. Initial experience indicated several operational constraints. Using the Perdix dive computer with its default gradient factors (GFs) resulted in a significant reduction of the no-decompression limits (NDL) compared to earlier practice using the Cochran computer, yielding either shorter usable work time underwater, or the introduction of mandatory decompression stops. For 'decompression dives', substantial longer required decompression times were observed. Therefore, the main purpose of this research

was to provide recommendations to increase usable work time under water while maintaining safety, and to provide guidelines for GF selection during both no-decompression and decompression diving.

Methods

The basic decompression algorithm used in the Shearwater Perdix™ dive computer is a gas content model, the Bühlmann ZH-L16C model, with the use of GF[2] to modify the original equations.[3] The 'C' version of the ZH-L16 model was developed specifically for use in computer algorithms and dive computers. In this model, the human body is represented by 16 theoretical tissue compartments and the model assumes that inert gas exchange occurs at an exponential rate, both during inert gas uptake and elimination.[4] If the inspired inert gas pressure and the exposure time are known, the equalisation of pressure is calculated by means of different half-time values for different tissue compartment. Each tissue is considered to have a different maximum permissible inert gas tension, the 'M-value', as a function of the ambient pressure, which is defined by two parameters *a* and *b*:

$$P_{T,N2,tol} = \left(\frac{P_{amb}}{b}\right) + a \quad (1)$$

Inversely, it is possible to calculate the maximum tolerated ambient pressure, or safe ascent depth, for each of the 16 tissue compartments based on the prevailing inert gas tension:

$$P_{amb,tol} = \left(P_{T,N2} - a\right) \times b \quad (2)$$

The highest tolerated ambient pressure, or safe ascent depth, defines the shallowest depth to which a diver can ascend, and determines the depth of the decompression stop (a multiple of 3 m by convention). Ascending beyond this decompression 'ceiling' would violate the maximum permissible inert gas tension limit. The diver then waits at this decompression stop while inert gas is progressively eliminated from the body until the inert gas tension in all tissue compartments has decreased sufficiently to allow ascent to the next decompression stop.

A different approach to decompression, embodied in bubble decompression algorithms, suggest that decompression safety might be improved by adding stops at greater depths than those calculated with the gas content models. In essence, this principle can be mimicked by modifying the original M-values and thus forcing a gas content model to impose deeper stops. Baker proposed the use of GFs in the Bühlmann ZH-L16C model to add a margin of safety by lowering the allowed tissue compartment overpressure.[2] Gradient factors are expressed as a decimal fraction or a percentage of the difference between the ambient pressure and the original M-value. They modify the decompression profiles by deviating from the original, experimentally validated M-values of the Bühlmann ZH-L16 model:

$$P_{T,N2,tol,GF} = P_{T,N2,tol} \times GF + (1 - GF) \times P_{amb} \quad (3)$$

As presented in Figure 1, the GF setting consists of a set of two parameters, the 'low' setting (GF_{LO}) and the 'high' setting (GF_{HI}). Although different notations for GFs can be used (e.g., 0.3/0.7, 30%/70% or 30/70), the most common notation is GF_{LO}/GF_{HI}, e.g., 30/70, which will be used here. The actual applicable GF at a specific stop depth ($D_{stop,current}$) depends on the ambient pressure and a linear change from GF_{LO} at the first stop depth ($D_{stop,first}$) to GF_{HI} when surfacing ($D_{stop,final}$ = 0 msw):

$$GF = \left[\frac{GF_{HI} - GF_{LO}}{D_{stop,final} - D_{stop,first}}\right] * D_{stop,current} + GF_{HI} \quad (4)$$

Changing the GF results in a modified decompression profile; GF_{LO} mainly controls the depth of the first decompression stop, while lowering GF_{HI} results in longer decompression times at shallower stops. In the Shearwater Perdix™, the default GF is 30/70, with three pre-set alternatives (Firmware v84/BT 10: 45/95, 40/85, and 35/75), or the user may set custom GF with the constraint that $GF_{LO} < GF_{HI}$.[3]

In contrast to the original ZH-L16 model, the use of GF in itself is not directly linked to experimentally validated decompression profiles. In order to develop such a relation, a method was developed to map experimentally validated profiles onto the ZH-L16C deterministic gas content model. Both the air decompression tables in the US Navy Diving Manual (Version 6) and the Defence and Civil Institute of Environmental Medicine (DCIEM) dive tables are extensively experimentally validated and considered by the Belgian Navy as an acceptable standard for safe decompression in terms of no-decompression limits, stop depths and stop times. Hence, these tables were used in the current study as the reference decompression profiles. The parameters of our deterministic overpressure model consist of the half-time values, the original M-values and the GF which modify these limits. The half-time values and the two parameters *a* and *b* defining the original M-values were kept fixed in the current study and only the GF were modified. The objective was then to find the values of the GF that enables the deterministic ZH-L16C model to produce decompression schedules that are as close as possible to those prescribed by the US Navy and the DCIEM tables.

This study considered air dives up to a depth of 60 metres of seawater (msw), and no-decompression diving and decompression diving were considered separately. The decompression diving segment was divided into two sections, i.e., the normal air diving limit and the 'exceptional exposure' dives.[5] Gradient factor selection guidelines were

Figure 1

Left panel: the blue line presents the combined inert gas tension curve and the dive profile (ambient pressure) for a single tissue compartment during a hypothetical dive to 30 msw (4 bar). Because of the maximum permissible tissue tension (M-value line), a decompression stop is included at 3 msw (1.3 bar ambient pressure). Right panel: a gradient factor (GF) is a fraction of the difference between the ambient pressure lines and the M-value line. The two parameters, GF_{LO} and GF_{HI}, modify the original M-value line, thereby changing the decompression profile

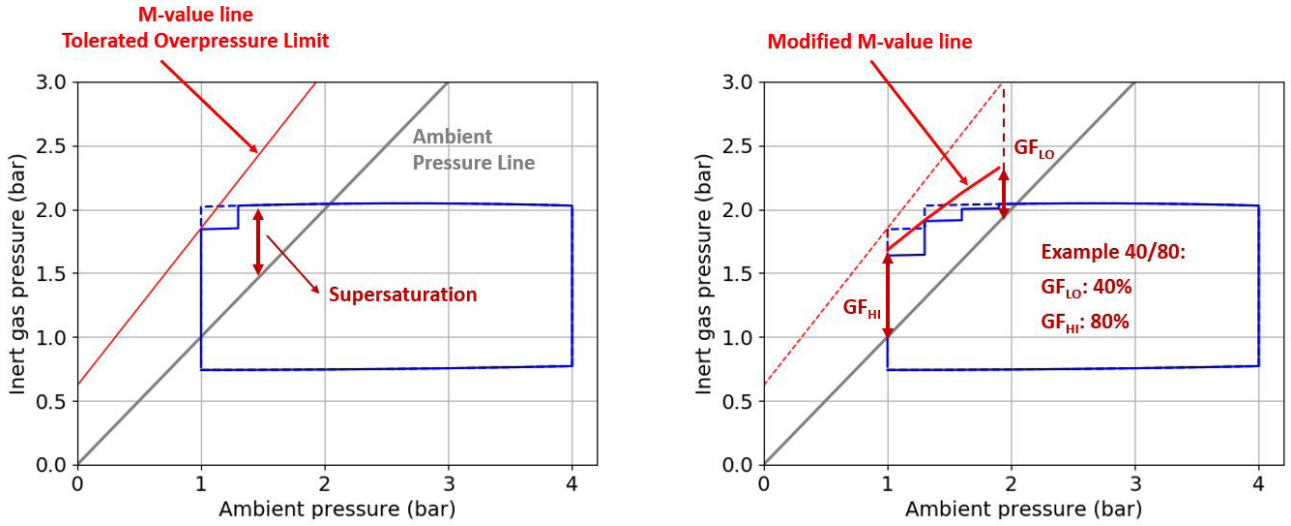


Figure 2

Sequential search of the gradient factor (GF) parameter space; the optimal solution is the GF setting which results in the minimum integrated difference between the reference profile, e.g., a Thalmann or DCIEM profile, and the decompression profile for a particular GF setting

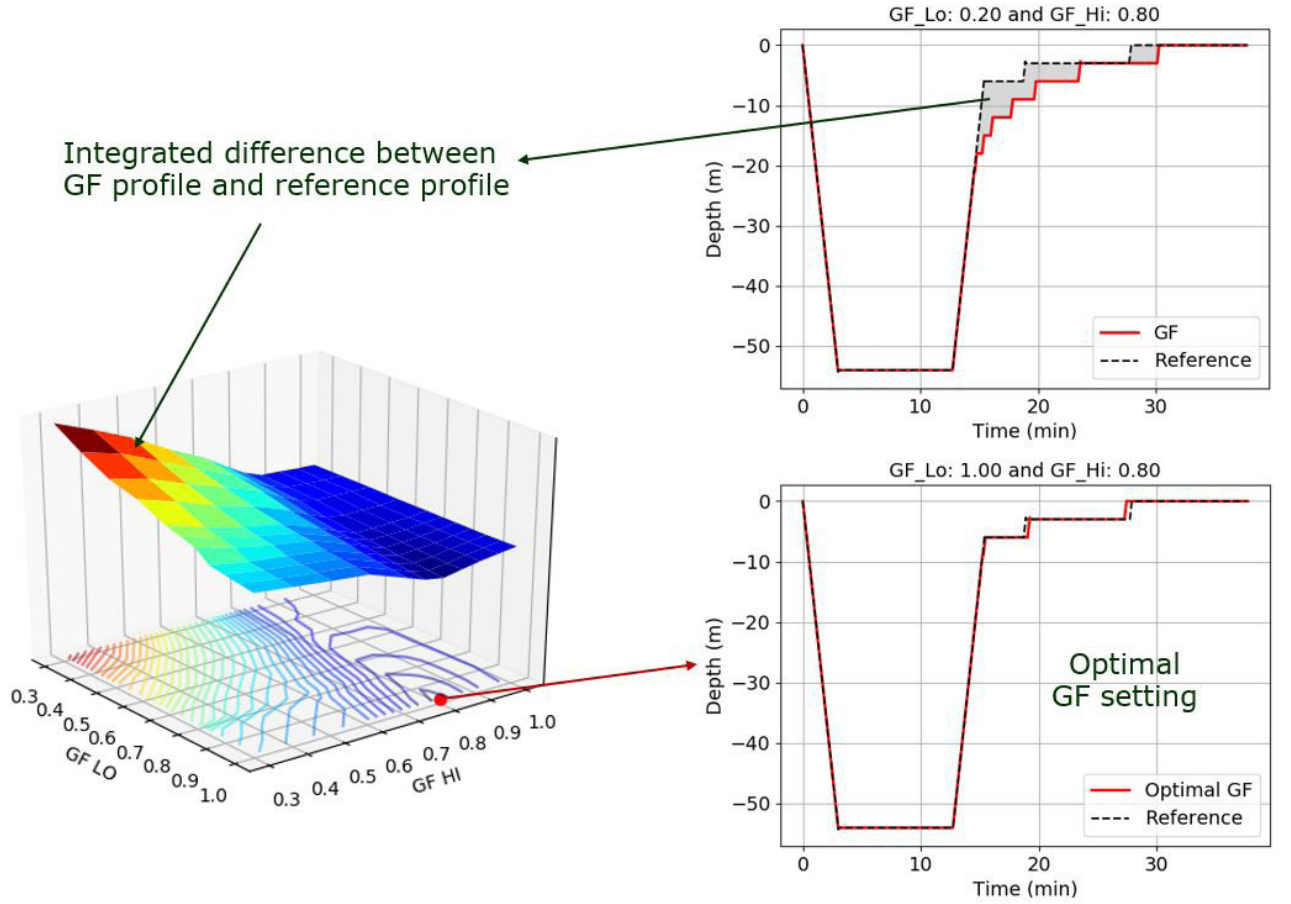
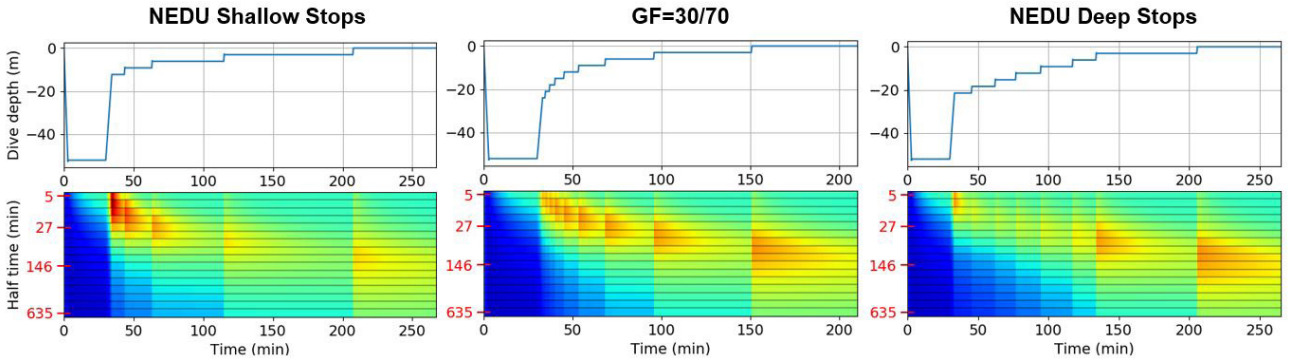


Figure 3

Comparison of three different decompressions for an air dive of 30 min to a depth of 52 msw: the NEDU shallow-stops schedule, the deep-stops schedule and a schedule using gradient factors (GF) 30/70. The upper panel presents the resulting dive profiles. The lower panel presents the evolution of the tissue ratio ($P_{T,N2}/P_{amb}$) for each of the 16 compartments of the Bühlmann ZH-L16C model (yellow-orange-red indicates a supersaturation, with red being the highest encountered supersaturation).



provided both for maximising work time under water without any decompression obligation, and for longer and deeper decompression dives, with the aim of approaching the familiar US Navy and DCIEM decompression procedures.

NO DECOMPRESSION LIMITS

The no-decompression limits (NDLs) were calculated using a two-step approach routine, based on an algorithm from the Thalmann algorithm decompression table generation software.[6] The reported NDLs included the descent time to the dive depth and were computed to the nearest second and assumed an ascent rate of 10 msw·min^{-1}.

DECOMPRESSION DIVING

The Bühlmann ZH-L16C algorithm was programmed in Python and implemented as a real-time algorithm, similar to the real-time algorithm as described by Thalmann.[7] The algorithm updates the inert gas tension in the 16 tissue compartments every two seconds and all calculations are based on the current depth and breathing gas. The algorithm then compares these tissue tensions with the ascent criteria, which are the maximum permissible tissue (inert gas) tensions at each of the 3 msw incremental stops depths, as calculated using Equations 1, 3 and 4. The algorithm determines the shallowest stop depth at which none of the current tissue tensions will be greater than their respective M-value. This depth is the safe ascent depth. The dive profile ascends to this decompression stop depth and waits for the safe ascent depth to decrement to the next 3 msw shallower stop until surfacing.[7] By using such a real-time algorithm, the inert gas dynamics during ascent (either continuing uptake or release of inert gas) is taken into account, in contrast to assuming an instantaneous ascent to the stop depth. The output of this real-time algorithm, i.e., the stop times and depths, was compared with and validated against publicly available data, including Bühlmann (2002)[4] and Thalmann (1984),[7] and the commercial available MultiDeco V4.19 software.

In the next step, this validated algorithm was used to determine which GF result in a schedule that approaches the reference decompression profiles. The GF optimisation was done using a sequential search of the parameter space, with the cost function being the integrated difference between the decompression profile for a particular GF setting and the reference decompression profile (Figure 2). The integrated difference was calculated starting at the beginning of the ascent and ended when surfacing. The time increment was identical to the two second time step of the algorithm. No additional constraints, e.g., the first stop depth, were incorporated. All possible combinations of GF_{LO} and GF_{HI} between 0.3 and 1.0, with increments of 0.05, were tested, and the GF setting which resulted in the minimal integrated difference between the resulting decompression profile and the reference profile was selected as the optimal solution. Afterwards, all optimal solutions were visually inspected to confirm the goodness-of-fit, and no manual corrections were required.

First, the GF settings were investigated for a single dive of 30 minutes to a depth of 52 msw. Two decompression schedules for this particular dive have been extensively tested.[8] These schedules both had 174 minutes of decompression time but differed by having either traditional shallow stops (resulting in 3 DCS in 192 man dives) or deep stops (11 DCS in 198 man dives and higher VGE grades) (Figure 3 left and right panels). The large size and clear outcome of this trial was considered a strong reference case for optimisation. Figure 3 (lower panels) presents the calculated tissue ratio, i.e., $P_{T,N2}/P_{amb}$, for the 16 compartments of the Bühlmann model for both decompression schedules. An increased (calculated) supersaturation of the slower tissues is currently the most plausible theoretical cause for the increased DCS incidence observed using the deep-stops schedule.[8] In the current study, the optimal GF was calculated to approach the shallow-stops profile and the standard GF setting of the Shearwater Perdix™ (30/70) was investigated in terms of supersaturation and compared to the two decompression schedules (Figure 3 centre panel).

Finally, the GF settings for many other different air decompression dives up to a depth of 60 msw were investigated by using the DCIEM decompression tables as reference dives.[7]

SHEARWATER PERDIX™ SOFTWARE INPUT RESTRICTIONS

The current software inside the Shearwater Perdix™ does not allow the selection of just any GF_{LO} and GF_{HI} combination. The following three embedded rules in the Perdix software enforce input restrictions on the GF setting:[9]

1. GF_{LO} must be less than or equal to GF_{HI}
2. GF_{HI} must be greater than 30
3. GF_{LO} must be greater than 10

Therefore, the optimisation of GF to approach the reference decompression profiles was done twice: once with and once without using the Shearwater Perdix™ software constraints on GF selection.

SIMULATION PARAMETERS

All DCIEM dive profiles were calculated using 18 msw·min^{-1} as the descent and ascent speed. For dives from the US Navy Manual, the descent speed and ascent speed were set to 18 msw·min^{-1} and 9 msw·min^{-1} respectively. The last decompression stop was performed at 3 msw. A water density of 1,019 kg·m^{3}, a pressure increase of 1 bar per 10 msw, and an atmospheric pressure of 1.01325 bar at surface were used to calculate the ambient water pressure at depth.

Results

NO DECOMPRESSION LIMITS

The no-decompression limits are determined by the GF_{HI} setting. Figure 4 shows the NDL as a function of the dive depth and for different GF_{HI} settings, together with the DCIEM NDL for comparison. These DCIEM NDLs were approached by the Shearwater Perdix™ when the GF_{HI} was set to 90. As the default GF_{Hi} setting of the Perdix computer is 70, this reduces the NDL considerably, and can be seen to be responsible for the operational constraints reported by the Belgian Navy divers. Considering the DCIEM NDL as a safe standard, a GF_{HI} of 70 is too conservative.

DECOMPRESSION DIVING

Figure 5 (left panel) presents the Bühlmann ZH-L16C decompression profile with the default Perdix GF selection, i.e., 30/70, for a single compressed-air dive of 30 min to a depth of 52 msw, compared to the NEDU study shallow-stops and deep-stops profiles. The total decompression was shorter by more than 50 min with respect to the NEDU profiles, and the decompression schedule included deeper stops during the initial phase of the ascent, similar to the NEDU deep-stops profile. Figure 3 (lower panel) presents the evolution of the tissue ratio for each of the 16 Bühlmann compartments (with half-times ranging from 5 min to 635 min) using the 30/70 setting. As a result of the deeper stops during the initial phase of the ascent, the supersaturation was reduced for the faster tissue compartments at the beginning of the ascent but increased for slower tissue compartments later on in the decompression and after surfacing, similar to the NEDU deep stops profile.

Figure 4

No decompression limit (NDL) as a function of the dive depth for different GF_{HI} settings in the range between 70 and 100; the black dashed line represents the Defence and Civil Institute of Environmental Medicine (DCIEM) NDL, which is approached when the GF_{HI} is set to 90

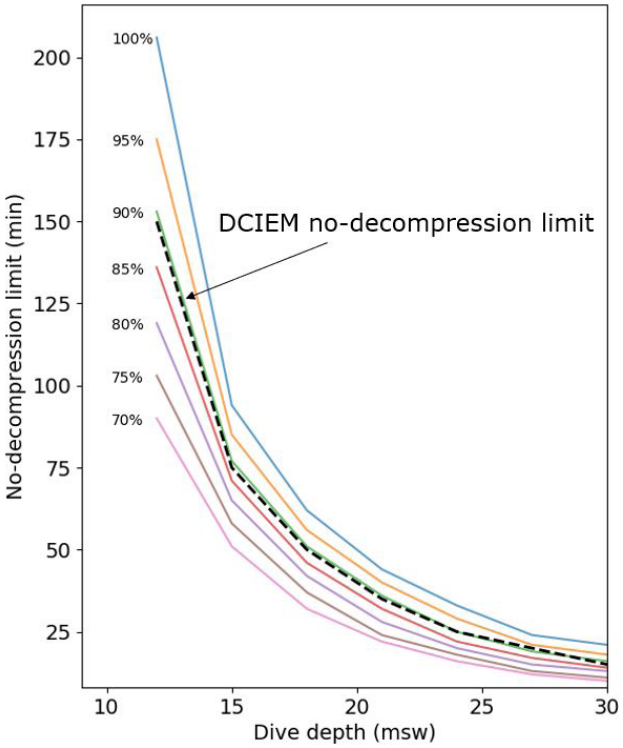


Figure 5 (right panel) illustrates the GF with which a Bühlmann decompression schedule approaches best the NEDU shallow-stops schedule: GF_{LO} and GF_{HI} were set to 100 and 40, respectively. This GF_{LO} ensures that the first stop depth is as shallow as possible, while a lower GF_{HI} increases the stop times. Note that this setting is not possible with the software restrictions in the Perdix dive computer.

Figure 6 shows the GF settings that best approached the air decompression dives of the DCIEM table: GF_{LO} was kept equal to 100 and only GF_{HI} was decreased. This is in line with the previous optimization result for the NEDU reference profile. The minimum GF_{HI} of 75 was obtained near the NDL boundary. Again, the current software inside the Shearwater Perdix dive computer does not allow for the selection of these 'optimal' settings. Taking into account the constraints, 'sub-optimal settings' were

Figure 5

Left panel: The ZH-L16C GF 30/70 decompression profile, compared to the shallow-stops and deep-stops schedules used in the NEDU study; using gradient factors (GF) 30/70 results in a shorter total decompression time and in deeper decompression stops during the initial phase of the ascent. Right panel: The optimal ZH-L16C GF 100/40 decompression profile approaches the shallow-stops schedule. A GF_{LO} of 100 ensures that the first stop depth is as shallow as possible, and the reduced GF_{HI} prolongs the decompression time. N2 – nitrogen

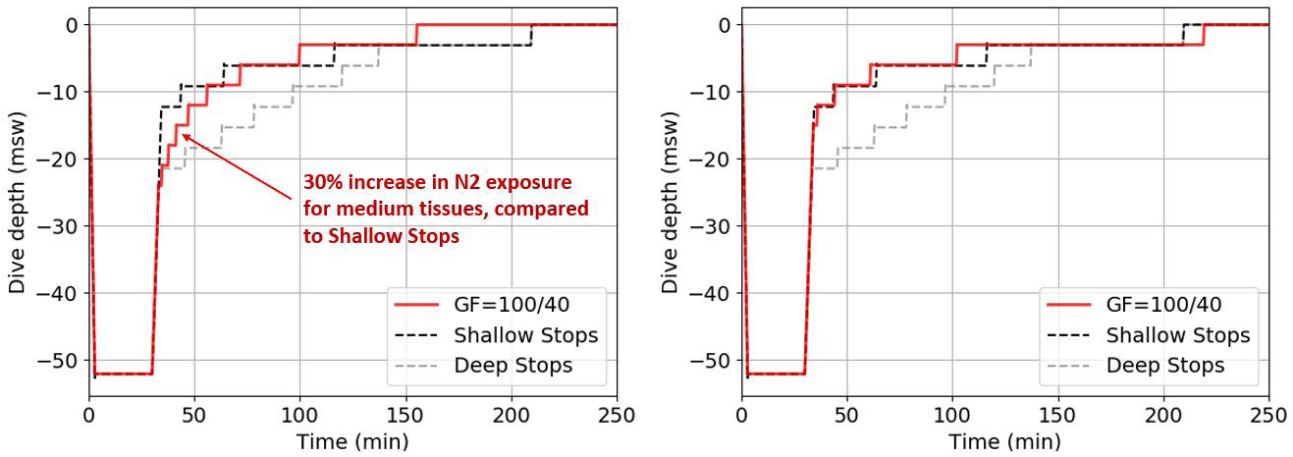


Figure 6

Optimal gradient factor (GF) settings to approach air decompression dives of the DCIEM table, for several selected dive depth/time combinations; the grey area represents the no-decompression dive range; the blue line divides the decompression dive segment into normal air dives and exceptional exposure dives. No air diving is allowed in the red area

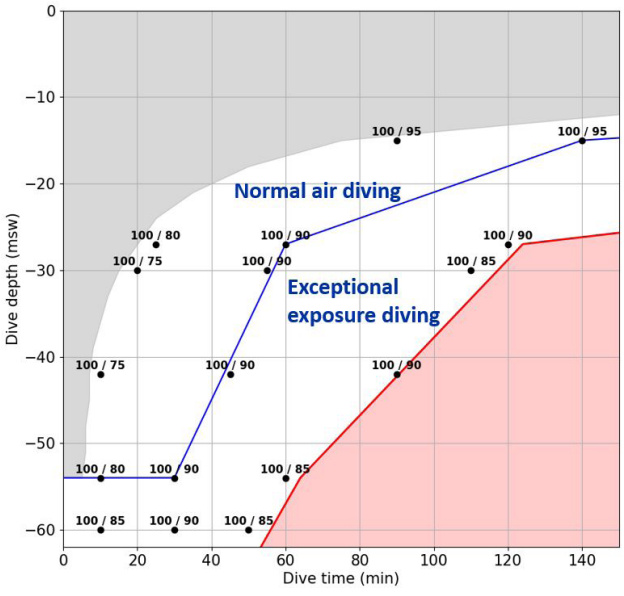
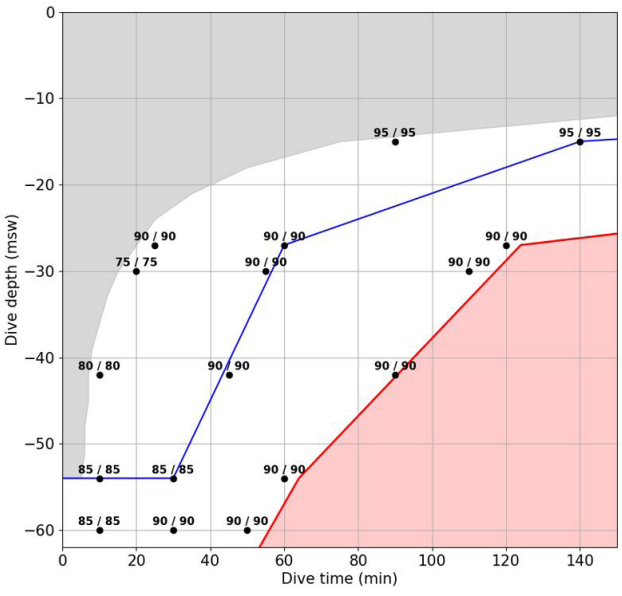


Figure 7

Sub-optimal GF settings, compliant with the GF input restriction in the Shearwater Perdix™ software, to approach air decompression dives of the Defence and Civil Institute of Environmental Medicine (DCIEM) table, for the same dive depth/time combinations as in Figure 6



calculated, compliant with the Perdix software constraints (Figure 7). For each of the dives, the sub-optimal solution was a symmetrical GF. Overall, a symmetric GF of 90/90 was the best suboptimal setting to approach the DCIEM tables, except for short bottom times where the lowest GF found amongst all the dives was 75/75. Using a symmetrical GF setting of 90/90 for all dives within the normal air dive boundary resulted in a maximum difference of five minutes of decompression time at a shallow stop depth, compared to the DCIEM tables.

Discussion

The efficacy of GFs is only supported by anecdotes[10] and to our knowledge, no large-scale experimental trial has ever been undertaken to examine the DCS incidence for

decompression with and without the use of GFs. Modifying ZH-L16 with GFs represents an extrapolation beyond the original ZH-L16 experimental validation data that may not be reliable in all circumstances since the actual biophysical processes can be altered to an unknown extent, leading to a different probability of DCS. Therefore, in the current research, GFs are considered just as two mathematical parameters to modify the validated M-values and to change the resulting decompression profile, rather than in terms of adding conservatism per se: a 'deviation' factor instead of a 'conservatism' factor.

Essential in the current air decompression diving analysis is the unchanged GF_{LO} parameter and the decrease of the GF_{HI} parameter. Our optimisation analysis indicates that it is never required to use a GF_{LO} as low as the Perdix default setting of 30 to approach the reference decompression profiles. Also, using the Bühlmann ZH-L16C model with the Perdix default GF settings (30/70) the calculated tissue supersaturations closely resemble the NEDU deep-stops schedule, suggesting a potential increased DCS risk for these GF settings. Although no actual gas exchange measurements have been made, an increased supersaturation of the slower tissues is currently the most plausible cause for the increased DCS incidence when using the deep-stops schedule.[8] This would mean that the default GF settings do not only introduce operational constraints but can (according to this comparison) potentially lead to an increased DCS risk, which is the exact opposite of the desired effect.

In 2017, DAN Europe published an analysis of 320 dives included in the DAN Europe Diving Safety Laboratory database (DAN DSL), having resulted in DCS symptoms.[11] No information about the actual GF setting in the dive computers was available and instead, max GFs were calculated according to the Bühlmann ZH-L16C model: inert gas tensions were calculated for the 16 compartments and represented as a fraction of the M-value, which was then considered as the instantaneous GF along the ascent. Then, the maximum GF during the ascent was labelled as the corresponding max GF, i.e., the 'GF_{HI} value', for that particular dive. They concluded that 73.3% of all DCS cases had a GF_{HI} between 70 and 90, and that only 2.5% had a GF > 100. However, in the light of the current analysis, it is argued that this does not show the full picture for the DCS dives from the DAN DSL as no information is presented about the lowest GF along the ascent, i.e., a 30/70 setting appears as a 'GF_{HI} 70' value in the data, as does a 70/70 setting. Therefore, referring to Figure 3, the DAN analysis focusses on the overpressure in the fast tissues, and does not cover the critical overpressure in the slower tissues.

In 2018, Fraedrich evaluated several commercial dive computer algorithms, including the Bühlmann ZH-L16C algorithm. The ZH-L16C total decompression time (TDT) was compared to TDT from a model based on a large database of air decompression schedule validation dives, specifying the DCS incidence risk as a function of the bottom time and TDT.[12] However, this approach does not seem to be universally applicable: indeed, the NEDU study presents a clear example of two decompression profiles, with identical bottom time and TDT, but with vastly different DCS incidence. Moreover, another study showed that the use of multiple deep stops and longer ascents (increased TDT for an identical bottom time) increased bubble generation.[13] Therefore, we chose to use the complete decompression profile as a test for comparison, rather than the TDT alone.

Similar to our results, Fraedrich found a GF_{HI} equal or lower than 70 to be required to get a TDT comparable to the validated US Navy dive profiles.[12] We found that a GF_{HI} of 40 approaches the NEDU shallow-stops decompression profile, while GF_{HI} settings of 75–90 are required to approach the DCIEM air decompression dives (that used a different experimental dataset for validation). The NEDU shallow stops schedule, based on the Thalmann algorithm with the VVal-18 parameter set, had a 1.5% incidence of DCS, which is very low for an exceptional exposure dive. It has been acknowledged that the VVAL-18 parameter set may result in inordinately long decompressions,[14] and a modified version, the VVAL-18M parameter set, was used for the Air Decompression Tables as found in the US Navy Diving Manual (Version 6). This shorter (VVAL-18M) decompression for the NEDU reference dive depth and bottom time is approached with a GF_{HI} value of 80, which is in the same GF_{HI} range as for the DCIEM air decompression dives. Also, Fraedrich found a GF_{LO} equal or higher than 55 to be required to have the first stop shallower than the US Navy deep stops schedules.[12] We found that the optimal solution for the NEDU shallow stops schedule and all optimal solutions for DCIEM schedules keeps the GF_{LO} parameter fixed to 100 while only the GF_{HI} parameter is lowered to increase stop times at shallower stop depths.

Conclusions

No evidence was found that the default 30/70 setting, and the corresponding deeper stop depths, would lead to a safer decompression profile for non-repetitive air dives up to a depth of 60 msw during military training and operational dives. Our advice to the Belgian Navy divers has been, while using the GFs as they are currently implemented in the software, including the input constraints, to use a symmetric setting of 90/90 and to symmetrically decrease it when a higher safety margin is deemed appropriate. In any case, it is proposed to avoid decreasing GF below 75/75 to not induce deeper stops and keep the resulting decompression profiles not too far from the experimentally tested profiles, as that would induce unknown biophysiological changes not accounted for by the decompression model.

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Review articles

Diving with psychotropic medication: review of the literature and clinical considerations

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Fitness-to-dive; Medications; Mental health; Pharmacology; Psychiatry; Scuba

Abstract

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This review discusses the safety concerns associated with diving while using psychotropic medication and the limited literature available on the topic. Despite the risks, some divers continue to dive while taking these medications, and their reasons for doing so are unclear. The exact mechanisms of action of these drugs in hyperbaric environments are poorly understood. While current standards and advice for fitness-to-dive assessments are based on limited evidence and expert opinion, developing evidence-based strategies could improve patient care and optimise diving safety. This review appraises relevant literature in diving medicine and provides clinical perspectives for diving physicians conducting fitness-to-dive assessments on patients using psychotropic medication.

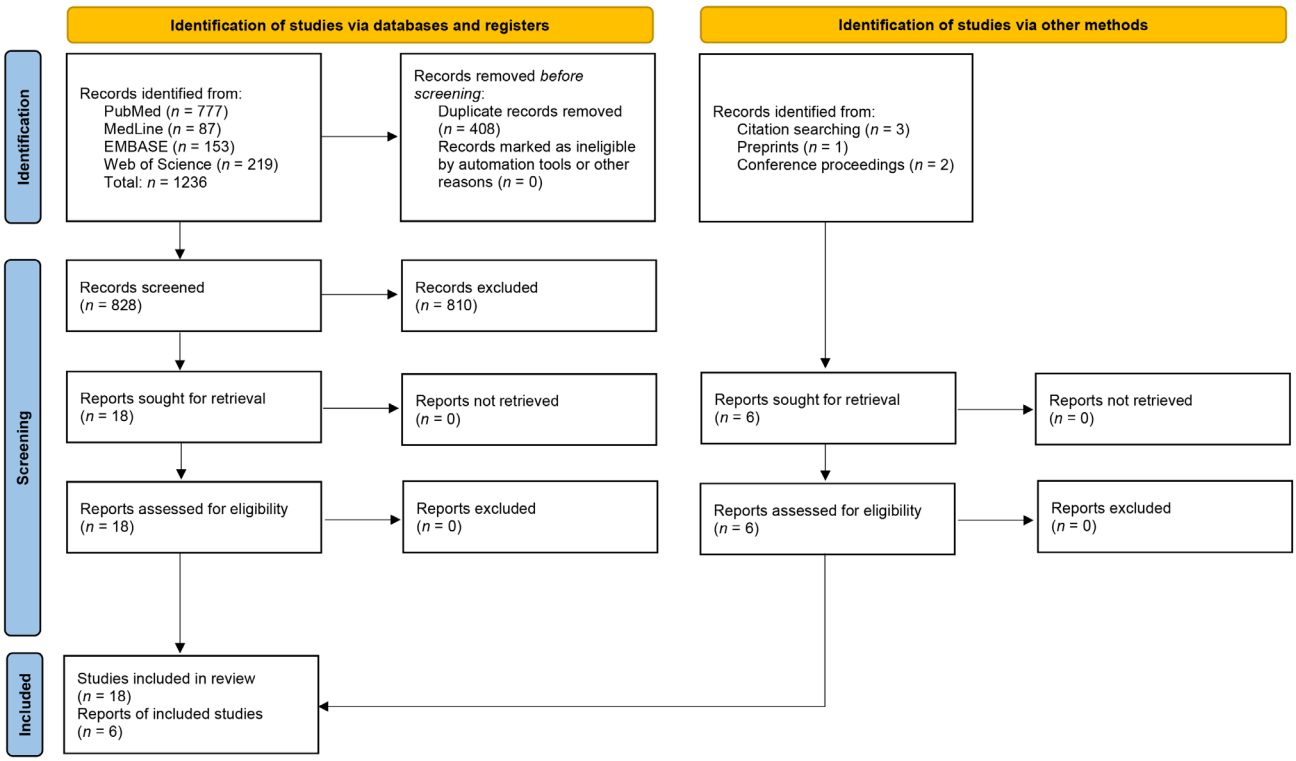
Introduction

Despite the generally healthy status of the diving community, diving with comorbidities or medications seems to be increasing.[1,2] This trend has raised questions regarding the safety of diving under these conditions and underlines the importance of standards and policies for preventing diving accidents. Despite the risks associated with diving under conditions sometimes considered incompatible with diving, such as the use of psychotropic medications, studies have shown that some divers continue to do so.[3-6] Interestingly, divers do not always consider diving with psychotropic medication a safety risk.[7] The reasons for this behaviour are unclear and it is uncertain if these divers have refrained to seek advice from a physician or were given incorrect advice. Diving under medically hazardous conditions might increase the risk of diving accidents, but evidence remains equivocal.[5,8] More specifically, while many reports on diving accidents are published worldwide, the link between psychotropic medication and the outcome of an incident might not be picked up in an accident investigation due to the complex and multifactorial pathway in the chain of events leading up to an accident.[1,8]

Although numerous studies have reported the potential dangers associated with cardiovascular disease, the hazards stemming from the utilisation of psychotropic medication have received comparatively limited attention.[4,9–11] The findings of two surveys indicate that the utilisation of psychotropic medications among active divers is minimal and largely restricted to antidepressants, probably due to reporting bias.[5,12] This stands in contrast to the increasing use of psychotropic medication in the general population.[13,14] In addition, the exact mechanisms of action of psychotropic drugs are poorly understood. This holds especially true in hyperbaric medicine, and available data are mostly derived from animal studies.[12,15,16]

Recently, a large diving organisation launched a campaign to promote the positive effects of diving on mental wellbeing. This may attract more divers struggling with mental health issues and, consequently, users of psychotropic drugs.[17] Extensive fitness-to-dive assessments and guidance from a specialised physician are the cornerstones to prevent diving accidents. However, current standards and advice are mostly based on limited evidence, expert opinion, and theoretical concepts.[12] In addition, many standards in the field are

Figure 1
PRISMA diagram for the search and study selection



relatively restrictive regarding the use of psychotropic drugs in relation to diving, which might be at least partially explained by the fact that diving physicians are not always familiar with psychotropic medications. This may give rise to policies that are excessively permissive or restrictive, each with its unique set of attendant risks and cost. Developing evidence-based strategies for optimising diving safety and health could assist physicians in providing state-of-the art patient care.

Therefore, we reviewed the current literature to provide clinical guidance regarding the use of psychotropic medications during fitness-to-dive assessments for diving physicians. In addition, we reviewed relevant literature in psychiatry and share our clinical experience in fitness-to-dive assessments.

Methods

The protocol for objectives, literature search strategies, inclusion and exclusion criteria and outcome measurements were prepared a priori, according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement.[18] The objective of this search was to identify studies investigating the effects of the hyperbaric environment on the pharmacodynamic and pharmacokinetic characteristics of psychotropic medications and the associated risks of diving disorders when used while diving. Electronic database searches were conducted in PubMed, Medline, Embase Science Citation Index Expanded, and the Web of Science up to December 31, 2022. The search was conducted using the keywords "(scuba) diving", "hyperbaric", "medication", "drugs", and "psychiatry", and their synonyms. In case of limited results, fewer keywords were used to avoid missing relevant publications. Snowballing was performed to identify additional studies. Hits were screened on title and abstract. Studies were included if they were published in peer-reviewed journals and covered aspects of psychotropic medication and diving. We focused on five types of psychotropic medications: antipsychotics, antidepressants, sedatives, psychostimulants, and mood stabilisers. Two authors (BQ and TW) independently selected the studies and extracted the data. Any inconsistencies were discussed until consensus was reached. The evaluation of study quality was conducted using appropriate instruments tailored to the specific study type when applicable.

Results

A total of 1,236 studies were initially identified from the four electronic databases using the search strategy described above (Figure 1). After removing 408 duplicate records, 828 studies were screened for eligibility. After applying the inclusion and exclusion criteria, 18 studies were deemed relevant and included in the final analysis. An additional six studies were identified from the reference lists of the included studies, resulting in a total of 24 studies being included in the review. These are summarised in Table 1.

Table 1

Summary of studies included in this review; ADHD – attention deficit hyperactivity disorder; GABA – gamma-aminobutyric acid; NMDA – n-methyl-d-aspartate

Ref	Study type	Subject	Main outcome
19	Animals (mice)	Protection against nitrogen narcosis by lithium	Lithium prevented the nitrogen-narcosis-induced loss of righting response but significantly potentiated the pressure (depth) at which convulsions and tremors occurred
20	Animals (mice)	Protection against decompression sickness by fluoxetine	Platelets and red cells were significantly decreased after decompression in controls but not in the treated mice. Fluoxetine reduced circulating IL-6
2	Survey	Health status of divers	Better mental health in divers than in controls
21	Position paper	Diving by children	Clinical guidance on diving with ADHD in children
22	Animals (rats)	Risk factors associated with oxygen toxicity	Superoxide dismutase and catalase activity was reduced in the brains of rats exposed to hyperbaric hyperoxia. Glutathione peroxidase activity was reduced in the hippocampus
7	Survey (divers)	Usage of medication amongst divers	Fifteen percent of the divers used long-term medications when diving. 59.4% considered that they were putting themselves at risk by self-medicating
23	Animals (rats)	Effects of diazepam in hyperbaric conditions	Convulsions provoked by high helium pressure were prevented by diazepam and the symptoms of high-pressure nervous syndrome (HPNS) were reduced
12	Review	Effects of medication in hyperbaric conditions	No evidence of significant risks due to changes in pharmacologic mechanisms were revealed and most medication is not a contraindication to diving
3	Survey (divers)	Medication usage amongst divers	60% reported using non-prescribed medications; 1.2% reported psychiatric illnesses
5	Survey (divers)	Medication usage and effect on nitrogen narcosis	15.2% reported the use of psychiatric medications. No obvious effect on nitrogen narcosis
1	Survey (divers)	Medical conditions involved in diving accidents	Many divers have medical conditions and use medications
24	Review	Risk factors associated with oxygen toxicity	GABA and NMDA modulating drugs affect the risk of seizures related to oxygen toxicity
25	Position paper	Diving with antidepressants	Clinical guidance on diving with antidepressants
26	Position paper	Diving with ADHD	Clinical guidance on diving with ADHD medication.
27	Review	Diving with cardiovascular medications	A risk-based approach on diving with medications
15	Review	Effects of hyperbaric conditions on pharmacokinetics	Hyperbaric or hyperoxic exposure doesn't affect pharmacokinetics of drug elimination.
28	Case series	Effects of medication on oxygen toxicity	Antidepressants or tramadol might increase risk of central nervous system oxygen toxicity
11	Case series	Medication used in diving accidents	Several divers involved in accidents used (psychoactive) medications
6	Survey (divers)	Usage of recreational drugs amongst divers	Report on divers using illicit drugs, with a clear relation to depression or anxiety disorders
4	Survey (divers)	Diving with mental health issues	Similar incidence in mental health issues in divers compared to the general population and many of them are diving with psychoactive medications
10	Survey (divers)	Medication usage amongst divers	Less than 2% of divers use anti-epileptic, antidepressants, or antipsychotic medications
9	Survey(divers)	Medication usage in diving accidents	6.4% of divers reported the use of psychotropic medications
29	Animals (mice)	Protection of fluoxetine against nitrogen narcosis	Fluoxetine decreased decompression sickness severity and survival in mice
30	Review	Risks associated with oxygen toxicity	GABA and NMDA modulating drugs affect the risk of seizures related to oxygen toxicity

Discussion

To the best of our knowledge, this is the first structured literature review to investigate the effects of psychoactive medication on fitness-to-dive assessments. In the following section, we summarise our findings and share our clinical expertise for individuals who require psychotropic medication while diving. Following careful consideration of the available evidence and general considerations, we provide recommendations for the use of antipsychotics, antidepressants, sedatives, stimulants, and mood stabilisers.[31]

GENERAL CONSIDERATIONS

Psychotropic medications are widely prescribed, with their use on the rise.[14,32] While the present study does not aim to evaluate the validity of such prescribing practices, it is important to recognise that mental health complaints amongst divers can vary widely in severity. Furthermore, most psychotropic medications have multiple indications, making it overly simplistic to base dive medical advice solely on the drug used. We emphasise that the use of psychotropic medications is often underreported in fitness to dive assessments, and we urge diving physicians to take the time to explore this carefully.[4] When assessing a diver's use of psychotropic medications, it is essential to first consider the indication, as this may be a reason to temporarily restrict diving activity. While diving should not be seen as a treatment for psychiatric disorders, many divers report positive effects of diving on their mental wellbeing and its potential therapeutic benefits should be considered. Although severe adverse effects of psychotropic medications are possible, the majority of patients tolerate these drugs well and experience only mild side effects.[33] Side effects are especially common in the first few weeks of treatment and when multiple medications are used simultaneously. We advise colleagues who are unfamiliar with psychotropic medications to seek expert consultation when assessing fitness to dive, i.e., from a psychiatrist or clinical pharmacologist, to evaluate the possibly relevant side effects for diving. Finally, it should be noted that most psychotropic medications alter the activation threshold of nerve cells, and there is some evidence from animal studies in rats to suggest that these drugs may increase the risk of cerebral oxygen toxicity.[22,24] In a study of 4,357 hyperbaric oxygen treatments conducted in human subjects, all of whom were concurrently taking psychotropic medications (including antidepressants, sedatives, or narcotics), seven cases of seizures were reported.[28] Although other factors may have contributed to these cases, the association between neuromodulating agents and cerebral oxygen toxicity warrants further investigation.

When evaluating the fitness of a diver with mental health complaints, it is important to carefully consider the individual case rather than solely relying on the diagnosis or medication use. Factors such as therapy compliance and disease awareness should also be considered when determining a diver's ability to comply with diving restrictions and advice. Once a patient has stabilised and returned to normal daily activities, diving can be considered. However, when using multiple psychotropic medications, diving may be discouraged due to an increased risk of side effects. Lastly, given that side effects of psychotropic medications typically manifest within the initial weeks of initiation or dosage modification, a precautionary period of refraining from diving for a duration of up to three months may be advised. If side effects persist, diving should be approached with extreme caution. Moreover, as psychotropic medications could increase susceptibility to nitrogen narcosis or oxygen toxicity, a limitation on diving depth, e.g., 18 metres (60 feet), could be recommended to prevent these conditions.[30]

ANTIPSYCHOTICS

The use of antipsychotics is primarily indicated for psychotic disorders, acute mania, and severe agitation, although many off-label indications exist, and the registration of antipsychotics differs between the Food and Drug Administration (FDA) and the European Medicines Agency (EMA). Despite the often-used classification into first-, second-, and third-generation, antipsychotics are a heterogeneous drug class with diverse pharmacological and clinical properties. While the effectiveness of different antipsychotic medications is similar, with the exception of clozapine, their side effects vary.[34] Weight gain, sedation, drowsiness, and dry mouth are commonly reported side effects, particularly those with strong antihistaminergic and anticholinergic properties (such as quetiapine, olanzapine, clozapine and 'low-potency' first-generation antipsychotics).[35] Diving should be discouraged if the patient experiences drowsiness or sedation. Movement disorders, convulsions, orthostatic hypotension, and dysregulated blood glucose levels are also relevant side effects for sports diving.[36–38] Movement disorders, such as parkinsonism, acute dystonia and tardive dyskinesia, are mainly attributed to potent dopamine D_2-antagonism, a characteristic of several antipsychotics such as haloperidol, risperidone, and paliperidone. Partial D_2-agonists, such as aripiprazole, are particularly associated with the development of akathisia, although any antipsychotic medication has the potential to cause this side effect. To detect (subtle) movement disorders, a thorough neurological examination is required. Although hyperglycemia is more common in patients undergoing antipsychotic treatment than in the general population, hypoglycemia is one of the idiosyncratic potentially life-threatening adverse effects of antipsychotics due to increased insulin secretion. In cases with a history of uncontrolled hypoglycemia due to side effects or diet, disqualification for diving should be considered. The International Council on Alcohol, Drugs and Traffic Safety (ICADTS) has published considerations for medication use in driving that may support clinicians in assessing fitness to dive.[39]

Although seizures are rare, they can be fatal when they occur while diving. Therefore, the use of clozapine, a frequently prescribed antipsychotic with a relatively high risk of seizures, is not compatible with scuba diving. Although older antipsychotics such as chlorpromazine, promazine, thioridazine, and haloperidol have been associated with an elevated risk of seizures, evidence regarding the potential seizure risk of other antipsychotic medications is limited.[36] While most antipsychotic medications are associated with only modest QT-interval prolongation, aripiprazole, brexpiprazole, cariprazine, and lurasidone are typically considered to pose a relatively low risk in this regard. Conversely, sertindole is regarded as particularly high-risk, and its use should ideally be avoided due to its association with a heightened risk of arrhythmias. Other cardiovascular risk factors, such as smoking or hypertension, probably increase morbidity and mortality risks more in diving.[27,40] Diving with a low dose of antipsychotic medication in the absence of side effects or other cardiovascular risk factors is probably safe.

ANTIDEPRESSANTS

Antidepressants are prescribed primarily for major depression and anxiety disorders, but are also used for post-traumatic stress disorder (PTSD), obsessive-compulsive disorder (OCD), somatoform disorders, sleeping disorders, and chronic (neuropathic) pain syndromes as well as off-label indications.[41] There are several classes of antidepressants, including tricyclic antidepressants (TCAs), serotonin reuptake inhibitors (SSRIs), serotonin-noradrenaline reuptake inhibitors (SNRIs), monoamine oxidase inhibitors (MAOIs) and others such as bupropion (a noradrenaline-dopamine reuptake inhibitor, NDRI), mirtazapine (a noradrenergic and specific serotonergic antidepressant, NaSSA), trazodone (a serotonin-antagonist and reuptake-inhibitor, SARI) and vortioxetine (a multi-modal antidepressant, MMA).

Tricyclic antidepressants commonly cause side effects due to their anticholinergic (dry mouth, blurred vision, constipation, urinary retention, and tachycardia), antihistaminergic (weight gain, sedation, and drowsiness) and anti-adrenergic properties (orthostatic hypotension and sedation).[42] Although SSRIs and SNRIs are generally better tolerated than TCAs due to their more selective pharmacological properties, they still have a similar incidence of side effects, albeit qualitatively different.[43–45] Common side effects of SSRI's, SNRI's and vortioxetine include headaches, gastrointestinal symptoms, insomnia, and sexual dysfunction. Additionally, they can increase bleeding risk, cause weight gain, somnolence, agitation, and hyponatremia, although these side effects are less common. An increased bleeding tendency has a theoretical risk of making any neurological decompression sickness (DCS) worse but is only clinically relevant in patients using warfarin or non-steroidal anti-inflammatories (NSAIDs) and in patients with a history of bleeding complications.[25]

In the context of diving, the most relevant side-effects of antidepressants include sedation, hypotension, and seizures. In particular, tertiary TCA's and mirtazapine, due to their very high affinity for the histamine H_1-receptor, are not recommended for diving as they belong to the most sedating antidepressants. Besides TCA's, virtually all antidepressants, including SSRIs and SNRIs can induce hypotension, although SNRIs can also cause hypertension due to their noradrenergic enhancing properties by inhibiting the noradrenaline transporter (NET). Irreversible MAOIs are associated with a high probability of severe and dose-dependent orthostatic hypotension, which makes them unsuitable for diving.[46,47]

All antidepressants lower the threshold for epileptic seizures, with possibly a higher risk for clomipramine and bupropion.[48] Although the absolute risk is low, it might increase with long-term use.[49] The risk of convulsions with bupropion is dose-dependent, particularly with doses exceeding 450 mg and in immediate-release formulations.[50] Consequently, due to its potential to lower the seizure threshold, bupropion should preferably be avoided for diving. On the other hand, two studies in mice suggest a possible neuroprotective effect of fluoxetine (50 $mg \cdot kg^{-1}$) when administered before hyperbaric exposure, resulting in lower decompression sickness (DCS) incidence and better neurological recovery.[20,29] Although one study shows that fluoxetine might have subtle anti-inflammatory effects, these are most likely clinically insignificant in the prevention or treatment of DCS.[51] During fitness-to-dive assessments, if antidepressants are required, SSRIs and SNRIs should be suggested over TCAs, mirtazapine and trazodone due to their lower incidence of sedation and drowsiness.[25] However, diving with SSRIs and SNRIs should be temporarily discontinued when the dosage is reduced or the medication is stopped due to the risk of withdrawal symptoms.

SEDATIVES

Sedatives, such as benzodiazepines and Z-drugs, are frequently used in the treatment of sleep and anxiety disorders in individuals with psychiatric illness. However, chronic usage of these drugs may result in dependency, cognitive impairment, and an increased risk of falls and accidents due to sedation, drowsiness, confusion, dizziness, and amnesia.[52] Most sedatives are classified as ICADTS category III, which prohibits their use when operating a vehicle.[39] Additionally, although animal studies suggest that diazepam may prevent high pressure nervous syndrome (HPNS), the sedative effects of this medication likely outweigh any potential benefits.[23] Therefore, diving while taking benzodiazepines or other Z-drugs is contraindicated. If these medications are used occasionally to treat sleep disorders, diving may be permitted after discontinuation for at least one week, depending on the specific drug's elimination half-life. In individuals with anxiety disorders, the underlying illness should be prioritised, and diving may be considered only after an appropriate medication-free

period has elapsed. It is important to note that discontinuing or abruptly reducing the dosage of these drugs in long-term usage may cause withdrawal effects. Consultation with an expert should be sought if discontinuation or dosage reduction is being considered.

PSYCHOSTIMULANTS

Stimulants are commonly used to treat attention deficit hyperactivity disorder (ADHD) and narcolepsy, among other off-label indications. A small study has suggested that ADHD is fairly common among commercial divers.[53] Psychostimulants include methylphenidate, amphetamines (such as mixed amphetamine salts, dexamfetamine and lisdexamfetamine) and modafinil. Also, non-stimulants are used in ADHD, such as the alpha-2 adrenergic agonists (clonidine and guanfacine), atomoxetine, and bupropion. The most common side effects of psychostimulants and atomoxetine are mild, such as insomnia, reduced appetite, headache, and palpitations. Among adults, treatment with psychostimulants has been linked to a modest increase on average in systolic blood pressure (2 mmHg) and heart rate (5.7 beats·min^{-1}).[54] Although these cardiovascular changes are not typically considered clinically significant in the short term, there are concerns regarding their long-term implications. However, observational studies have shown that the use of psychostimulants does not significantly increase the risk of cardiovascular events at the population level, or only to a limited extent.[55] It is important to note, however, that in individual patients, blood pressure and heart rate can increase substantially, with increases of approximately 10–25 mmHg and 15 or more beats·min^{-1} respectively.

Psychostimulants and bupropion may lower the convulsive threshold in patients with a prior history of seizures, and rarely in patients without a history of convulsions and no electroencephalogram abnormalities.[26,56] Relevant side effects of alpha-2 adrenergic agonists include somnolence, headache, and fatigue, and they can also cause a serious withdrawal syndrome characterised by an excessive increase in heart rate and blood pressure. In general, psychostimulants and atomoxetine are well tolerated, and are not considered contraindicated for diving in the absence of serious side effects.[53] However, due to their sedative effects, particularly during the initiation of treatment, diving with clonidine and guanfacine is not advisable.

MOOD STABILISERS

Mood stabilisers are primarily used to treat bipolar disorders and include lithium, anticonvulsants, and antipsychotics. Lithium, valproic acid, carbamazepine, and lamotrigine are the most commonly used mood stabilisers. Lithium, although effective, has a significant side effect profile with polyuria, polydipsia, tremors, and weight gain. Moreover, long-term use of lithium is associated with gradual decline of renal functioning and diabetes insipidus, leading to dehydration.[57] Interestingly, there are some data suggesting that lithium reduces the effects of nitrogen narcosis, but potentiates convulsions and tremors.[19] Valproic acid is generally well-tolerated, with side effects such as gastrointestinal complaints, weight gain, sedation, and tremor.[58] Carbamazepine can cause gastrointestinal complaints, drowsiness, and weight gain.[59] Lamotrigine is well tolerated, with some studies even suggesting that its side effect profile is comparable to placebo.[60] In general, side effects of these anticonvulsants are dose-dependent and tend to decrease when the dosage is reduced. In fitness-to-dive assessments, lithium is the most problematic mood stabiliser. Dehydration caused by lithium is further potentiated in diving due to breathing dry air, immersion diuresis, and sweating when diving in tropical environments. This can be addressed by adequate rehydration after diving. Additionally, the narrow therapeutic range of lithium can be disturbed by the aforementioned factors, leading to lithium toxicity. Therefore, diving with lithium should be considered cautiously. Conversely, the other mood stabilisers are generally well-tolerated and are not contraindicated for diving, taking the general considerations into account.

LIMITATIONS

While this review provides recommendations for diving with psychotropic medications based on the available literature, it is important to note that very little literature is currently available in this area. Therefore, this review should be regarded as an effort to provide guidance for diving physicians, considering that recommendations are sometimes based more on expert opinion and pharmacology than on rigorous scientific evidence. To further develop the knowledge base in this area, we encourage diving physicians and psychiatrists with experience in the treatment of divers to publish their experiences and results.

Additionally, it is important to acknowledge that there is likely an underreporting of psychotropic medication usage in divers, which may limit the practical application of the recommendations provided in this review. We urge clinicians to actively inquire about psychiatric conditions and medication usage during dive medical assessments.

As a side note, in relation to diving incidents seemingly linked to use of psychotropic medications it is often difficult to assess which of the medication or the psychiatric illness that it has been prescribed for is the most important contributor to the chain of events leading to the diving accident. Therefore, it is difficult to appraise the actual risk of diving due to psychotropic medications *per se*. Another important implication is that although the focus of this review has been on psychopharmacology, irrespective of the suitability of a particular drug for diving, assessing physicians should also consider the compatibility of each individual patient's psychiatric presentation with this activity.

It should also be noted that this review is focused on adult recreational divers, and whether the recommendations can be applied to commercial or military divers remains to be determined. Furthermore, diving by children who have psychiatric conditions presents unique challenges that have not been thoroughly explored.[21] Lastly, it is crucial to acknowledge that psychiatry represents a specialised area of medicine where the application of standardised guidelines and checklists is often limited. This is particularly relevant when assessing psychiatric patients who engage in diving activities, given the unique challenges and considerations that arise in this context. As such, we recommend that diving physicians consult with psychiatrists when they are uncertain about the suitability of diving for a particular patient.

Conclusions

In conclusion, this review provides a summary of the limited literature available on psychotropic medication use and fitness to dive, supplemented with expert opinion from psychiatrists with medical expertise in the field. The recommendations presented in this paper aim to provide guidance for diving physicians, but it is crucial to emphasise the importance of individual patient assessment and case-by-case decision making. Diving with psychiatric disease and psychotropic medication use is often possible, but it requires careful evaluation and consideration of potential risks and benefits. We encourage further research and publication in this area to continue advancing the knowledge base and supporting safe diving practices.

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A systematic review of electroencephalography in acute cerebral hypoxia: clinical and diving implications

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Abstract
(Wong NYE, van Waart H, Sleigh JW, Mitchell SJ, Vrijdag XCE. A systematic review of electroencephalography in acute cerebral hypoxia: clinical and diving implications. Diving and Hyperbaric Medicine. 2023 September 30;53(3):268–280. doi: 10.28920/dhm53.3.268-280. PMID: 37718302.)
Introduction: Hypoxia can cause central nervous system dysfunction and injury. Hypoxia is a particular risk during rebreather diving. Given its subtle symptom profile and its catastrophic consequences there is a need for reliable hypoxia monitoring. Electroencephalography (EEG) is being investigated as a real time monitor for multiple diving problems related to inspired gas, including hypoxia.
Methods: A systematic literature search identified articles investigating the relationship between EEG changes and acute cerebral hypoxia in healthy adults. Quality of clinical evidence was assessed using the Newcastle-Ottawa scale.
Results: Eighty-one studies were included for analysis. Only one study investigated divers. Twelve studies described quantitative EEG spectral power differences. Moderate hypoxia tended to result in increased alpha activity. With severe hypoxia, alpha activity decreased whilst delta and theta activities increased. However, since studies that utilised cognitive testing during the hypoxic exposure more frequently reported opposite results it appears cognitive processing might mask hypoxic EEG changes. Other analysis techniques (evoked potentials and electrical equivalents of dipole signals), demonstrated sustained regulation of autonomic responses despite worsening hypoxia. Other studies utilised quantitative EEG analysis techniques, (Bispectral index [BIS™], approximate entropy and Lempel-Ziv complexity). No change was reported in BIS™ value, whilst an increase in approximate entropy and Lempel-Ziv complexity occurred with worsening hypoxia.
Conclusions: Electroencephalographic frequency patterns change in response to acute cerebral hypoxia. There is paucity of literature on the relationship between quantitative EEG analysis techniques and cerebral hypoxia. Because of the conflicting results in EEG power frequency analysis, future research needs to quantitatively define a hypoxia-EEG response curve, and how it is altered by concurrent cognitive task loading.

Introduction

Comprehensively understanding how hypoxia affects the brain will not only benefit clinicians managing cerebral hypoxia in the context of trauma, neurosurgery or anaesthesia, but will also benefit divers. Unanticipated severe hypoxia can occur in divers due to failure of close circuit rebreather devices resulting in rebreathing of a hypoxic gas mixture, or open circuit divers breathing the wrong gas mixture at the wrong depth.[1]

The central nervous system is highly susceptible to hypoxia.[2] An interruption in cerebral blood flow or impaired oxygenation of arterial blood can reduce oxygen availability and failure to meet the demands of the central nervous system. This can result in transient or permanent neurological symptoms.[3] Symptoms associated with mild cerebral hypoxia include difficulties with complex learning tasks, inattention, and amnesia.[4] In moderate cerebral hypoxia, reduced motor co-ordination, and impaired higher-order cognitive functions can arise.[4] In severe cerebral hypoxia, syncope, seizure and neurological death can ultimately result.[4] It is notable that humans appear to have a poor appreciation of hypoxia symptoms as they develop, reducing the chance of recognition and self-rescue.

Given the importance of recognising and reversing hypoxia whilst diving, there is a need for an improved method of hypoxia detection. One proposed method is through the use of electroencephalography (EEG); a promising indicator of

hypoxia with the advantages of being non-invasive, sensitive, objective and measuring in real-time.[5]

Electroencephalography provides a graphical representation of potential differences between cerebral locations.[6] The most frequently used method for classifying EEG waveforms is by frequency. Alpha waves (8–12 Hz) are present in normal awake EEG recordings and are an indication of relaxed wakefulness. Alterations to alpha waves are considered a sign of generalised cerebral dysfunction.[7] Beta waves (13–30 Hz) can increase in the frontal region at low amplitude during active thinking, focus and concentration.[8] Delta waves (< 4 Hz) physiologically occur in deep sleep, and can pathologically occur with focal cerebral dysfunction.[7] Theta waves (4–7 Hz) are present in states of drowsiness and early stages of sleep.[7]

In the last decade, technological advancements have allowed continuous EEG monitoring in clinical practice to be more feasible. Difficulties associated with continuous EEG monitoring are more logistical in nature, such as difficult electrode maintenance, lack of effective computer algorithms and lack of clinical expertise for interpretation.[9] Quantitative EEG analysis techniques, such as bispectral index (BIS™), approximate entropy and Lempel-Ziv complexity utilise advances in computer power and algorithms to allow continuous monitoring of cerebral function, particularly in intensive care and anaesthesia.[9] In theory, the use of continuous EEG in the clinical environment could translate into underwater use, employing algorithms that successfully detect hypoxia. Our group is currently engaged in a program to investigate the utility of multipurpose EEG algorithms for detecting adverse gas effects during diving such as nitrogen narcosis, hypoxia, hyperoxia and hypercapnia.

The objective of this systematic review was to explore and critically analyse the current literature investigating the relationship between EEG changes and acute cerebral hypoxia in healthy adults. Having an in-depth understanding of how EEG is affected by acute cerebral hypoxia could contribute towards the development of future technology to monitor and reduce the risk of fatal cerebral hypoxic events occurring clinically and during diving.

Methods

SEARCH STRATEGY

A systematic electronic search was performed in MEDLINE, EMBASE, Scopus and Web of Science on 12 April 2021. The search term was drafted with the assistance of a University of Auckland librarian, resulting in this MEDLINE search string: ((Electroencephalography/ OR (eeg or eegs or electroencephalogra*).ti,ab,kw,kf.) AND (Hypoxia/ OR Hypoxia, Brain/ OR (hypoxi* or hypox?em*).ti,ab,kw,kf. OR (anoxi* or anox?e*).ti,ab,kw,kf. OR (oxygen adj2 deficien*).ti,ab,kw,kf. OR Altitude/ OR altitude?.ti,ab,kw,kf.)) not (exp animals/ not humans.sh.)

The MEDLINE search string was translated into formats compatible with the other three databases with the aid of a Polyglot webtool and cross-checked by the university librarian.

STUDY SELECTION

Search results from all four databases were exported to a reference managing database (Covidence, Melbourne, Australia) for de-duplication and review. Preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines were followed.[19] All non-English language articles were translated for the screening and review process. Three reviewers (NW, XV, HvW) independently screened all titles and abstracts in double and excluded irrelevant studies. No date or language limits were set. Exclusion criteria included systematic reviews, meta-analyses, letters, editorials, case reports, paediatric studies, animal studies or studies investigating participants with pathologies. Any disagreements over inclusion or exclusion of studies were discussed between the three reviewers to reach consensus.

Full text copies of all potentially relevant studies were obtained for review. In addition to the exclusion criteria utilised for title and abstract screening, full-text studies were excluded if some index of the hypoxic severity was not described (i.e., inspired partial pressure of oxygen [PiO_2] or peripheral oxygen saturation [SpO_2] were not stated), or if there was no clear qualitative or quantitative description of EEG outcomes. Additional studies were retrieved by liaising with content experts (senior anaesthetists and research professors) to contribute any relevant grey literature that was not captured by our search strings. Reference lists of all included full-text studies were also manually reviewed for additional relevant publications (literature snowballing).

DATA EXTRACTION

Data extraction was performed by NW and XV and entered into a pre-designed electronic table. Data extracted included: participant demographics, study design, extent of hypoxia achieved, EEG results and analysis. The quantitative data extracted was too varied to reliably perform a formal meta-analysis.

QUALITY OF EVIDENCE

The Newcastle-Ottawa scale was applied to assess quality of evidence. This scale assesses the methodological quality of: cohort selection (4 points), comparability of the study groups (2 points), and quality of outcomes assessed (3 points).[10] Cohort selection is determined by the representativeness of the exposed cohort to the target population, how the non-exposed cohort is selected, the ascertainment of the exposure, and demonstration that the outcome of interest was not present at the start of the study. Study group comparability is based on whether the study controls for potentially confounding factors (such as age and gender).

Quality of outcomes assessed is based on whether there was independent blind assessment of outcomes, whether sufficient time was allowed for outcomes to occur, and the adequacy of follow-up of cohorts. A score is formulated out of a maximum of nine points. The thresholds for converting the Newcastle-Ottawa scale into an objective assessment of study quality (as per the Agency for Healthcare Research Quality standards) is as follows:

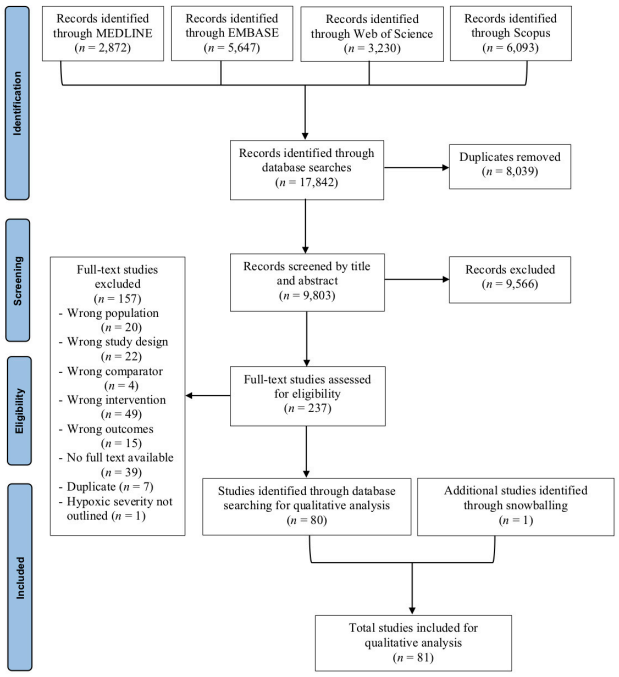
- Good quality – 3 or 4 points in selection domain AND 1 or 2 points in comparability domain AND 2 or 3 points in assessed outcome domain.
- Fair quality – 2 points in selection domain AND 1 or 2 points in comparability domain AND 2 or 3 points in assessed outcome domain.
- Poor quality – 0 or 1 point in selection domain OR 0 points in comparability domain OR 0 or 1 points in assessed outcome domain.

Results

STUDIES INCLUDED FOR ANALYSIS

The search identified 17,842 records, with 9,803 remaining for title and abstract screening after duplicates were removed. After initial screening, 237 full-text studies were assessed for eligibility. Eighty studies met the inclusion criteria. One study was identified through snowballing, resulting in 81 studies for analysis (Figure 1). A complete list of these studies can be found in online supplementary Table S1*.

Figure 1
PRISMA flow diagram for the review



QUALITY OF EVIDENCE

The overall quality of evidence was poor. Only ten studies were graded as being of good quality. The comparability domain scored the worst, where more than 80% of studies scored no points (Table 1).

TYPE OF STUDIES

Thirteen controlled trials were identified, which involved randomisation to another intervention. The remaining studies identified were experimental cohort studies.

STUDY PARTICIPANTS

The majority of studies were very small, with less than 20 participants recruited. The greatest number of participants (n = 203) were recruited in an experimental cohort study that investigated the tolerance time to hypoxia in trainee pilots at progressively increasing altitudes simulated in a hypobaric chamber.[11] Only four studies recruited more females than males. The majority of participants were under 40 years of age, with the oldest participant aged 57.[12] All participants were healthy volunteers who were naïve to hypoxic or altitude exposure. In particular, three studies recruited athletes,[13–15] one study recruited divers,[16] and six studies recruited pilots, parachutists or mountaineers (Table 2).[11,17–21]

MODERATE VS SEVERE HYPOXIA

Seventeen studies investigated more than one hypoxic exposure (nine studies investigated two exposures; six studies investigated three exposures; two studies investigated four exposures), producing a total of 108 exposures for analysis. The 108 exposures were categorised into two groups for comparison: moderate and severe hypoxia. Moderate hypoxia was defined as SpO_2 75–90% and severe hypoxia was defined as SpO_2 < 75%. For studies that did not state an SpO_2 outcome, severity of hypoxia was categorised based on PiO_2 exposure as a secondary outcome. In these studies, moderate hypoxia was defined as PiO_2 ≥ 10 kPa and severe hypoxia was defined as PiO_2 < 10 kPa. This categorisation produced 48 moderate hypoxia exposures and 60 severe hypoxia exposures for analysis and comparison.

INDUCTION OF HYPOXIA

Hypoxia was induced in a hypobaric chamber or an altitude exposure, or via normobaric hypoxia (inhalation of a hypoxic gas mixture). The most prevalent method of inducing moderate hypoxia was in a hypobaric chamber, whilst the most prevalent method of inducing severe hypoxia was via an inhaled hypoxic gas mixture. All altitude exposures produced a moderately hypoxic environment (Figure 2).

Footnote: * Table S1 in spreadsheet format is available on DHM Journal's website: https://dhmjournal.com/images/HTML/Wong_TableS1.htm. A table of the references cited in S1 is also available: https://www.dhmjournal.com/index.php/journals?id=320

Table 1

Newcastle-Ottawa scale quality of evidence; [a] Selection domain maximum 4 points; [b] Comparability domain maximum 2 points; [c] Assessed outcome domain maximum 3 points; N/A – Not applicable

Score	Selection [a]	Comparability [b]	Assessed outcome [c]
≥ 3 points	26	N/A	55
2 points	40	0	22
1 point	15	15	4
0 points	0	66	0
Overall assessment	**Good**	**Fair**	**Poor**
Number of studies	10	5	66

Table 2

Study participant characteristics

Parameter	*n* studies
***n* subjects**	
< 10	14
10–19	35
20–29	11
30–39	7
40–49	5
≥ 50	9
Male	
100%	39
50–99%	24
< 50%	4
Not stated	14
Upper age (years)	
18–19	1
20–29	17
30–39	24
40–49	14
50–59	6
Not stated	19
Characteristic	
Unspecified healthy volunteers	60
Students	9
Athletes	3
Divers	1
Pilots/parachutists/ mountaineers	6
Not stated	2

Figure 2

Methods of inducing hypoxia employed in the studies

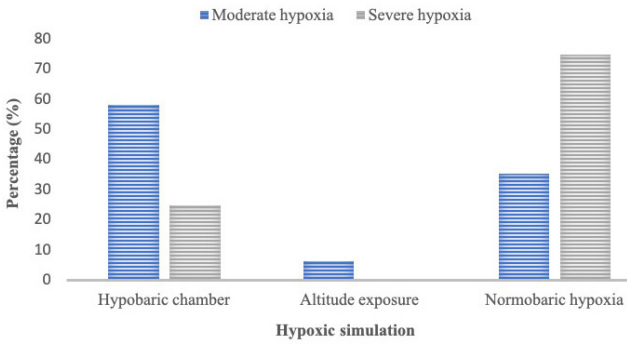
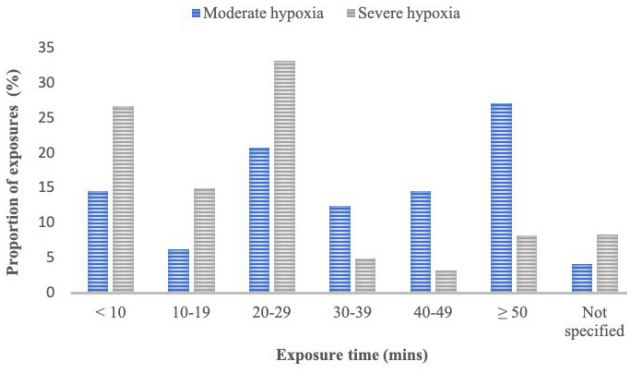


Figure 3

Hypoxia exposure times employed in the studies



EXPOSURE TIME

Hypoxic exposures were more frequently longer in the moderate hypoxia group, with the longest hypoxic exposure being 30 days secondary to ascent to higher altitude.[22] This is in contrast to the higher proportion of short exposures in the severe hypoxia group, where the shortest exposure was 12 seconds in a hypobaric chamber (Figure 3).[23]

EEG FREQUENCY ANALYSIS

Only 12 studies reported numerical changes in spectral power of EEG frequencies (Table 3). The remaining studies provided either written descriptions or graphs showing general trends in EEG frequency changes, from which specific numerical values could not be accurately ascertained.

Table 3

Experimental cohort studies that reported numerical changes in spectral power of EEG frequencies (n = 12); reference 31 was a randomised controlled trial. Reference 13 specifically recruited athletes, swimmers and skiers. Participants in all studies were described as healthy volunteers. FiO_2 – fraction of inspired oxygen; PiO_2 – pressure of inspired oxygen; SpO_2 – peripheral oxygen saturation EEG - electroencephalography

Study		Participant demographics			Study design						% change in EEG spectral power			
Ref	Year	*n*	Gender	Age range	Induction of hypoxia	Exposure (mins)	FiO_2 (%)	PiO_2 (kPa)	SpO_2 (%)	Hypoxia classification	Alpha	Beta	Delta	Theta
13	2012	24	100% Male	18–26	Inhaled hypoxic gas mixture	25	10	9.5	74	Severe	-29.4			+5.2
24	2020	12	100% Male		Inhaled hypoxic gas mixture	25	8	7.6		Severe	+30.9		+302.7	+287.4
25	2020	12	100% Male	20–25	Inhaled hypoxic gas mixture	600	12	11.4		Moderate	+31.0			+29.6
26	2005	10	50% Male; 50% Female	26–57	Brought to higher altitude	6	21	14		Moderate	+15.0	+30.0		
27	2008	10	100% Male	21–30	Hypobaric environment 4,000 m	6	21	11.6	83.8	Moderate	+46.1	+64.5		
28	1988	36	100% Male	18–27	Hypobaric environment	10	21	8.4	70	Severe	-20.6		+34.7	+17.3
29	2007	10	50% Male; 50% Female	21–41	Hypobaric environment 7,620 m, 6,096 m, 4,572 m	90	21	6.6, 8.5, 10.7		Severe at 7,620 m and 6,096 m; Moderate at 4,572 m	+22.2 (7,620 m) % change at other altitudes not stated			+23.8 (7,620 m) % change at other altitudes not stated
30	2003	3	33% Male; 67% Female	24–32	Rebreathing from a 5 L bag of room air	4.85	21		73	Severe	-40.5	-17.2		+16.5
31	1987	15	47% Male; 53% Female	21–41	Inhaled hypoxic gas mixture	23	9.8	9.3		Severe	-23.0	+157.1	+1.5	+1.5
32	1986	8	100% Male		Inhaled hypoxic gas mixture	20	10.5	9.98		Severe	-10.5	+15.5	-19.0	-9.0
22	2002	68	100% Male	18–19	Ascent to altitude – 3,600 m	43,200	21	12.3		Moderate	+56.9			+40.0
33	2002	12	67% Male; 33% Female	19–27	Inhaled hypoxic gas mixture	130			80	Moderate	-9.2		-3.6 at SpO_2 90%; % increase at SpO_2 80% not stated	-7.2 at SpO_2 90%; % increase at SpO_2 80% not stated

Table 4
Descriptive changes in EEG frequencies under moderate and severe hypoxic conditions; ↑ – Increased activity; ↓ – Decreased activity; ↔ – No change in activity

Hypoxic severity	Electroencephalogram frequency band															
	Alpha				Beta				Delta				Theta			
	↑	↓	↔	Not stated	↑	↓	↔	Not stated	↑	↓	↔	Not stated	↑	↓	↔	Not stated
Moderate (n = 48)	5	13	3	27	9	3	1	35	7	1	1	39	11	5	0	32
Severe (n = 60)	6	33	1	20	9	6	1	44	27	3	1	29	35	2	0	23

Table 5
Descriptive changes in EEG frequencies with cognitive testing compared to no cognitive testing under hypoxic conditions; ↑ – Increased activity; ↓ – Decreased activity; ↔ – No change in activity

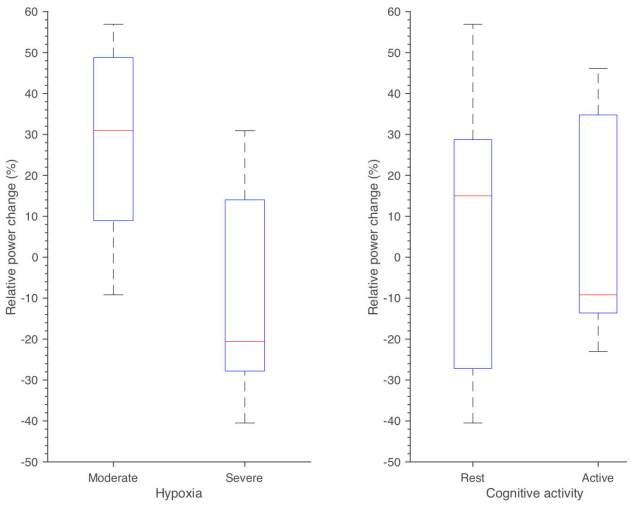
Cognitive testing	Electroencephalogram frequency band															
	Alpha				Beta				Delta				Theta			
	↑	↓	↔	Not stated	↑	↓	↔	Not stated	↑	↓	↔	Not stated	↑	↓	↔	Not stated
Yes (n = 34)	7	14	0	13	6	3	1	24	13	2	1	18	16	1	0	17
No (n = 74)	4	32	4	34	12	6	1	55	21	2	1	50	30	6	0	38

Alpha waves

Based on the 12 studies that specified numerical changes in spectral power of EEG frequencies, moderate hypoxia caused a 31% increase, compared to a 20.6% decrease in median alpha spectral power under severe hypoxic conditions (Figure 4). However, when including the qualitative studies, there appeared to be an overall decrease in alpha activity under both moderate and severe hypoxic exposures, where the decrease in alpha activity was more frequently reported under severe hypoxia compared to moderate hypoxia (Table 4). In general, the quantitative studies were more recent, with modern technology, while the studies with qualitative data were older, which mean that most results were based on visual interpretation of the EEG, which has severe limitations.

Amongst studies that reported an increase in alpha activity, a large proportion (seven out of 11) occurred in those where participants performed simple cognitive tasks during the hypoxic event, while 32 out of 46 showed a decrease in alpha power when no cognitive testing was performed (Table 5). This relationship is not depicted in Figure 4, which demonstrates a 15% increase in median alpha spectral power when no cognitive tasks were performed, and a 9.2% decrease in median alpha spectral power when cognitive tasks were performed under hypoxic conditions. However, only seven out of the 34 studies that utilised cognitive testing specified numerical changes in alpha spectral power.

Figure 4
Box plot of relative change in spectral power in the alpha band; study results are split between moderate and severe hypoxia (left) and rest or active cognitive function (right). The box plot shows the median (red line), interquartile range (blue box) and the whiskers show the range



Delta and Theta waves

Delta and theta waves generally increased under both moderate and severe hypoxic conditions (Table 4, Figures 5 and 6). The increase in delta activity under moderate

hypoxia is depicted as a red line without box and whiskers in Figure 5. This is because out of the seven studies that described an increase in delta activity under moderate hypoxia, only one study quantified these changes.[33] The authors of this study reported a 3.6% decrease in delta spectral power at SpO_2 90%, but an unspecified increase in delta spectral power at SpO_2 80%.[33]

The increase in both delta and theta activity was more frequently reported under severe hypoxic conditions (Table 4). More than 50% of studies did not report any changes in delta or theta activity, pertaining mainly to those that had a moderate hypoxic exposure. An increase in these frequencies was also more frequently reported in the absence of cognitive testing (Table 5), corresponding to 168.7% and 16.9% increases in median delta and theta spectral powers respectively, (Figures 5 and 6). Two studies noted an increase in theta activity predominantly over the frontal and parietal cortices.[22,34] One study reported a decrease in theta activity which was only observed over the frontal and central cortices.[35] Burykh, et al. demonstrated an increase in theta spectral power of 287.4%,[24] corresponding to the upper limit depicted in the severe hypoxia and resting state boxplots in Figure 6. Whilst this is a notable outlier, those authors investigated a small population and several EEG frequency changes were analysed by visual inspection.[24]

Beta waves

More than 70% of studies did not comment on nor specify any changes in beta activity. Studies that did generally reported an increase in beta spectral power for both moderate and severe hypoxic exposures (47.3% and 15.5%, respectively) (Table 4 and Figure 7). The majority of studies that utilised cognitive testing during the hypoxic event demonstrated an increase in beta activity (Table 5), which was also reflected by the 64.5% increase in median beta spectral power in studies that utilised cognitive testing during the hypoxic event (Figure 7).

OTHER EEG ANALYSIS TECHNIQUES

Nine studies utilised other analysis techniques rather than analysis of specific EEG frequencies (Table 6). These other analyses can be broadly categorised as evoked potentials, source localisation and quantitative analysis techniques.

Evoked potentials

Evoked potentials provide an insight into the processes underlying sensory load perception.[45] One study measured respiratory-related evoked potentials concluding that hypoxic conditions suppress respiratory afferent input to the medulla.[37] Three studies utilised auditory evoked potentials.[40,41,43] All three demonstrated a reduction in amplitude of endogenous positive and negative slow waves, corresponding to a disruption in focus, attention and auditory processing secondary to hypoxia.[40,41,43]

Source localisation

Electrical equivalents of dipole signals, which measure the electrical activity of subcortical regions,[42] were analysed

Figure 5

Box plot of relative change in spectral power in the delta band; study results are split between moderate and severe hypoxia (left) and rest or active cognitive function (right). The box plot shows the median (red line), interquartile range (blue box) and the whiskers show the range

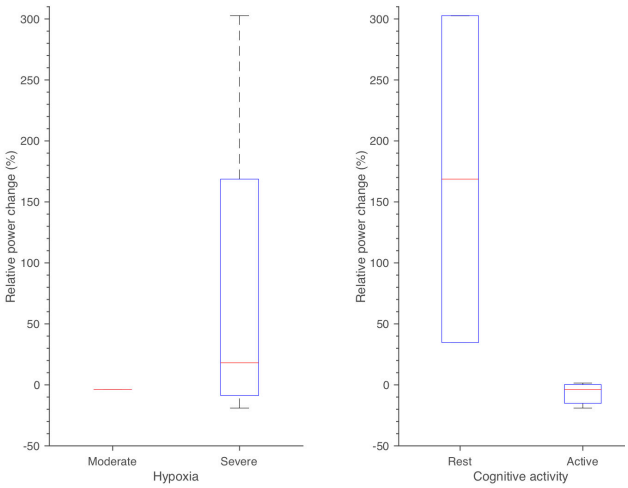


Figure 6

Box plot of relative change in spectral power in the theta band; study results are split between moderate and severe hypoxia (left) and rest or active cognitive function (right). The box plot shows the median (red line), interquartile range (blue box) and the whiskers show the range. The outlier (red +) is larger than 1.5 times the interquartile range and is outside the scale (dotted line) at 287.4%

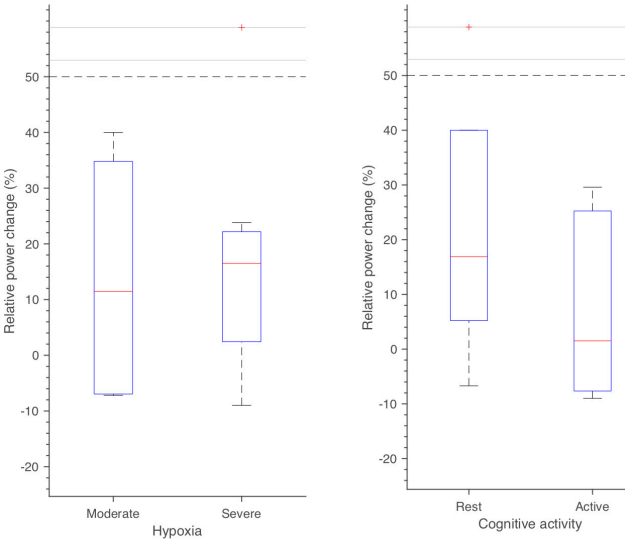
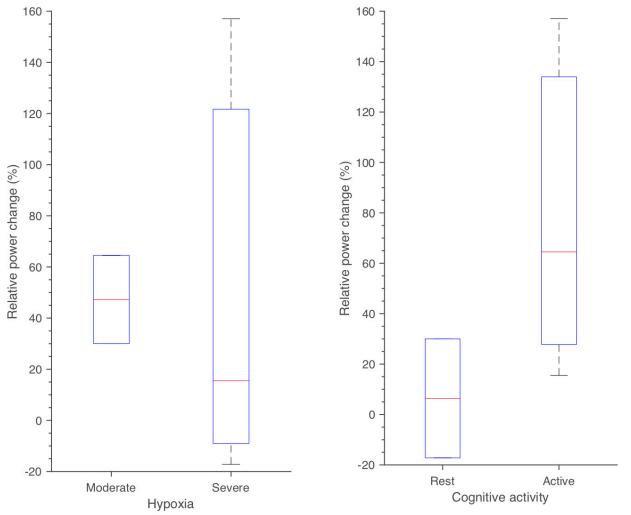


Figure 7
Box plot of relative change in spectral power in the beta band; study results are split between moderate and severe hypoxia (left) and rest or active cognitive function (right). The box plot shows the median (red line), interquartile range (blue box) and the whiskers show the range



in two studies.[42,44] Both demonstrated an increased signal density in the hypothalamus, forebrain, temporal lobe and increased activation of the limbic system with initial hypoxic exposure. The authors concluded that this signal redistribution demonstrates the dynamic adaptive functions and self-regulatory mechanisms of the brain, resulting in a stable regulation of physiological parameters despite a worsening oxygen deficit.[42,44]

Quantitative analysis techniques

Bispectral index (BIS™) is a proprietary signal-processed EEG that produces a single dimensionless number to provide an indication of depth of anaesthesia.[46] One study demonstrated no change in BIS™ across a range of SpO_2 readings from normal to as low as < 69%.[38]

Approximate entropy is a statistical approach to characterising EEG signals.[36] Lempel-Ziv complexity tests the randomness of a sequence to assess patterns within a deterministic, non-linear EEG signal.[36,39] One study utilised both approximate entropy and Lempel-Ziv complexity, showing a progressive increase in both values with worsening hypoxia.[36] The authors proposed that these methods can be used to evaluate changes in neurological function at different hypoxic severities.[36] These changes in complexity could be explained by the changes in beta frequency power reported earlier.

Discussion

This systematic review aimed to explore and critically analyse current literature investigating the relationship between EEG changes and acute cerebral hypoxia in healthy adults. There was evidence for EEG frequency changes associated with varying severities of hypoxic exposure although quantitative EEG analysis techniques have the greatest potential for evaluating the presence and severity of cerebral hypoxia.[6]

EEG FREQUENCY ANALYSIS

Alpha waves

Most studies in this systematic review reported changes in alpha waves. Under normal physiological conditions, alpha waves are associated with a state of relaxed wakefulness, where an alteration can be attributed to generalised cerebral dysfunction.[7] Overall, the reviewed studies confirmed a decrease in alpha wave activity under both moderate and severe hypoxic conditions (Table 4). One study reported that task difficulty and visual stimulus handling could affect alpha frequency amplitudes during continuous EEG monitoring.[47] Similarly, a large proportion of studies that reported an increase in alpha activity were those in which cognitive tasks were performed during the hypoxic exposure (Table 5). It is possible that there are other circumstances where alpha activity may also increase, and therefore cause an erroneous interpretation of an individual's hypoxic state.

Delta and Theta waves

Both delta and theta waves are physiologically associated with sleep.[7] It was therefore anticipated that these frequencies would increase during a hypoxic event and in the absence of cognitive testing, which aligns with the results of this systematic review (Tables 4 and 5). Nearly 50% of studies did not observe significant changes in delta or theta waves, whilst those that did, tended to observe an increase in delta and theta waves at severe hypoxic thresholds (Table 4). It is possible that these studies may also have other factors contributing to the observed increase in delta and theta waves. Whilst an increase in delta and theta activity may be a useful indicator of severe cerebral hypoxia, identification of earlier stages of cerebral hypoxia may be missed if relying on delta and theta waves alone.

Beta waves

More than 70% of studies did not comment on beta waves. The few studies that did, predominantly reported an increase in beta activity (Table 4). Beta waves are associated with active thinking, focus and concentration,[8] which aligns with the fact that the majority of studies where cognitive testing was utilised, reported an increase in beta activity (Table 5). Xi, et al. demonstrated that sedatives can also increase beta activity.[48] Like alpha waves, there may be other factors that could also contribute towards an increase in beta activity, resulting in an inaccurate interpretation of an individual's hypoxic state. This highlights the need for a more specific and reliable method of EEG analysis to monitor cerebral hypoxia.

Table 6

Experimental cohort studies that utilised other EEG analysis techniques ($n = 9$); reference 38 specifically recruited members of the defence force. Participants in all studies were described as healthy volunteers. BIS™ – Bispectral Index; EEG – electroencephalography; EEDS – Electrical equivalents of dipole signals; FiO_2 – fraction of inspired oxygen; PiO_2 – pressure of inspired oxygen; SpO_2 – peripheral oxygen saturation

Study		Participant demographics			Study design						Other EEG analysis techniques
Ref	Year	*n*	Gender	Age range	Induction of hypoxia	Exposure (mins)	FiO_2 (%)	PiO_2 (kPa)	SpO_2 (%)	Hypoxia classification	
36	2005	3	100% Male		Inhaled hypoxic gas mixture – 3,500 m	30	21	12.5		Moderate	Lempel-Ziv complexity and approximate entropy values increased with hypoxia
37	2005	11	55% Male; 45% Female		Inhaled hypoxic gas mixture	30	9	8.6	82.2	Moderate	Reduced P1 (32.4%) and P2 (20.2%) amplitudes (positive slow waves) of respiratory-related evoked potentials
38	2009	11	82% Male; 18% Female	20–46	Hypobaric environment – 7,620 m	5	21	6.6	69	Severe	No change in BIS™ value with a hypobaric hypoxia exposure (SpO_2 69%) compared to hypobaric normoxia and sea level exposures
39	2006	12	42% Male; 58% Female	26–32	Rebreathing from a 5 L bag of room air	7	21		75	Moderate	Lempel-Ziv complexity increased in subjects who experienced anxiety. No appreciable change in Lempel-Ziv complexity in subjects who were not anxious. EEG frequencies remained unchanged at SpO_2 75%
40	1993	38	100% Male	22–40	Hypobaric environment – 3,000 m, 4,000 m, 5,000 m, 6,000 m	45	21	12.8; 11.6; 10.0; 8.6	63.4 at 6,000 m	Moderate at 3,000 m, 4,000 m and 5,000 m; Severe at 6,000 m	Both negative and positive slow waves of auditory evoked potentials decreased in amplitude with increasing altitude
41	1975	10	100% Male	27–30	Inhaled hypoxic gas mixture	20	8	7.6		Severe	Decreased amplitude and latency of auditory evoked potentials
42	2009	11	100% Male	25–34	Inhaled hypoxic gas mixture	20	8	7.6	35–65	Severe	Increased density of EEDS foci in hypothalamus, thalamus, pons, temporal lobe and limbic system
43	2020	40	67.5% Male; 32.5% Female		Hypobaric environment	27	10.6	10.1	75	Moderate	Reduced P3a amplitude of auditory evoked potentials within the first 9 mins of hypoxic exposure. Reduced auditory processing with hypoxia
44	2007	37		25–34	Inhaled hypoxic gas mixture	22.5	8	7.6	62	Severe	Redistribution of EEDS foci. Increased density in hypothalamus, forebrain and limbic system

OTHER EEG ANALYSIS TECHNIQUES

EEG can be a complex, chaotic time series signal. As a result, researchers and clinicians (particularly anaesthetists and intensive care specialists) generally use EEG analysis techniques other than frequency analysis to aid with interpretation.

Evoked potentials and source localisation

Respiratory related evoked potentials provide an insight into the sensory processes underlying respiratory load perception.[45] Electrical equivalents of dipole signals measures the electrical activity of subcortical regions.[42] Studies that utilised respiratory related evoked potentials or electrical equivalents of dipole signals demonstrated stable regulation of cardiovascular and respiratory parameters despite an oxygen deficit.[37,42,44] This was supported by another study which demonstrated no change in either respiratory rate or heart rate with prolonged duration or worsening hypoxic severity.[49] Edlinger, et al. also demonstrated splay between EEG frequency changes and autonomic vital signs with worsening hypoxia.[12] Overall, these studies indicate that autonomic responses can be impaired secondary to cerebral hypoxia. This therefore suggests that solely monitoring autonomic vital signs may not be a useful technique for monitoring cerebral hypoxia and could lead to an inaccurate interpretation of the severity of an individual's hypoxic state.

Quantitative analysis techniques

Bispectral index (BIS™) is a proprietary signal-processed EEG index that produces a single dimensionless number quantifying the shift to predominance of low frequencies – and hence to provide an indication of depth of anaesthesia.[46] The only study reporting the effect of hypoxia on BIS™ readings in adults showed no change across a range of peripheral oxygen saturations from normal to 69%.[38] Most probably the increase in both delta and beta power, as seen in severe hypoxia, would counterbalance each other – thus causing no consistent change in the BIS™ index.

Approximate entropy is a statistical approach of characterising and classifying EEG signals.[36] Lempel-Ziv complexity measures complexity as defined by Kolmogorov, by testing the randomness of a sequence to assess patterns within a deterministic, non-linear EEG signal.[36,39] One study concluded that there is a progressive increase in both approximate entropy and Lempel-Ziv complexity values with worsening hypoxia,[36] whilst another only demonstrated a significant increase in Lempel-Ziv complexity in subjects who were anxious during the hypoxic exposure.[39] Interestingly Jernajczyk, et al. failed to demonstrate significant changes in EEG frequencies in response to hypoxia, but noticed that changes in cortical electrical activity became more apparent by utilising Lempel-Ziv complexity.[39] This emphasises the difficulties, limitations and potential for inaccurate interpretations that can arise by analysing EEG frequency changes alone, and infers greater potential efficacy of quantitative EEG analysis techniques.

LIMITATIONS

Inevitably, there are some limitations in this review. Firstly, the overall quality of evidence was poor, with the comparability domain scoring lowest. The majority of studies did not control for age or gender, nor specify the type of participants recruited, thus study cohorts were poorly comparable with each other. Secondly, the high quantity of articles that were evaluated during the screening process could have created a degree of selection bias. However, each title, abstract and full-text study was reviewed at least twice by three independent reviewers, reducing the probability of incorrectly excluding a relevant study. Thirdly, participants of studies analysed in this review were young. Since EEG changes with age,[50] the results from this systematic review may not accurately reflect how EEG changes with cerebral hypoxia in an older individual and could therefore not be applicable to an older population (> 60 years). Lastly, only 12 studies specified numerical changes in spectral power of EEG frequencies. Whilst these values largely correlated to the overall trends in EEG frequency changes outlined in other studies, further research is needed to properly validate the extent of these changes for each EEG frequency under varying hypoxic exposures and cognitive loads.

FUTURE IMPLICATIONS

Further research is required to investigate the practical and clinical utility of EEG frequency analysis to monitor cerebral hypoxia. Research is also required to evaluate how various factors, such as cognitive load, could influence EEG frequencies. This could involve directly measuring EEG frequencies in participants whilst they perform cognitive tasks in progressively deteriorating hypoxic environments compared to participants who are not subjected to a cognitive load. This would allow more specific assessment into the progression and pattern of EEG changes that can be expected at different hypoxic thresholds and determine how factors such as severity of hypoxic exposure or cognitive load could influence EEG frequencies.

This systematic review has identified quantitative EEG analysis techniques (BIS™, approximate entropy, Lempel-Ziv complexity), that could provide greater clinical utility for monitoring cerebral hypoxia compared to the assessment of EEG frequencies. These quantitative analysis techniques are advantageous as they are usually less expensive and require less expertise to interpret.[9] Further research is required to comprehensively assess how BIS™, approximate entropy and Lempel-Ziv complexity are affected by cerebral hypoxia

in healthy adults, and whether these modalities are more reliable for continuous EEG monitoring compared to the assessment of EEG frequencies.

Conclusions

There is a relationship between acute cerebral hypoxia and EEG frequency changes. Alpha waves decrease whilst delta and theta waves increase in response to moderate and severe hypoxic exposures, although these changes may be affected by factors such as severity of hypoxic exposure and cognitive load. Current evidence on the relationship between quantitative analysis of EEG and cerebral hypoxia is limited. Future research is required to quantitatively define a hypoxia-EEG response curve and how factors such as cognitive load affects this relationship. The comparative utility of analysing EEG frequencies versus quantitative analysis of the EEG also requires further investigation. As part of a study to determine whether prior hypoxic exposure helps recognition of a subsequent hypoxic event among divers, our group is undertaking a series of hypoxic exposures with EEG monitoring of sufficient quality to allow all relevant analyses. A related aim in our currently funded research program is the development of technologies that improve real-time hypoxia detection and reduce the risk of fatal cerebral hypoxic events occurring in divers.

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Conflicts of interest

Simon J Mitchell is the Editor of Diving and Hyperbaric Medicine. He took no part in the peer-review and decision-making processes for this paper, which was managed entirely by the Deputy Editor, Dr Lesley Blogg. There were no other conflicts of interest.

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Short communication

Investigation into the effect of hyperbaric hyperoxia on serum cardiac Troponin T levels as a biomarker of cardiac injury

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Keywords
Biomarkers; Cardiovascular; Health; Heart; Hyperbaric oxygen treatment

Abstract
(Marjot J, Mackenzie J, Jepson N, Reeves E, Bennett M. Investigation into the effect of hyperbaric hyperoxia on serum cardiac Troponin T levels as a biomarker of cardiac injury. Diving and Hyperbaric Medicine. 2023 September 30;53(3):281–284. doi: 10.28920/dhm53.3.281-284. PMID: 37718303.)
Introduction: There is clinical equipoise as to whether hyperoxia is injurious to the myocardium, both in the setting of acute ischaemic insults and on the stable myocardium. This study examined the effect of extreme hyperoxia – in the form of hyperbaric oxygen treatment – on the myocardium through measurement of high-sensitivity cardiac troponin.
Methods: Forty-eight individuals were enrolled to undergo a series of 30 exposures to hyperbaric oxygen for treatment of non-cardiac pathologies. High-sensitivity troponin T was measured before and after each session.
Results: There was no clinically significant difference in troponin measurements following acute or recurrent sequential exposures to extreme hyperoxia, despite the studied patient population having a high rate of previous ischaemic heart disease or cardiovascular risk factors.
Conclusions: This study demonstrates that profound hyperoxaemia does not induce any measurable cardiac injury at a biochemical level. Neither is there a reduction in cardiac troponin to suggest a cardioprotective effect of hyperbaric hyperoxia. This provides some reassurance as to the cardiac safety of the routine use of hyperbaric oxygen treatment in management of non-cardiac pathology.

Introduction

Hyperbaric oxygen treatment (HBOT) is an intervention in which an individual intermittently breathes near 100% oxygen while inside a hyperbaric chamber pressurised to greater than 101.3 kPa (1 atmosphere absolute [atm abs]). At 203 kPa (2 atm abs) arterial oxygen tension is expected to be over 1,000 mmHg.[1]

The cardiac effects of HBOT have been studied in the context of acute cardiac insults, such as acute coronary syndromes, carbon monoxide poisoning, angioplasty and coronary artery bypass grafting.[1–3] These human and animal studies suggest HBOT has a protective effect on the myocardium as evidenced by reduced area of necrosis, lower levels of necrosis biomarkers, improved cardiac function, or improved clinical outcomes. A 2015 Cochrane review of effects of HBOT in acute coronary syndromes found some evidence from small trials to suggest that HBOT is associated with a reduction in the risk of death, the volume of damaged muscle, and the risk of major adverse cardiac events. However, insufficient evidence was found to support the routine use of HBOT in this setting.[1]

Conversely, there is evidence that hyperoxia may in fact be damaging to the myocardium in the setting of an acute ischaemic insult. Oxygen therapy (normobaric oxygen – NBO) in patients with ST-segment elevation myocardial infarction (STEMI) but without hypoxia may increase early myocardial injury and result in larger myocardial infarct size at six months.[4]

However, a subsequent randomised control trial comparing the effect of supplemental oxygen versus ambient air in patients with symptoms suggestive of acute myocardial infarction, but with oxygen saturations of 90% or higher, did not demonstrate any significant difference in peak troponin measurement or 1-year all-cause mortality.[5]

Thus, the effect of hyperoxia, whether that be normobaric or hyperbaric, on the acutely ischaemic myocardium remains unclear.

Figure 1
Possible mechanisms by which hyperbaric hyperoxia may have a protective or injurious effect on human myocardium; cTN – cardiac troponin; TPR – total peripheral resistance

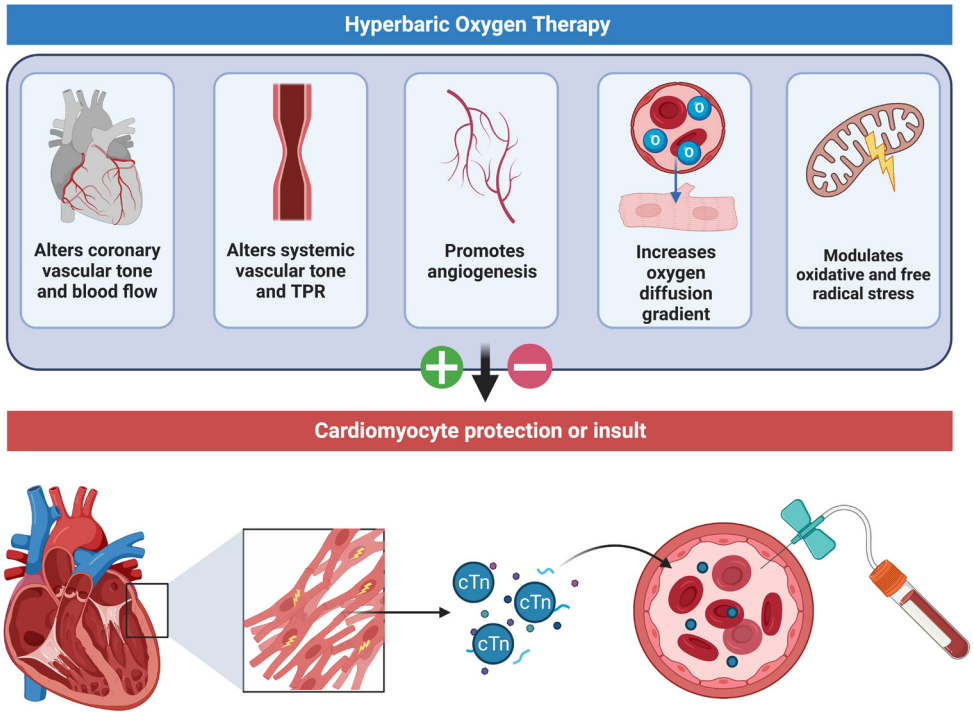

Table 1
Statistical analysis of paired troponin levels measured in ng·L^{-1}; HBOT – hyperbaric oxygen treatment; hs-cTnT – cardiac troponin; SEM – standard error of mean

Paired hs-cTnT measurements before and after 90 min exposure to HBOT				
Treatment number	Number of paired samples	Mean (SEM) pre-treatment	Mean (SEM) post-treatment	*P*-value
1	35	18.56 (6.03)	18.76 (7.37)	0.91
10	30	18.83 (9.29)	18.27 (8.43)	0.62
20	33	20.60 (7.94)	19.79 (7.76)	0.22
30	28	20.23 (9.05)	19.28 (8.28)	0.24
Paired hs-cTnT measurements before and after a course of 30 HBOT exposures				
Treatment number	Number of paired samples	Mean (SEM) treatment 1	Mean (SEM) treatment 30	*P*-value
1 vs 30	35	18.07 (6.06)	18.77 (7.25)	0.64

With respect to the stable myocardium, in the absence of an acute ischaemic insult, the effect of hyperoxia is also uncertain. A 2009 review found that hyperoxia resulting from NBO exposure resulted in increased coronary vascular resistance and reduced coronary blood flow.[6] These experimental studies produced modest hyperoxia in the range of 250 to 450 mmHg, markedly less than the arterial oxygen tensions achieved during HBO exposure. A small study in patients with stable multivessel coronary artery disease has indicated that inhalational hyperoxia in these patients may be associated with adverse effects of regional myocardial deoxygenation.[7]

Hyperbaric oxygen is used globally in the treatment of a wide range of non-cardiac pathology. However, many of these patients have risk factors for coronary artery disease. Thus, there should be an index of concern for the effect of repeated HBOT exposures on the myocardium, even in the absence of an acute

ischaemic insult. HBOT represents a possible double-edged sword with biological plausibility for cardio-protection or injury (Figure 1).

In this study we sought to assess any injurious effect of hyperbaric hyperoxia on the myocardium in patients undergoing a course of HBOT for non-cardiac pathologies, through measurement of the cardiac-restricted biomarker of necrosis, troponin T.

Methods

The study was approved by South Eastern Sydney Local Health District human research ethics committee (HREC: 2021/ETH00415).

Cardiac troponin T (hs-cTnT) was measured using a high-sensitivity assay (Roche Elecsys),[8] in consecutive patients planned to undergo greater than twenty HBOT exposures for treatment of non-cardiac pathology. Each treatment consisted of 90 min exposure to > 203 kPa (2 atm abs) pressure whilst intermittently breathing 100% oxygen, and the planned treatment course consisted of five exposures a week for 4–6 weeks.

Individuals were not enrolled if they had suffered an acute coronary syndrome or undergone cardiac intervention in the last six months, or if they had known chronic kidney disease stage four or greater.

Forty-eight individuals were prospectively enrolled (mean age 69 years, 65% male). Two participants withdrew from the study before the collection of any paired samples and were not included in data analysis; one due to patient preference, and one due to reporting chest pain before commencing treatment. Participants represented a high-risk demographic for cardiac disease: 25% had a known history of established ischaemic heart disease (IHD), 13% had previous coronary stents or bypass grafting, 13% were diabetic, and 52% were on at least one antihypertensive or cholesterol lowering agent. Of measured baseline troponins, 36% had serum hs-cTnT greater than the upper reference limit of 14 ng·L^{-1}.

Serum hs-cTnT was measured before and after HBOT sessions 1, 10, 20, and 30 (a total of four paired hs-cTnT measurements). Logistical issues affecting HBOT treatments and phlebotomy during the COVID-19 pandemic meant that a complete set of paired samples was not achieved for all participants. Nonetheless, all participants contributed at least one paired set of data to the analysis, and 83% of individuals contributed 2, 3, or 4 paired sets of data. No participant experienced cardiac symptoms during acute exposure to HBO.

Where available, paired samples were compared before and after individual treatments to assess for acute changes in hs-cTnT following exposure to HBOT. Comparison was also made between the 1st and 30th treatment, to assess for chronic changes to hs-cTnT during a course of repeated HBOT exposure.

Results

No statistically significant difference in hs-cTnT was detected either as the result of acute or chronic repeated exposures to HBOT (using paired *t*-test) (Table 1). Repeated measures ANOVA also failed to demonstrate significant changes in hs-cTnT when applied to the 24 subjects who had a complete set of hs-cTnT measurements before their 1st, 10th, 20th, and 30th treatments (Greenhouse-Geisser correction $P = 0.767$).

Discussion

To our knowledge this is the first study to rigorously assess the effect of hyperbaric hyperoxia on baseline myocardial health through measurement of hs-cTnT. This was a cohort of patients with a high prevalence of established IHD, or risk factors for such. This is reflected in the fact that the mean hs-cTnT levels were consistently above the 99th percentile upper reference limit of the Roche Elecsys assay.[9]

In this population, our study demonstrated that exposure to profound hyperoxaemia did not induce any measurable cardiac injury at a biochemical level. Equally, there was no reduction in circulating serum hs-cTnT to suggest a cardioprotective effect of hyperbaric hyperoxia. This provides some degree of reassurance as to the cardiac safety of HBOT for patient undergoing routine hyperbaric treatment for non-cardiac pathologies.

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Roche provided discounted assay reagents but had no role in study design or implementation, or in analysis of results.

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Case report

Atypical distally distributed cutis marmorata decompression sickness associated with unconventional use of thermal protection in a diver with persistent foramen ovale

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Abstract

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Cutis marmorata is a mottled, marbling, livedoid rash caused by vascular inflammation and congestion in cutaneous decompression sickness. It may occur during or after ascent due to the formation of bubbles from dissolved nitrogen accumulated throughout the dive. It is strongly associated with the presence of right to left shunts, particularly persistent (patent) foramen ovale (PFO). We report a case of cutis marmorata decompression sickness of an unusual pattern associated with unconventional use of thermal protection (a 'shorty' wetsuit worn over full suit) by a diver with a PFO. The patient also had neurological manifestations of decompression sickness. The distal lower limb pattern of involvement favours the hypothesis that cutis marmorata in humans is likely to be due to bubbles in the skin itself and/or adjacent tissues rather than cerebrally mediated.

Introduction

Scuba and deep-water divers are at risk of decompression sickness (DCS) during or after ascent, due to the formation of bubbles from dissolved nitrogen accumulated throughout the dive. The presence of a persistent (patent) foramen ovale (PFO) allows paradoxical gas embolism, whereby bubbles cross from the venous circulation directly into the arterial circulation, bypassing lung filtration. These bubbles then grow upon reaching vulnerable supersaturated tissues. Intravascular bubbles cause vascular inflammation and congestion which manifests as a myriad of symptoms and signs, one being a rash characterised by the terms cutis marmorata or livedo racemosa;[1–3] a cutaneous discolouration with characteristic persistent, erythematous or violaceous change, in a broken net-like pattern. The typical mottled, marbling, livedoid rash of cutis marmorata often crosses the midline and is seen in areas rich in subcutaneous fat, namely the chest and abdomen,[4] bilateral thighs,[5] back, buttocks, breast and upper arm.[1]

While there is consensus that bubble formation secondary to dissolved inert gas is involved in the pathogenesis of cutis marmorata DCS, further details pertaining to the pathophysiology remain a bone of contention amongst researchers. There are three main hypotheses for the formation of cutis marmorata:

1. Paradoxical embolism of venous bubbles across a right-to-left shunt followed by augmentation in bubble size on arrival at supersaturated skin via the arterial circulation.[3]
2. *In situ* bubble formation at the skin itself or within its circulation.[1,6]
3. Entry of bubbles to the arterial cerebral or brainstem circulation resulting in a sympathetically mediated vasomotor response.[7]

Many case reports of cutaneous DCS involving the legs state that the typical rash does not extend below the level of the patella.[4,8–10] To our knowledge, it is rare and/or underreported that a cutaneous DCS rash involves the shins. In this case there was an unusual distal rash distribution associated with an unconventional pattern of thermal protection worn by the diver. This may give further insight into the pathophysiology of DCS, in particular cutaneous DCS.

Case report

The patient gave written consent for her case details to be reported.

A 34-year-old previously healthy lady with a history of 35 uneventful dives performed the following two dives on open circuit air. During both dives she wore a 3 mm 'shorty' wetsuit over a 5 mm full suit.

1. Dive 1: maximum depth 38.1 metres of sea water (msw), absolute bottom time (ABT) 12 min and total time of dive (TTD) 32 min followed by a surface interval (SI) of 1 hour 43 min. The water temperature was 17ºC.
2. Dive 2: maximum depth 37.8 msw, ABT 12 min and TTD 44 min, water temperature 17ºC. She noted an erythematous, lacy rash present below the knees bilaterally which improved on surface oxygen. She therefore did not seek medical advice.

The following day she performed another two dives on open circuit air using the same wetsuit configuration.

1. Dive 1: maximum depth 31.3 msw, ABT 27 min and TTD 56 min followed by a SI of 1 hour 31 min, water temperature 20ºC.
2. Dive 2: maximum depth 30.4 msw, ABT 19 min and TTD 46 min, water temperature 24ºC.

All dive profile information (Figure 1) was sourced from the diver's Ratio iDive dive computer (Buhlmann ZHL-16 B decompression algorithm). The dive profile analysis included the computation of the surfacing gradient factor (GF) values while taking into account residual gas loadings, which for the four consecutive dives yielded: 0.64, 0.60, 0.88, and 0.83 respectively.

For all dives, the diver used a 5 mm full wetsuit. On top of the full suit she also wore a 3 mm 'shorty' which terminated just above her knees and elbows. Five minutes after surfacing from the second dive she claimed to have rushed to get an oxygen cylinder for another diver after which she complained of sudden bilateral thigh pain which progressed to both arms. She also noted lethargy and according to her dive buddy an erythematous, lacy rash was present on both legs below the lower third of the thigh and lower back.

Upon review at the emergency department, the diver had a good volume regular pulse at 70 beats per minute and a blood pressure of 110/52 mmHg. She had a normal cardiorespiratory examination as well as normal power and reflexes in both upper and lower limbs. Gait was normal, however the patient complained of dizziness and nausea on walking. No pronator drift, nystagmus or cerebellar signs were observed but a Romberg's test was positive. The rash was noted on both shins and over the extensor surface of the right knee and the distal right thigh. It consisted of flame-shaped erythematous, blanching, papular lesions (Figures 2 and 3). It did not involve the proximal thighs, abdomen, breasts or trunk. Blood investigations showed no abnormalities apart from neutrophilia (18.11 cells x10^{9}·L^{-1}).

Figure 1

Dive profile data uploaded from Ratio iDive diving computer. A: Dive 1 on day 1; B: Dive 2 on day 1; C: Dive 1 on Day 2; D: Dive 2 on Day 2. msw: metres sea water

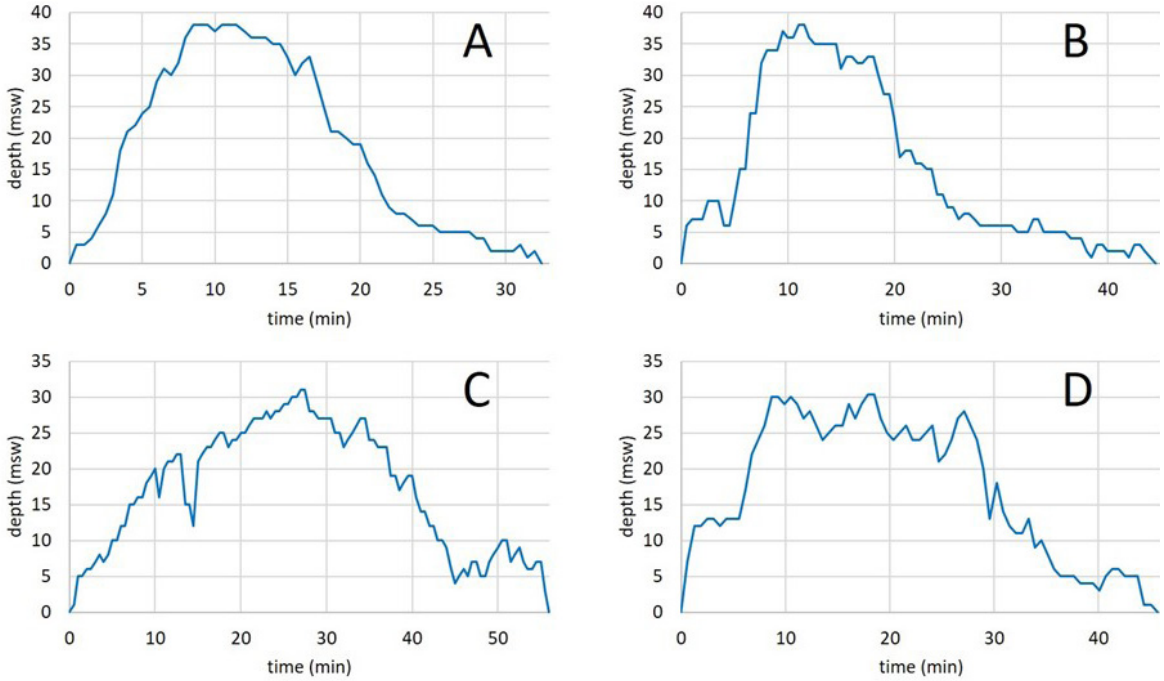


Figure 2
Pre-treatment; right distal thigh and knee with cutis marmorata rash visible

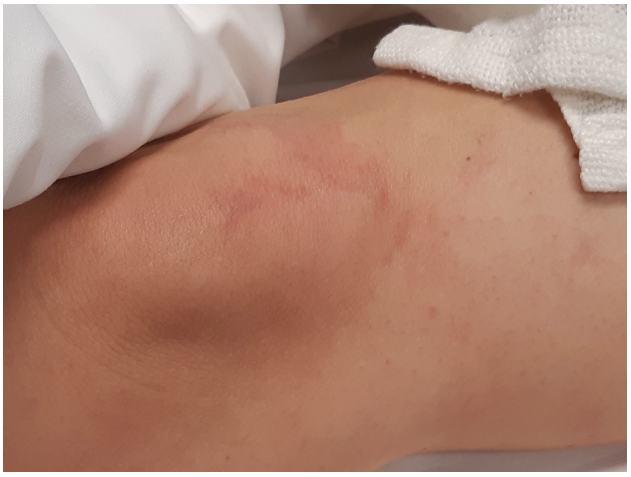


Figure 3
Pre-treatment; left shin bruise and cutis marmorata rash visible

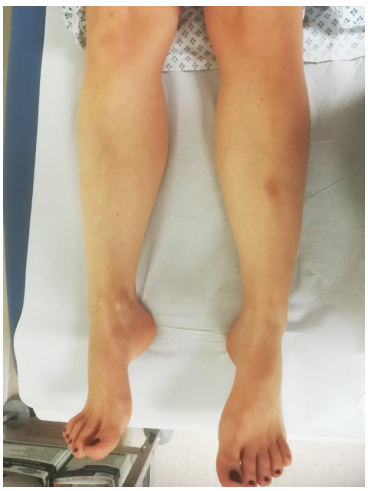
Hartmann's solution and normal saline were given, together with 100% oxygen via demand-valve mask. The patient was treated on US Navy treatment table 6 (USN TT6), initiated on oxygen at 284 kPa. After around 15 minutes, the rash improved and the patient was able to walk around the chamber during the second air break without any dizziness or nausea. A negative sharpened Romberg test was elicited.

The patient was sent home, where she later noted a return of the bilateral thigh pain and lethargy. On examination the following morning, a sharpened Romberg test was positive. The left shin manifested an oval shaped, erythematous, blanching, macular lesion measuring approximately 4 cm by 2 cm (Figure 4). She received follow-up hyperbaric treatment (Royal Navy 60).

Upon review a day later, the patient reported feeling much improved. She reported mild generalised aches and pains in both thighs, left shoulder and upper arm not requiring analgesia. Her gait was normal, a sharpened Romberg test was negative, and she had full power in both arms and legs.

A further follow-up hyperbaric treatment was given (US Navy treatment table 9) with further improvement of symptoms. The residual left shin oval-shaped macular lesion showed skin colour changes consistent with that of a bruise. A transthoracic echocardiogram with agitated saline bubble contrast study was carried out four weeks later. Right–to-left shunting of bubbles was observed both at rest as well as after a Valsalva manoeuvre, suggestive of a PFO. The examination was otherwise within normal limits.

Figure 4
Post-treatment; left shin bruise still visible, no further cutis marmorata rash



Discussion

We describe a case of cutis marmorata DCS demonstrating an atypical distal distribution of the irregular, mottled, erythematous rash in a patient with a right-to-left shunt. The rash was observed over both shins and the extensor surfaces of the right distal thigh and knee. The thermal protection employed by the diver consisted of a 3 mm 'shorty' neoprene wetsuit worn over a 5 mm neoprene full wetsuit. Analysis of the diving profiles indicated that the dives on the day of the incident were provocative, reaching a surfacing GF of 0.88. A recent study analysing Divers Alert Network (DAN) database diving data reported maximum GF-values of 0.70–0.90 in the majority of DCS cases.[11]

Published studies demonstrate that the diving wetsuit exerts various effects on human body physiology. In a non-immersed study, wetsuit wearing was associated with decreases of heart rate and cardiac output, vital capacity and expiratory reserve volume, possibly secondary to the compressive effect exerted on the chest.[12] A tight-fitting wetsuit increases mean arterial pressure and affects heart rate variability resulting in a decrease in the LF/HF ratio

(low and high frequency ratio).[13] It may also compress deep limb veins and peripheral blood vessels, resulting in central pooling of venous blood and an increase in cardiac preload.[14] Wetsuit use has been suggested as a predisposing factor for swimming induced pulmonary oedema[15] and it influences hydromineral homeostasis resulting in increased urine output both during scuba diving and in dry conditions.[16]

To our knowledge, there are no studies linking patterns of wetsuit use to specific forms of DCS. We hypothesise that in our diver, at some point during the scuba dives, the shorty neoprene wetsuit exerted a tourniquet effect at the level of the distal third of the thighs. It appears plausible that the distribution of the rash was a result of impaired off-gassing of inert gas due to reduced perfusion in the distal thigh and leg tissues during ascent.

The tourniquet effect may have been established the moment the wetsuits were worn on the surface. However, as a diver descends, bubbles in the neoprene rubber are compressed, resulting in a looser wetsuit during the deep part of the dive and probable reversing of the tourniquet effect. During experimental research, Bühlmann observed that if limb circulation, for example of an arm, is compromised by the cuff of a sphygmomanometer during decompression that is critical for the skin, red spots and swelling may develop on the arm.[17] In this patient, reversal of the tourniquet effect in the early phase of the dive due to neoprene compression may have permitted sufficient on-gassing of distal thigh and leg tissues. During ascent, a return of the tourniquet effect in the shallows may have impaired off-gassing of the distal lower limb tissues. Moreover, enhanced levels of vasoconstriction due to cold exposure towards the latter stages of the dive may have further hampered off-gassing of inert gas during ascent. Cold exposure during diving is one of the mechanisms which contributes to the dive reflex and results in peripheral vasoconstriction.[18,19]

It is well-established that cutis marmorata DCS is associated with an intra-cardiac right-to-left shunt.[2,3,6] Several mechanisms for its pathophysiology have been proposed and have been mentioned above. There has been debate on whether the rash could also be cerebrally mediated following a study in swine. Internal carotid artery gas injection in anaesthetised pigs was followed by the appearance of a cutis marmorata mottled rash similar in appearance to cutis marmorata / livedo racemosa.[7] Notably, the 30–40 kg pigs were injected with 0.25 and 1 ml·kg^{-1} air producing a sympathetic surge similar to that noted in subarachnoid haemorrhage and other catastrophic brain injuries. Most human patients with cutaneous DCS have no neurological symptoms and if they do, they are rarely catastrophic.[20] Moreover, a recent human study using ultrasound technique confirmed the presence of bubbles in skin microcirculation affected by cutis marmorata.[1] This adds more weight to the mechanism of cutis marmorata in human cases being non-cerebrally mediated.

The case described in this paper illustrates that bubble amplification is likely occurring in the skin and surrounding tissues themselves, rather than the rash being cerebrally mediated. It is thought that the unusual use of thermal protection resulted in supersaturated tissues distal to the level of the shorty wetsuit. This resulted in either in situ bubble formation or local augmentation of paradoxical gas emboli arriving in the distal lower limbs.

Of note, this diver also complained of dizziness, observed at the emergency department shortly after gait assessment. While this symptom may have been due to non-documented postural hypotension, the Romberg's test was positive indicating likely vestibular DCS which is also strongly associated with a PFO.[21] These findings are further in-keeping with paradoxical air embolism with subsequent bubble enlargement at the legs as the most likely mechanism of cutis marmorata formation in this case.

Conclusions

Cutaneous DCS is a well-documented condition strongly associated with the presence of a right-to-left shunt. The distal lower limb pattern of involvement and other circumstances of this case favour the hypothesis that cutis marmorata in humans is likely to be due to bubbles in the skin itself and/or adjacent tissues rather than cerebrally mediated. It illustrates how thermal protection employed by the diver may influence the distribution of a cutis marmorata rash in a diver with a right-to-left shunt.

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Letters to the Editor

Commentary on Plogmark, et al. Agreement between ultrasonic bubble grades using a handheld self-positioning Doppler product and 2D cardiac ultrasound

We read with great interest the paper "*Agreement between ultrasonic bubble grades using a handheld self-positioning Doppler product and 2D cardiac ultrasound*".[1] We agree with authors general conclusion regarding a lower sensitivity to microbubbles, especially to the small ones, of the subclavian Doppler sensor O'Dive when compared to 2D medical echocardiography with a premium level echograph. However, we found this conclusion incomplete, because it lacked the analysis of observed decompression sickness (DCS) in relationship to venous gas emboli (VGE) scores.[2]

The authors recorded VGE with the O'Dive for 152 controlled experimental air dives in a hyperbaric chamber in addition to medically diagnosed DCS (the study's primary endpoint) and VGE measured using echocardiography (the study's secondary endpoint). Courtesy of the authors we have accessed paired data of O'Dive VGE grades blindly scored by Azoth Systems and medically diagnosed DCS (of which Azoth Systems was not aware). The analysis of such medical data is of a great interest to divers and is rare because the O'Dive is designed for self-positioning and autonomous use by recreational or professional divers without any medical assistance.

For the purpose of analysis, we used a classical binary cut-off to convert each dive's maximum O'Dive grade into 'high bubble grade' (HBG, grades 3 to 4, n = 40) versus 'low bubble grade' (LBG, grades 0 to 2, n = 112) and investigated the association between O'Dive's HBG and DCS status. Firstly, we considered only oxygen-treated DCS cases (n = 6, including three cases necessitating hyperbaric treatment), then we enlarged the analysis to all possible DCS cases (n = 13, including untreated minor cutaneous stress and one doubtful case). The data used in both analyses are summarised in Table 1.

Fisher's exact test confirmed a significant association between HBG and DCS for both treated DCS ($P < 0.001$) and all DCS ($P < 0.001$). All treated DCS cases (n = 6) were associated with O'Dive HBG, with a 15% treated DCS rate for HBG (n = 40) and no treated DCS for LBG (n = 112). When including minor cutaneous stress DCS, nine were associated with O'Dive HBG (22.5% DCS for HBG) and four were associated with O'Dive LBG (3.6% DCS for LBG). These DCS rates are comparable with those found in the literature for conventional echocardiography HBG[3] or Doppler HBG[4].

O'Dive HBG had both high specificity (77% for treated and 78% for any cases) and sensitivity to DCS (100% for treated and 69% for any cases). The positive predictive values of HBG were low (15% for treated DCS and 23% for any DCS), which are typical for VGE which are considered an unsuitable parameter for DCS diagnosis, but an important risk indicator. In contrast, the negative predictive values of LBG were high (100% for treated DCS and 96% for any DCS).

We stress that VGE gives only partial information regarding DCS risk,[5] which is why the O'Dive application computes a 'dive quality index' (QI, ranging from unsafe 0% to safe 100%) integrating information from both the dive profile and maximum VGE grade. Unfortunately, we could not analyse the association between QI and DCS in the present study because the authors did not communicate the dive profiles.

However, VGE can drive QI, as illustrated by the case of an O'Dive user diagnosed with cutaneous DCS after a 44 msw CCR dive, with QI = 59% and VGE grade 4 (described with permission of the user and his medical doctor P Germonpré).

These results, while beyond the original scope of the article,[1] are of interest for divers and they confirm that higher bubble grades (as measured by the O'Dive) seem associated with a higher risk of DCS.

Table 1

DCS cases versus high (HBG) or low bubble grades (LBG) from O'Dive (based on data provided by Dr O Plogmark, published with permission of the authors)

Parameter	Oxygen-treated DCS n = 6	Oxygen-treated DCS rate	Any DCS n = 13	Any DCS rate
O'Dive LBG n = 112	0	0%	4	3.6%
O'Dive HBG n = 40	6	15%	9	22.5%

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Asya Metelkina and Axel Barbaud work for Azoth Systems, the company who manufactures the O'Dive Doppler sensor and application.

Keywords
Decompression sickness; Doppler; Risk Factors; Risk Management; Scuba diving; Venous gas emboli

Response to Metelkina and Barbaud

We welcome this letter, which presents supplementary statistics regarding the association between intravascular bubbles detected using a handheld self-positioning Doppler and decompression sickness (DCS).

The present dataset is relatively small, 152 dives, and with only three DCS cases treated with hyperbaric oxygen (HBO). However, despite the modest size, this dataset holds the potential to make meaningful contributions to larger analyses with pooled data. It is important to underscore that, within the context of such larger analyses, the inclusion of solely HBO-treated DCS cases is recommended due to our observation of a problematic diagnostic variability among cases treated with normobaric oxygen.

We believe that future studies aiming to evaluate methods for predicting the risk of DCS, whether based on grading of intravascular bubbles or involving other algorithms, should use receiver operating characteristic (ROC) and compare the area under the curve (AUC). These analyses can then be utilised to select methods, with appropriate cut-offs for different pre-test probabilities, for research or diagnostics.

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Carbon monoxide poisoning: lest we forget

With reference to the paper by Lippman, et al. 'Compressed gas diving fatalities in Australian water 2014–2018.[1]

Of the 42 deaths reported three were due to immersion pulmonary oedema (IPO) and three to carbon monoxide poisoning (COP). It is acknowledged that both conditions often go unrecognised.[2-4]

A lot of attention is currently, rightly being directed at IPO with COP often forgotten so these data are a good reminder.

The deepest dive in this series was to 39 metres sea water. The risk of COP increases with depth where even very small amounts of carbon monoxide (CO) contamination can cause toxicity.

Admittedly in the current report, two of the three COP deaths relate to surface-supplied breathing apparatus (SSBA) that is recognised to have a higher risk of COP. Nonetheless, gas cylinders analysis was rather alarming. Of the 20 cases reported, the results indicated 25% did not meet accepted air purity standards. With elevated water vapour and potentially lethal levels of CO and CO_2 being other contaminants identified.[1]

The Divers Alert Network (DAN) has published a safe standard for CO as less than 5 ppm for routine dives and advocated that levels should be lower for deep technical diving.[5] Producing nitrox by either gas separation or pressure swing absorption has the potential to concentrate any CO present in the original air into the final nitrox gas mix.[5]

Additionally, it is less well known, CO can be produced within a compressor as a result of the breakdown of the lubricant oil caused by heat (chemical decomposition or pyrolysis) this may occur when the system is hot but not necessarily overheating and it may be short term and thus missed by periodic examination of the gas sample.[6]

We agree with the authors that CO alarms are important and that meticulous quality control in gas supplies is vital to avoid contamination.

Portable handheld CO detectors are especially helpful particularly for deep technical dives as many dive sites and cylinder refills are in remote locations.

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Conflicts of interest and funding: nil

Keywords
Diving; Fatality; Gas supply; Scuba; Surface supply; Toxicity

Notices and news

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

SPUMS President's report

Neil Banham

The 52nd SPUMS Annual Scientific Meeting (ASM) will be held at the Pearl Resort, Pacific Harbour, Fiji from Sunday 12 May – Friday 17 May 2024. Registration will be possible by the time you read this.

Conference theme: *A plunge into recreational diving and diver health*

Convenors: David Smart and Neil Banham (Scientific Convenor)

The Pearl Resort offers direct access to Beqa Lagoon with its famous soft corals and shark feeding dives. The diving will be organised by Diveplanit, who coordinated the diving at our 2023 Cairns ASM and who have experience in conference diving planning at the Pearl Resort.

Our Keynote Speaker will be Dr Peter Wilmshurst, a British cardiologist who some will remember from our highly successful 2014 Bali ASM, which culminated in the publication of the SPUMS and the United Kingdom Sports Diving Medical Committee (UKSDMC) Joint Position Statement (JPS) on persistent foramen ovale (PFO) and diving in 2015.[1] Peter is a world authority on PFO and diving as well as immersion pulmonary oedema (IPO), reporting the first case.

Workshops will be held with a view to update the JPS on PFO and diving and to develop one for return for diving (or not) following an episode of IPO.

Supporting speakers include Dr John Lippmann and Professor Simon Mitchell, both very knowledgeable and engaging speakers. Simon will present an update on decompression illness and John will talk on diving accidents.

Registration and further information can be found at: https://spums.au/index.php/asm-registration.

A form for submitting an Abstract is available by following this link: https://spums.au/index.php/submission-of-abstracts.

On behalf of SPUMS, I sincerely thank David Smart for again taking on the Convenor role. David and our Web Manager Nicky Telles have already spent countless hours working on the next ASM as well as updating the SPUMS website to make organisation of future ASMs much easier.

Deliberations are continuing as to the venue for our 2025 ASM. Those with suggestions for a suitable location or who wish to assist with convening this are welcome to contact me.

The ANZHMG Introductory Course in Diving and Hyperbaric Medicine will be next held 19th February – 1st March 2024, again in Fremantle, link to the course details: https://spums.au/index.php/education/spums-approved-courses-for-doctors.

Scholarships for trainees to attend this course are available thanks to the generosity of the Australasian Diving Safety Foundation. Please contact John Lippmann at johnl@adsf.org.au for more information.

Dr Neil Banham
SPUMS President

Reference

1 Smart D, Mitchell S, Wilmshurst P, Turner M, Banham N. Joint position statement on persistent foramen ovale (PFO) and diving. South Pacific Underwater Medicine Society (SPUMS) and the United Kingdom Sports Diving Medical Committee (UKSDMC). Diving Hyperb Med. 2015;45:129–31. PMID: 26165538.

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

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

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

SPUMS

South Pacific Underwater Medicine Society

52nd Annual Scientific Meeting

FIRST NOTICE – SAVE THE DATES

Sunday 12 May – Friday 17 May 2024

The Pearl Resort, Pacific Harbour Fiji

THEME:

A plunge into recreational diving and diver health.

KEYNOTE SPEAKER:

Dr Peter Wilmshurst

UK Cardiologist and world authority on patent foramen ovale and diving and immersion pulmonary oedema

Registrations open 01 September 2023

https://spums.au/index.php/asm-registration

The Australian and New Zealand Hyperbaric Medicine Group

Introductory course in diving and hyperbaric medicine

Dates: 19 February – 01 March 2024
Venue: Hougoumont Hotel, Fremantle, Western Australia
Cost: AUD$3,200.00 (inclusive of GST) for two weeks

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

The course content includes:

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

Contact for information:

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

HBOE
HBOEvidence

HBOEvidence

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

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

Royal Australian Navy Medical Officers' Underwater Medicine Course

Date: 16–27 October 2023, 18–29 March 2024
Venue: HMAS Penguin, Sydney

Cost: The course cost remains at AUD$1,355.00 (excl GST).

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.

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*

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SPUMS on Facebook

ADSF
AUSTRALASIAN DIVING SAFETY FOUNDATION

An Australian Health Promotion Charity encouraging the prevention and control of diving related illness and injury through Research or Diving Safety Promotion Grants.

APPLY FOR A GRANT NOW
www.adsf.org.au

SPUMS Diploma in Diving and Hyperbaric Medicine

Requirements for candidates (May 2014)

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

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

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

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

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

Additional information – prospective approval of projects is required

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

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

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

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

Projects will be deemed to have lapsed if:

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

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

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

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

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

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

Notices and news

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

EUBS Notices and news

EUBS Member-at-Large elections

The EUBS Elections were again held electronically, using the ElectionRunner software, to elect one Member-at-Large for the four-year period 2023–2027. There were three candidates: Dr Michal Hajek from Czech Republic, Dr Pedro Barata Coelho from Portugal and Dr Kurt Magri from Malta. The candidate who scored best was Michal Hajek, and he will take office as from our General Assembly on 16 September 2023.

We will be saying goodbye to our 2020 Member-at-Large, Dr Oscar Camacho, during 'his' EUBS2023 Scientific Meeting. We thank Oscar for his efforts for our Society and hope be able to count on him in the future.

Thanks to all EUBS members who have voted, if you have any comments on the voting process or software used, please send us an email at: secretary@eubs.org.

EUBS2023 Annual Scientific Meeting, Porto, Portugal, from 13–16 September 2023

As this issue of *Diving and Hyperbaric Medicine* journal is published, we will have had the pleasure to unite again in Porto, from 13–16 September 2023. While this text is written before the meeting, we are certain it will have been a great pleasure to see our friends again, and we are confident that the 47th Annual Scientific Meeting of EUBS will have been a great success.

Next year, the EUBS meeting will be in Brest, France, from 16–20 September 2024, so please keep these dates free in your busy agendas. Also, plan to have some days off before and after the conference to enjoy the Breton Peninsula, and maybe do some diving?

EUBS General Assembly

This is a formal invitation to participate in our EUBS Annual General Assembly, which will take place during the EUBS Annual Scientific Meeting, on Saturday 16 September from 9.00 to 10.00 am, in the main conference hall. The agenda will be customary to discuss all items relevant to the function of the Society, as discussed by ExCom during their meeting on Tuesday 12 September and will be posted on the information board. All EUBS members with voting rights are cordially invited.

EUBS website

As always, please visit the EUBS website (www.eubs.org) for the latest news and updates. Do not forget to renew your membership annually – each member will receive a personal renewal invitation one month before expiry; even if your membership has expired, you can easily renew it when trying to log in again. In case of problems, do not hesitate to contact the EUBS secretary at secretary@eubs.org.

EUBS website and OXYNET

Occasionally, we use the EUBS website newsletter as a tool to seek help for our members, it is a perfect way to reach all of the EUBS members and because communication, networking and interaction are prime goals of our Society.

The OXYNET database of hyperbaric centres is presented as an interactive map page on the EUBS Website. ExCom is looking for member in each country help us to keep the database up to date – let us know if you are willing to help.

A Help Requests page on our EUBS website has been created (EUBS Members Help Requests, under the 'Activities' menu on the homepage). Please check this page and try to help. In case you need help as well and would like to use this service, please contact the webmaster (webmaster@eubs.org).You should also consult the page (http://www.eubs.org/?page_id=284) where research projects seeking collaborators and international participation are presented.

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.

Courses and meetings

Scott Haldane Foundation

As an institute dedicated to education in diving medicine, the Scott Haldane Foundation has organized more than 300 courses all over the world, over the past 31 years. SHF is targeting on an international audience with courses world wide.

Below the schedule of upcoming SHF-courses in 2023 and first half of 2024.

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

2023

4–11 November	Medical Examiner of Divers part 1 (level 1) Manado, Indonesia
11–18 November	In-depth course Brain under pressure (level 2d) Manado, Indonesia
18–25 November	In-depth course Brain under pressure (level 2d) Manado, Indonesia

2024

27 January	Refresher course Diving Medical in Practice Bunnik, The Netherlands
5–6 April	Medical Examiner of Divers part 1 (level 1) Zeist, The Netherlands
11–13 April	Medical Examiner of Divers part 2 (level 1) Amersfoort, The Netherlands
11–18 May	Medical Examiner of Divers part 2 (level 1) Bonaire, Dutch Caribbean
14–15 June	In-depth course Brain under pressure (level 2d) Putten, The Netherlands
On request	Internship HBOt (level 2d) NL/Belgium

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

Joint Scientific Conference of British Hyperbaric Association and UK Dive Medical Committee 2023

Thursday 2 November 8:45 am – Saturday 4 November 2023 5:00 pm

This is a hybrid conference, where virtual/online participation, presentation will be possible. The conference venue is the Museum of London Docklands in Canary Wharf and will provide an environment where discussion, debate, learning and development will thrive.

For any queries please contact pkarpati@gmail.com or liz@londonhyperbaric.com.

Looking forward to seeing you there.

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.

Historical Diving Society Australia - Pacific

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

The Science of Diving

Support EUBS by buying the PHYPODE book '*The science of diving*'. Written for anyone with an interest in the latest research in diving physiology and pathology. The royalties from this book are being donated to the EUBS.

Available from:
Morebooks
https://www.morebooks.de/store/gb/book/the-science-of-diving/isbn/978-3-659-66233-1

Diving and Hyperbaric Medicine: Instructions for Authors

(updated February 2023)

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

***Address*:** The Editor, Diving and Hyperbaric Medicine, Department of Anaesthesiology, University of Auckland, Private Bag 92019, Auckland 1142, New Zealand
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***Phone: (mobile)*:** +64 (0)27 4141 212
***European Editor*:** euroeditor@dhmjournal.com
***Editorial Manager*:** editorialassist@dhmjournal.com
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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 username and password (keep a record of these for subsequent use). The process of uploading the files related to the submission is simple and well described in the on-screen help provided 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 the word count); include an informative **Abstract** of no more than 300 words (excluded from the total word count); structure of the article and abstract is at the author(s)' discretion.

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

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

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

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

Formatting of manuscripts

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

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 2023 (this document)
DHM Keywords 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

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

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+64-9-445-8454 (International)

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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.
