Diving and Hyperbaric Medicine

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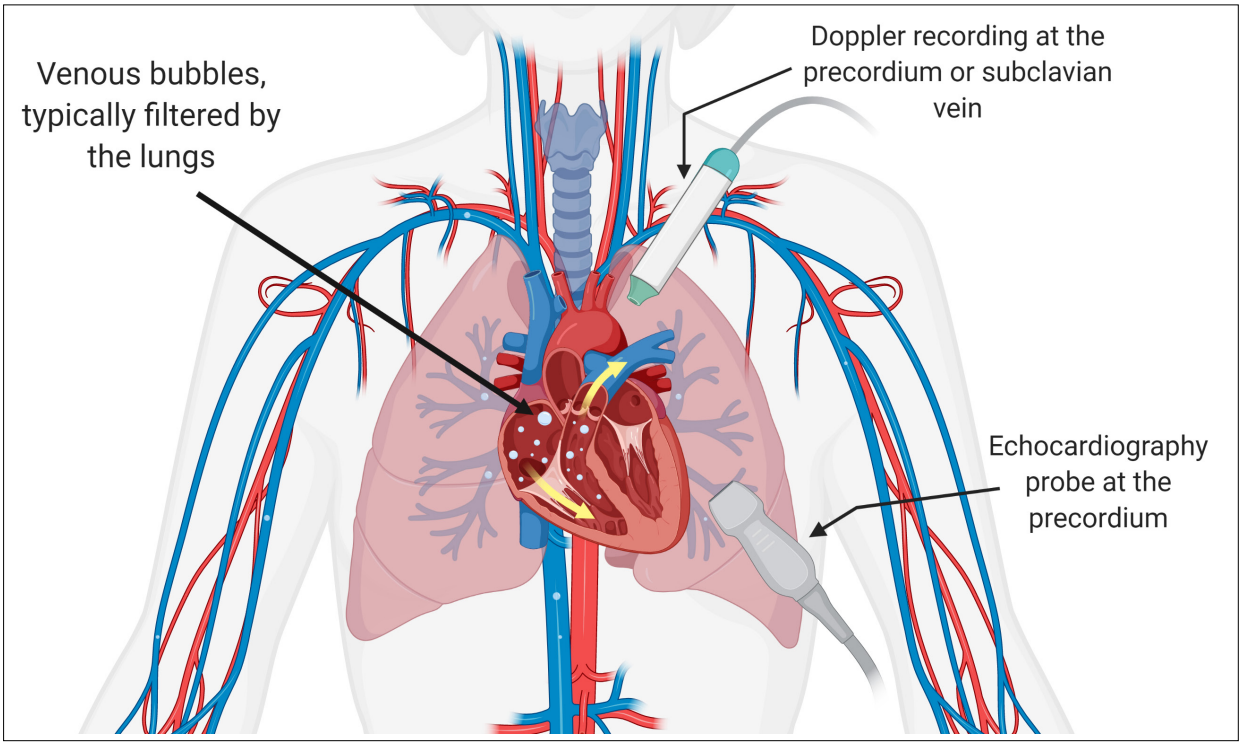

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PURPOSES OF THE SOCIETIES

To promote and facilitate the study of all aspects of underwater and hyperbaric medicine
To provide information on underwater and hyperbaric medicine
To publish a journal and to convene members of each Society annually at a scientific conference

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DIVING AND HYPERBARIC MEDICINE

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The Editor's offering

This second issue of *Diving and Hyperbaric Medicine* (DHM) in 2022 contains some high quality and important articles of interest to those on both 'sides' of the field. On the diving side there is only the second fully published randomised study of any intervention in decompression sickness (DCS). It compared shorter versus longer 284 kPa recompressions in treatment of mild DCS, and found the shorter table just as (perhaps even more) effective in relation to tempo of recovery. Although the final outcomes were no different, as would largely be expected no matter what treatment is provided in mild DCS, these results will inform discussions about recompression options in mild cases.

A second important and fascinating diving study surveyed active Finnish technical divers about DCS symptoms and the divers' responses to them over a one-year period. This study sheds light on what technical divers have known for some time; that the incidence of mild DCS symptoms is high among this craft group, and that self-treatment or even no treatment and no consultation with medical authorities is common. The study reported the natural history of 26 mild DCS cases that were not treated with hyperbaric oxygen; all fully recovered. This is supportive of the interpretation of that natural history in the 2005 and 2018 consensus on treatment of mild DCS.[1] However, as codified in that consensus and as the authors of the present paper point out, consultation with diving medicine experts and appropriate first aid (at the very least) should be part of managing such cases.

Other diving-related papers include a survey of rebreather divers to determine the proportion of respondents who had experienced a 'caustic cocktail' during diving. Perhaps most significantly, a need to improve education about appropriate first aid was identified. Also of relevance to technical divers was a study that measured the pressure exerted on the wrist and neck by seals on drysuits worn by real-world divers. At least some wrist seals exerted pressures in a range that might cause nerve injury. There is a comprehensive analysis of diving fatalities over 10 years to 2019 in Queensland, Australia's busiest diving state. Most fatalities occurred in supervised snorkelling activities. This reflected, at least in part, the large numbers of participants in such activities, but also a need to improve certain dive practices and optimise pre-participation health screening. There is a fascinating study on haemodynamic responses to administration of vasoactive drugs in rats selectively bred for resistance to DCS. Although there is some way to go, this study is indicative of ongoing efforts to identify the phenotypic features of DCS resistance. We are all watching that space with great anticipation. There is a study of the effect of pressure changes consistent with diving on the shear bond strength of different cement compounds use to attach orthodontic brackets to teeth. As is always encouraged in DHM, the authors were able to make some practical recommendations for application in divers. This issue's review article is a comprehensive account of ultrasound bioeffects of potential relevance to prolonged post-dive monitoring of venous gas emboli.

On the hyperbaric medicine side there is an update to a living systematic review published in the third issue in 2021, on the use of hyperbaric oxygen in treatment of severe COVID-19. This update includes the first two randomised trials published. It remains to be seen to what degree interest in this matter propagates as the pandemic wanes, but hospitals will be admitting sick COVID-19 for a while yet and in locations with access the question of the efficacy HBOT will be highly relevant, as will questions of safety. Commentary on the latter needs to be cautious and proportional to the published experience. This editor noted various optimistic pronouncements about 'safety' at the recent UHMS meeting, but on the basis of a total published experience of treating 114 patients (to date), nothing definitive can be claimed about safety. Finally, on the hyperbaric side, there is an interesting series of cases in a rare 'problem wound' arising from Nicolau Syndrome, a complication of injection of drugs intramuscularly.

Sadly, I must close this editorial with acknowledgement of the recent loss of two revered colleagues. First, Professor Alf Brubakk (Norway), a true senior statesman and leading academic in our field, passed away at age 81 in April. I am grateful for the obituary written by Dr Michael Lang which appears in this issue, and for permission from the editor of Undersea and Hyperbaric Medicine to reproduce it. Second, at a much earlier stage of her career, we tragically lost Dr Cecilia Roberts (South Africa) at age 43 after a car accident in May. Cecilia was an engaging, starbright woman whose infectious enthusiasm for our field was known worldwide. An obituary for Cecilia will appear in the September issue of DHM.

Professor Simon Mitchell
Editor, Diving and Hyperbaric Medicine Journal

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1 Mitchell SJ, Bennett MH, Bryson P, Butler FK, Doolette DJ, Holm JR, et al. Pre-hospital management of decompression illness: expert review of key principles and controversies. Diving Hyperb Med. 2018;48:45–55. doi: 10.28920/dhm48.1.45-55. PMID: 29557102. PMCID: PMC6467826.

Front cover: Schematic depiction of approaches to ultrasound-based quantification of decompression bubbles. Figure 1 from McCune, et al. in this issue.

Original articles

Decompression illness in Finnish technical divers: a follow-up study on incidence and self-treatment

Laura J Tuominen[1,2], Sofia Sokolowski[3], Richard V Lundell[1,4], Anne K Räisänen-Sokolowski[1,4,5,6]

[1] *Helsinki University, Helsinki, Finland*
[2] *Department of Anaesthesia, Tampere University Hospital, Tampere, Finland*
[3] *University of Eastern Finland, Kuopio, Finland*
[4] *Diving Medical Centre, Centre for Military Medicine, Finnish Defence Forces, Helsinki, Finland*
[5] *Department of Pathology, HUSLAB, Helsinki University Hospital, and Helsinki University, Helsinki, Finland*
[6] *DAN Europe Foundation, Finnish Division, Roseto, Italy*

Corresponding author: *Dr Laura Tuominen, Helsinki University, Yliopistonkatu 4, 00100 Helsinki, Finland*
tuominenl@gmail.com

Keywords
Cold; Decompression sickness; First aid oxygen; Epidemiology; Hyperbaric oxygen treatment; Technical diving; Trimix

Abstract
(Tuominen LJ, Sokolowski S, Lundell RV, Räisänen-Sokolowski AK. Decompression illness in Finnish technical divers: a follow-up study on incidence and self-treatment. Diving and Hyperbaric Medicine. 2022 June 30;52(2):78–84. doi: 10.28920/dhm52.2.78-84. PMID: 35732278.)
Introduction: Technical diving is increasing in popularity in Finland, and therefore the number of decompression illness (DCI) cases is also increasing among technical divers. Although hyperbaric oxygen treatment (HBOT) remains the standard of care, there are anecdotal reports of technical divers treating mild DCI symptoms themselves and not seeking a medical evaluation and possible recompression therapy. This study aimed to make an epidemiologic inventory of technical diving-related DCI symptoms, to establish the incidence of self-treatment and to determine the apparent effectiveness of different treatment methods.
Methods: A one-year prospective survey with online questionnaires was conducted. Fifty-five experienced and highly trained Finnish technical divers answered the survey and reported their diving activity, DCI symptoms, symptom treatment, and treatment outcome.
Results: Of the reported 2,983 dives, 27 resulted in symptoms of DCI, which yielded an incidence of 91 per 10,000 dives in this study. All of the reported DCI symptoms were mild, and only one diver received HBOT. The most common self-treatments were oral hydration and rest. First aid oxygen (FAO_2) was used in 21% of cases. Eventually, none of the divers had residual symptoms.
Conclusions: The incidence of self-treated DCI cases was 27 times higher than that of HBO-treated DCI cases. There is a need to improve divers' awareness of the importance of FAO_2 and other recommended first aid procedures and to encourage divers to seek medical attention in case of suspected DCI.

Introduction

Scuba diving in Finland can be challenging due to poor visibility and chilling water temperatures. Year round, the temperature is 4°C at depths below 30 metres. Furthermore, during the winter months, the surface water temperature is nearly freezing and varies from -1 to 2°C. Abandoned mines with crystal clear water and deep passages have become very popular dive sites instead of murky lakes. However, deep passages mean deep diving, which in turn requires demanding technical training. Technical divers use advanced equipment and mixed breathing gases, such as nitrox or trimix, in order to do dives that are deeper and/or longer than recreational dives. Furthermore, deep dives lead to long exposures in cold water. Despite these challenging conditions, Finnish divers commonly perform decompression dives. Figure 1 shows the crystal clear, but cold 4°C water found in Finnish mines.

It is known that deep trimix dives and cold are important risk factors for decompression illness (DCI).[1,2] The average number of recompressed DCI patients in Finland is 29 per year (range 16–38).[3] An increasing number of cases with technical divers has been described over the years, reflecting the increasing popularity of technical diving in the Finnish diving community.[3] Anecdotal reports of technical divers treating mild DCI symptoms themselves, or even denying the symptoms and not seeking a recompression facility, are not unusual. However, there are no data describing how often this occurs, how severe the cases are, and how cases are self-treated or managed.

Figure 1
Finnish technical divers at a crushing station at 138 metres of fresh water in the Montola mine, Finland; photo by Patrik Grönqvist

Recompression in a chamber to facilitate hyperbaric oxygen treatment (HBOT) is the standard care for DCI. Nonetheless, given the favourable natural history of 'mild' DCI[4] it has been suggested that some mild DCI cases might be managed without recompression, especially if it is difficult or dangerous to access as is often the case in remote locations. The consensus guideline for pre-hospital management of DCI from 2018 defines mild symptoms as musculoskeletal pain, rash, constitutional symptoms, subcutaneous swelling, and some cutaneous sensory changes.[4] Even in apparently mild cases, significant neurological dysfunction should be excluded by a competent examiner, and designation of a case as mild (and not in need of recompression) should always involve a diving medicine physician.[4,5]

The common practice for early management of DCI is to breathe normobaric first aid oxygen (FAO_2), hydrate orally, lie down in a horizontal position, and keep warm but not hyperthermic. Treatment with a non-steroidal anti-inflammatory drug is also appropriate if there are no contra-indications.[4–6] Furthermore, the use of normobaric FAO_2 increases recompression efficacy and decreases the number of recompression treatments required if given within four hours after surfacing.[7] There is also evidence that diving causes dehydration, which would at least in theory support the role of post-dive hydration.[8]

Another option for early management of DCI is to perform in-water recompression (IWR). One significant advantage of IWR is the ability to treat the diver within a short time frame from symptom onset. However, this method is controversial due to the potential risks and the difficulty in selecting the divers whose condition justifies the risks of IWR.[9] The greatest concern is for central nervous system (CNS) oxygen toxicity and the risk of drowning in case of a seizure. Thus, IWR should only be performed in cases when the patient's safety can be ensured and with appropriate training, equipment, and a full understanding of the necessary procedures.[5,6,9] Technical divers are in a unique position to potentially perform IWR due to their high level training, advanced equipment, good supporting divers and easy access to 100% oxygen. Technical diving is more often done in remote locations and conditions in caves and mines are usually predictable. On the other hand, there is indefinite evidence that a delay in recompression would have a negative effect on the treatment outcome, except in the severe cases.[9,10] Therefore, further studies are needed to address this issue.

The aim of this research was to determine the incidence of technical diving-related DCI symptoms in Finnish divers, to find out if self-treatment occurs, and to determine the effectiveness of different treatment methods. Most of what is known about the incidence of DCI is based on data related to cases requiring hyperbaric treatment.[11–13] In addition, there are only a few prospective and retrospective studies with data on DCI symptoms and treatment outcomes gathered with questionnaires from recreational divers.[14–18]

Methods

Ethical approval was granted by the Ethical Committee of Helsinki University Hospital (HUS/976/2019). The study adhered to the Declaration of Helsinki.

STUDY DESIGN

The study was designed as a prospective longitudinal cohort study. The target group consisted of experienced technical divers who planned to take part in a one-year follow-up carried out with online questionnaires. Participants were recruited from the Finnish recreational technical diving community. Researchers contacted known technical divers at Finnish dive sites and via email. Trained technical divers who perform decompression dives with mixed breathing gases in caves, mines, or wrecks were included in this study. All subjects participated voluntarily and gave their informed consent for the study. The researchers did not examine any of the subjects, and the divers were free to dive according to their usual diving practice.

DATA GATHERING

Three online questionnaires were created on Microsoft Office 365 Forms (Microsoft Corp., Redmond, Washington, USA) under license from Helsinki University Hospital. In order to answer the questionnaires anonymously, the participants were given a research identity code that was used to combine information from different questionnaires. Only the researcher responsible for recruitment (LT) was aware of the identities in order to keep track of the answers given.

Information containing sex, age, and anthropometric data (height, weight) were requested in the first questionnaire (Questionnaire for Demographic Data) (*Appendix 1). Body mass index (BMI) was calculated based on the reported data. Additional information on previous HBOT-treated DCI, the use of nicotine-containing products, and diving history were also requested in this questionnaire.

The second questionnaire (Questionnaire for Diving Activity) (*Appendix 2) collected data on the number of dives, the depth range, and the maximum depth during the one-year follow-up period from 01 July 2020 to 30 June 2021. The divers completed this questionnaire every two months, thus six times during the follow-up period.

The third questionnaire (Questionnaire for DCI Symptoms) (*Appendix 3) collected data about the dives that led to possible DCI-related symptoms, the diver's symptom profile, how these symptoms were treated, and treatment outcome. The divers were instructed to complete the third questionnaire each time symptoms occurred.

STATISTICS

Continuous variables are presented using medians and interquartile ranges (IQRs), while categorical variables are presented using counts and percentages. The divers were divided into two groups: the divers who experienced DCI symptoms ('DCI'); and the divers who did not experience any DCI symptoms ('no DCI') during the one-year follow-up period. The groups were compared using Mann-Whitney U tests for continuous variables and Fisher's exact tests for categorical variables. *P*-values < 0.05 were considered significant. All analyses were done using IBM SPSS Statistics version 27 (IBM Corp, Armonk, NY, USA).

Results

SUBJECTS

Fifty-five volunteers (nine women, 46 men) met the criteria and were included in the study. Three divers declined to participate in this research. All study participants responded to every questionnaire. The average diving experience was 16 years (range 5–51 years). The divers were highly trained: 84% had their highest certification as full cave or equivalent and 75% had full trimix or equivalent; 25% of divers had normoxic trimix or equivalent. Fifty divers (91%) used a closed-circuit rebreather (CCR), and five divers (9%) utilised open-circuit (OC) scuba. Fifteen divers were active instructors who taught technical diving mostly in Finland during this period. There was no statistically significant difference in demographic data between the divers who experienced DCI symptoms (n = 17) and the divers who did not have any symptoms (n = 38). The demographics are shown in Table 1.

Table 1

Description of 55 participants; divers in the DCI group reported at least one dive leading to DCI symptoms. Data are simple numbers or median (IQR). There was no statistical difference between the groups in any parameter. HBO – hyperbaric oxygen

Parameter	DCI n = 17	No DCI n = 38
Male	13	33
Age, years	43 (40–50.5)	47 (40.8–50.3)
Body mass index, kg·m^{-2}	27.1 (24.5–28.7)	26.5 (24.5–28.1)
Smoking	2	4
Previous DCI treated with HBO	6	7
Diving years	18 (8–27)	13 (10–18)
Number of dives	1,000 (682–1,750)	800 (608–1,325)
Rebreather used	15	35
Full trimix or higher	13	27
Full cave or higher	14	32
Instructor	7	8

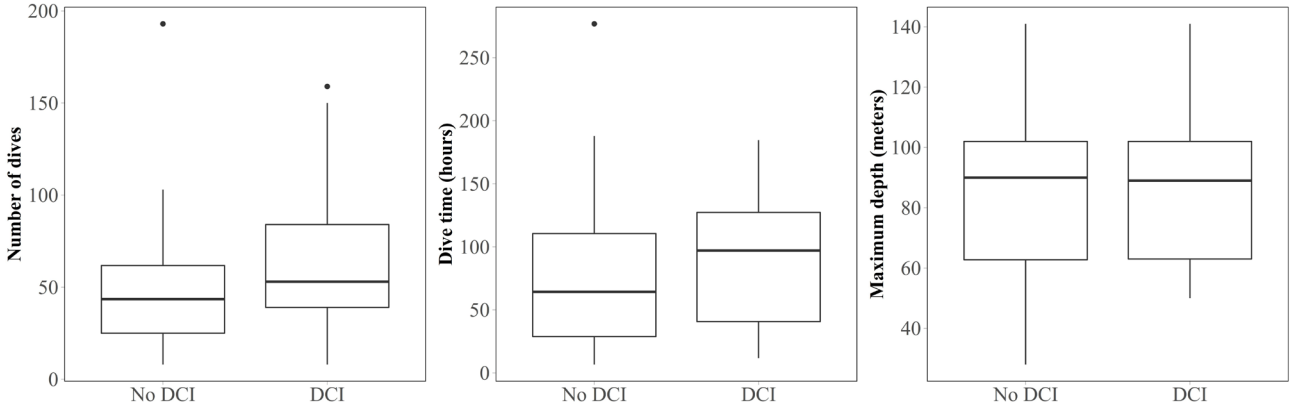
DIVING ACTIVITY

During the one-year follow-up period, the divers performed a total of 2,983 dives and 4,554 hours of dive time. Of these dives, 1,200 (40.2%) or 1,911 hours of dive time (42%), were done during the colder winter months (November to April). The maximum depth reached was 141 metres of fresh water (mfw) during the summer months and 137 mfw during the winter months. There was no significant difference in diving activity between the divers who experienced DCI symptoms and the divers who did not experience any DCI symptoms (P = 0.10). There was also no significant difference between these groups in respect of maximum depth (P = 0.91) or dive time (P = 0.24). Diving activity for the follow-up period is presented in Figure 2.

Footnote: * Appendices 1–3 are available on DHM Journal's website: https://www.dhmjournal.com/index.php/journals?id=294

Figure 2

The number of dives, dive time, and maximum depth stratified into groups reporting and not reporting DCI symptoms over the one-year study period. The boxes show median and first and third quartiles. The whiskers extend up to 1.5 times the IQR and observation outside that range are shown as dots. There was no statistical difference between groups on any of the three measures, *P*-values being 0.10, 0.24 and 0.91 respectively



SYMPTOMS

DCI-related symptoms occurred in 17 divers after 27 dives; thus, the apparent incidence of DCI was 91 per 10,000 dives in this study. The divers reported 33 dives followed by symptoms but after a review by three physicians in the research team, six cases were determined as not being caused by DCI: two divers had symptoms caused by hypercapnia, one suffered from dehydration due to diarrhoea with no DCI symptoms, one was diagnosed with immersion pulmonary oedema (IPO), one was suspected to have pulmonary oxygen toxicity, and one had a frostbite-type of sensation in his feet caused by a leaking dry suit.

Most of the reported symptoms were mild, only one diver reported severe symptoms (pulmonary symptoms, vertigo). The most common symptoms were joint pain (n = 12), muscle pain (n = 10), tingling/itching (n = 6), and skin rash, swelling, and warmth (n = 6). The majority of divers had two or three different symptoms at the same time, e.g., tingling/itching + joint pain + numbness or skin rash + fatigue. The symptoms are shown in Figure 3. In the majority of cases the symptoms appeared within two hours of surfacing (12/27, 44.4%) or within 24 hours (8/27, 29.6%). Some divers experienced symptoms directly after surfacing (3/27, 11.1%) or even underwater (4/27, 14.8%). Divers who experienced symptoms underwater became asymptomatic during decompression stops, but the symptoms reappeared at the surface. Nineteen (70%) of the incident dives took place during the summer months and eight dives (30%) during the winter months.

TREATMENT AND OUTCOMES

After experiencing mild DCI symptoms, the divers tended to self-treat. In 20 events (74%) the divers hydrated orally (more than they normally would after a dive) and in 19 events (70%) the divers rested. In only six events (21%) the divers used FAO_2 and in one event the diver reported not to have treated their symptoms at all. One diver performed an IWR after experiencing mild fatigue and skin rash, swelling, pain and warmth in the upper limb after a dive to 76 mfw with a total dive time of 179 minutes. The delay to perform the IWR was three days. IWR was performed with two safety divers and the diver was utilising a CCR with a maximum inspired PO_2 of 1.7 atmospheres (172.2 kPa). The IWR profile consisted of descent to 35 mfw for two minutes and then a very slow ascent (over 55 minutes) to a 6 mfw habitat. The total duration of the IWR was 120 minutes. The IWR was considered successful as the diver eventually made a complete recovery. The pain, warmth and skin rash had vanished during IWR, and the swelling resolved within a week. Only two symptomatic divers contacted a recompression facility. One had such mild symptoms that the hyperbaric physician decided not to treat the diver with hyperbaric oxygen (HBO) and the symptoms resolved after rest and FAO_2. The other diver (referred to above as having severe symptoms) was recompressed twice in a chamber and recovered completely. In twenty-five events the divers reported complete recovery with the treatment without contacting any medical personnel. In three of these cases the divers reported that their symptoms diminished after self-treatment, but they also commented that the symptoms gradually diminished and all symptoms were gone within several days taking them longer to recover fully. The treatment reported by divers is shown in Figure 4.

PROPOSED CONTRIBUTING FACTORS

The divers suggested possible contributing factors leading to their DCI symptoms on the questionnaire. The most common suggested factor was dehydration, n = 12 (43%), even though the divers underlined in their questionnaire answers that they drank a lot before dives. Another commonly suggested contributing factor was successive days of diving, n = 11 (39%). Finnish divers often spend a weekend at a diving

Figure 3
Reported DCI symptoms in 27 incident dives during a one-year follow-up period

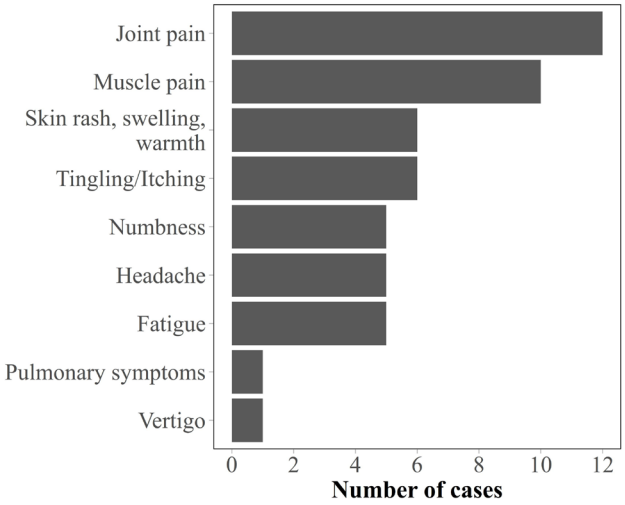
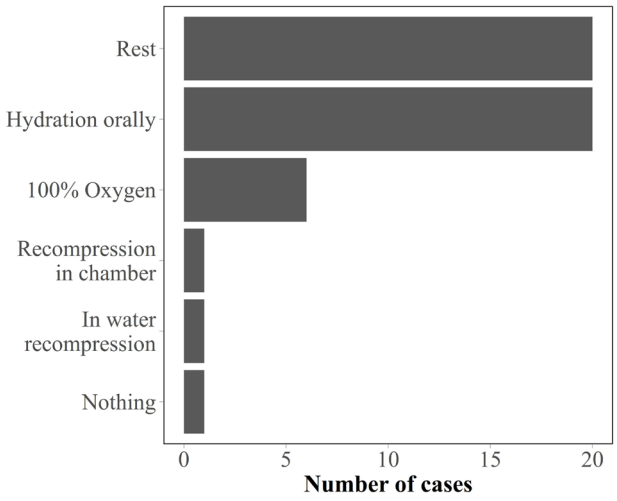


Figure 4
Initial treatment carried out by the divers after experiencing DCI symptoms; the diver might have used more than one initial treatment



site, as many of them are located far away from bigger cities. Therefore, it is common to dive two or three days consecutively. Surprisingly, only six divers (22%) described cold as a possible contributing factor despite extremely cold water temperatures.

Discussion

We hypothesised that demanding technical dives in an extremely cold environment involving many risk factors would be associated with a high incidence of DCI and that highly trained divers may practice self-treatment. In this study, the incidence of self-reported DCI symptoms was 91 per 10,000 dives, which is higher than in the previous questionnaire studies.[14–18] One study involved an analysis of the DAN Europe database including specific questionnaires for data collection, and reported the incidence of DCS at 81.8 per 10,000 dives, which is similar to our findings.[19] A literature review of questionnaire studies is summarised in Table 2.[14–19] In the present study, we recorded one case that received HBO for DCI symptoms among 2,983 dives (incidence of 3.3 per 10,000 dives). Although conditions were very cold and demanding this is consistent with a previous study done with technical divers in warm waters.[13] The majority of the injured divers in the present study treated themselves (n = 26, 96%) without receiving HBO. Most of the symptoms were so mild that the divers did not consider the need to contact a diving medicine physician.

In addition to the harsh diving environment, the high incidence might partly be explained by participation in a study with prospective data collection which may have encouraged divers to self-observe for symptoms more closely than usual. It has been suggested that an increase in annual diving is associated with fewer diving injuries.[17] This may explain why, despite the very cold conditions and demanding dives, we recorded mainly mild DCI symptoms although the incidence was high.

In our study, only six divers (21%) used FAO_2 after experiencing DCI symptoms, even though it is beneficial and recommended as soon as possible after the onset of symptoms.[5] This is an alarmingly low number, but it is consistent with earlier studies.[7,20] The assumption was that skilled and highly trained divers with easy access to oxygen would use FAO_2 more often. To determine why the great majority of these divers did not utilise FAO_2 after experiencing DCI symptoms, author LT interviewed some of the divers. The answers were "*symptoms were so mild or uncertain*", "*a little pain belongs to technical dives*", "*there are too many things to do after a dive, no time for oxygen*", "*some kind of shame in having symptoms*", "*do not know why I did not use it even though I teach other divers to use it*".

Despite the low number of divers using FAO_2 and only one diver receiving HBO, the outcomes were excellent. None of the divers had residual symptoms, and every diver eventually recovered. This is consistent with the present understanding that some mild DCI cases could be adequately managed without recompression with good outcome.[4,5]

Yet, along with rumors of technical divers treating mild DCI symptoms themselves, we have had anecdotal reports from diving physicians and divers themselves, of technical divers suffering recurrent mild DCI symptoms in the same part of the body and with the same symptoms very easily after their first incident. There is no scientific evidence supporting this, but it has raised concern that they might have some form of tissue damage predisposing divers to recurrent DCI or possibly long-term effects such as dysbaric osteonecrosis

Table 2
An overview of questionnaire-based studies on the incidence of DCI conducted on recreational divers

Location and citation	DCI cases	Dives	DCI cases per 10^4 dives	Years	Type of study
Sweden[14]	190	127,256	14.9	1999	Retrospective
Japan[15]	60	1,140,653	0.53	1996–2001	Prospective
Germany[16]	52	284,067	1.83	2003–2005	Retrospective
North America[17]	282	174,912	16.1	2010–2011	Retrospective
France[18]	146	683,171	0.21	2017–2018	Retrospective
Europe[19]	320	39,099	81.8	5 years	Retrospective

(DON). The occurrence of DCI has been linked to DON and recent studies have suggested technical divers are at greater risk than recreational divers due to repetitive, long, deep dives.[21–23]

Finnish technical divers not only perform challenging dives, they also do so in freezing cold conditions. This is especially emphasised during the winter months when there is a 'reverse thermocline' which results in the decompression being performed in even colder water than the constant 4°C water at the bottom depths. Surprisingly, cold was not the leading suggested contributing factor in this study. Perhaps Finnish divers are habitually accustomed to cold and therefore under-emphasise it in these arctic environments, despite cold being an important risk factor.[24,25]

In this study, 11 DCI cases out of 27 occurred after multiple days of diving. Only two cases occurred after a training dive, and all the rest of the DCI cases occurred after deep dives. Typically, divers suggested tiredness and dehydration along with multiple days of diving as contributing factors in DCI. There are several studies suggesting that multi-day hyperbaric exposure might give a protective (acclimatising) effect on DCI and would lower the incidence.[26] Despite the possible acclimatisation, diving deep and very long dives multiple days in a row seemed to increase the incidence of DCI in this study.

LIMITATIONS

Our results depend on self-reported data, which introduces some limitations. Firstly, recall bias may exist even though this is a prospective study. Secondly, there is always a chance that divers unintentionally over-report or under-report the symptoms. There are no records of all technical divers in Finland using mixed breathing gases, and therefore only the ones known by researchers were contacted causing a sample selection bias.

We studied a limited number of highly specialised divers that performed a total of 2,983 dives. Therefore, the results should not be generalised to different types of diving and other diving locations. Sadly, the study period coincided with the COVID-19 pandemic, which considerably reduced the number of dives and especially diving trips abroad. Another factor that reduced the number of dives was that Ojamo, a very popular mine-diving site, was not available for most of the participating divers.

Conclusions

This online survey serves to better determine the incidence of DCI symptoms among Finnish technical divers. The overall incidence of DCI symptoms aligns with previous research using the same methodology. However, the incidence of reported DCI symptoms was 27 times higher than for HBO-treated DCI cases. Divers seem to readily recognise even the mildest DCI symptoms very well. Due to the low rate of FAO_2 utilisation in this study, there appears to be a need to improve divers' awareness and education of the importance of FAO_2. Furthermore, there is also a need to emphasise the importance of seeking contact with expert diving medicine advice in order to assess the severity of the symptoms and consider medical input.

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A prospective single-blind randomised clinical trial comparing two treatment tables for the initial management of mild decompression sickness

Neil Banham[1], Philippa Hawkings[2], Ian Gawthrope[1]

[1] *Department of Hyperbaric Medicine, Fiona Stanley Hospital, Murdoch WA, Australia*
[2] *Medical Services, Alfred Health, Melbourne VIC, Australia*

Corresponding author: *Dr Neil Banham, Department of Hyperbaric Medicine, Fiona Stanley Hospital, 11 Robin Warren Drive, Murdoch WA 6150, Australia*
neil.banham@health.wa.gov.au

Keywords
Decompression illness; Diving research; Hyperbaric oxygen treatment; Recompression; Recreational diving; Scuba diving; Treatment sequelae

Abstract
(Banham N, Hawkings P, Gawthrope I. A prospective single-blind randomised clinical trial comparing two treatment tables for the initial management of mild decompression sickness. Diving and Hyperbaric Medicine. 2022 June 30;52(2):85–91. doi: 10.28920/dhm52.2.85-91. PMID: 35732279.)
Introduction: Limited evidence suggests that shorter recompression schedules may be as efficacious as the US Navy Treatment Table 6 (USN TT6) for treatment of milder presentations of decompression sickness (DCS). This study aimed to determine if divers with mild DCS could be effectively treated with a shorter chamber treatment table.
Methods: All patients presenting to the Fremantle Hospital Hyperbaric Medicine Unit with suspected DCS were assessed for inclusion. Participants with mild DCS were randomly allocated to receive recompression in a monoplace chamber via either a modified USN TT6 (TT6m) or a shorter, custom treatment table (FH01). The primary outcome was the number of treatments required until resolution or no further improvement (plateau).
Results: Forty-one DCS cases were included, 21 TT6m and 20 FH01. Two patients allocated to FH01 were moved to TT6m mid-treatment due to failure to significantly improve (as per protocol), and two TT6m required extensions. The median total number of treatments till symptom resolution was 1 (IQR 1–1) for FH01 and 2 (IQR 1–2) for TT6m ($P = 0.01$). More patients in the FH01 arm (17/20, 85%) showed complete symptom resolution after the initial treatment, versus 8/21 (38%) for TT6m ($P = 0.003$). Both FH01 and TT6m had similar overall outcomes, with 19/20 and 20/21 respectively asymptomatic at the completion of their final treatment ($P = 0.97$). In all cases where two-week follow-up contact was made, ($n = 14$ FH01 and $n = 12$ TT6m), patients reported maintaining full symptom resolution.
Conclusions: The median total number of treatments till symptom resolution was meaningfully fewer with FH01 and the shorter treatment more frequently resulted in complete symptom resolution after the initial treatment. There were similar patient outcomes at treatment completion, and at follow-up. We conclude that FH01 appears superior to TT6m for the treatment of mild decompression sickness.

Introduction

Decompression sickness (DCS) results in divers requiring lengthy treatments in a recompression chamber.[1] The current standard treatment, United States Navy Treatment Table 6 (USN TT6) commits a patient to a minimum 4 hour and 45 minute multiplace chamber treatment, although a United States Navy Treatment Table 5 (USN TT5) can be used for cases of musculoskeletal DCS where symptoms have resolved within 10 minutes of oxygen (O_2) breathing at 60 feet /18 metres of seawater depth equivalent (284 kPa).[2,3] USN TT5 is typically used where there is a short delay to recompression. A USN TT5 has a duration of approximately 2 hours and 15 minutes. Since USN TT6 was developed there has been no investigation of the optimum duration of treatment, although shorter treatment tables have been and continue to be used in some institutions (Cianci P, personal communication, 2020).

Both the USN TT5 and USN TT6 tables used in our monoplace chambers have been modified from the original published versions, with decompression from 284 kPa to 190 kPa and 190 kPa to 101 kPa ('surface pressure') over 10 minutes instead of the usual 30 minutes, as 10 minutes was the slowest decompression rate possible for the Sechrist 3200 chamber. To compensate for this, the modified TT6 (TT6m, Figure 1) and TT5 (TT5m, Figure 2) tables used in this study have an extra 20-minute O_2 breathing period at 284 kPa, as compared with standard published USN TT5 and TT6 tables.[4] The FH01 table (Figure 3) was developed by Dr Robert Wong, a previous medical director of Fremantle Hospital Hyperbaric Medicine Unit as a blend of USN TT5

Figure 1

Fremantle Hospital Hyperbaric Medicine Unit USN TT6 (modified) for monoplace chamber application; pressures are absolute pressures. The total time is 4 hours 35 minutes (275 minutes); compression rate 18 kPa·min^{-1}, decompression rate 9 kPa·min^{-1}; BIBS – built in breathing system; kPa – kilopascals; msw – metres of seawater; O_2 – oxygen; Pt – patient

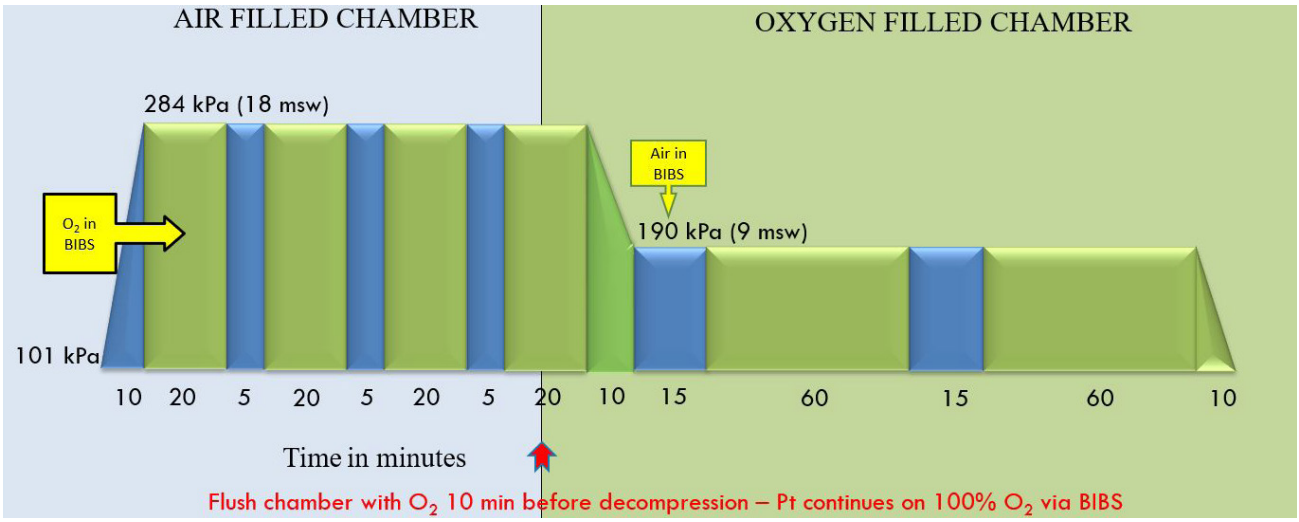
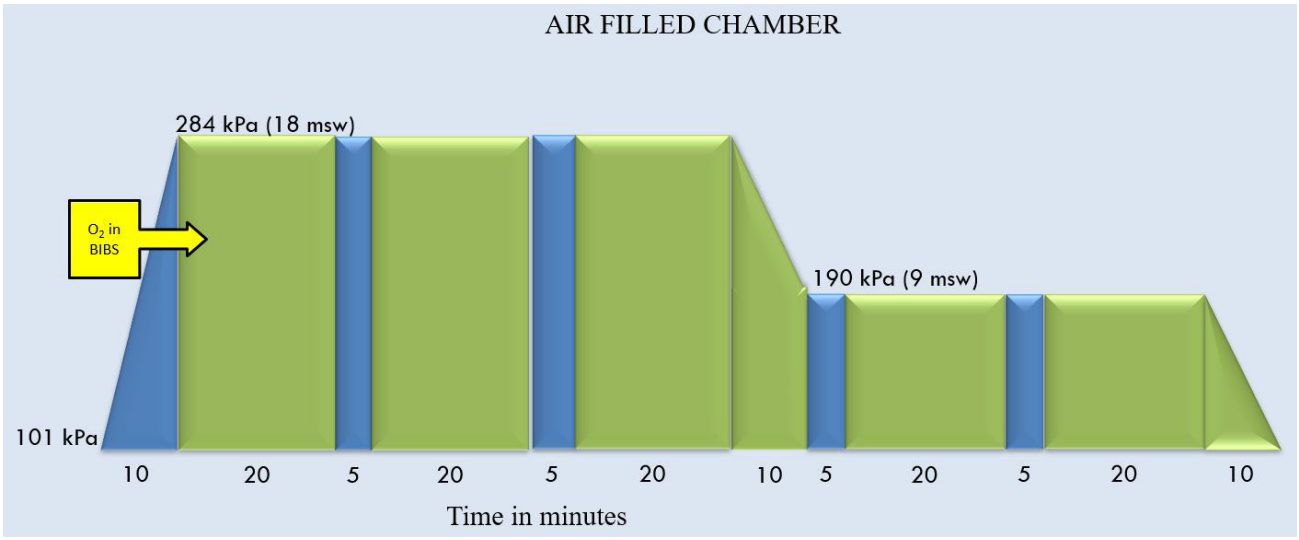


Figure 2

Fremantle Hospital Hyperbaric Medicine Unit USN TT5 (modified) for monoplace chamber application; pressures are absolute pressures. The total time is 2 hours 30 minutes (150 minutes); compression rate 18 kPa·min^{-1}, decompression rate 9 kPa·min^{-1}; BIBS – built in breathing system; kPa – kilopascals; msw – metres of seawater; O_2 – oxygen



and our 200 kPa (2 atmospheres absolute [atm abs]) no air break table (Figure 4). The decompression of FH01 from 284 kPa to 200 kPa and 200 kPa to 101 kPa is likewise over 10 minutes.

There is a recognised spectrum of DCS, ranging from mild non-specific symptoms to severe neurological or cardiopulmonary symptoms.[5] Our study focused on divers who presented at the milder end of the range (see *Appendix 1, Groups 3–6).[5] We included all divers where the presumptive diagnosis was DCS Grades 3–6. It is acknowledged that the natural history of mild DCS is toward spontaneous symptom resolution, and therefore, many such cases can be adequately treated without recompression.[6,7] However, there is also a consensus that symptom resolution is accelerated by recompression, and modern practice guidelines advocate recompression in mild cases if recompression is available without substantial logistic constraints.[7,8] It follows that the optimal approach to recompression in these patients remains a valid and

Footnote: * Appendix 1 is available on DHM Journal's website: https://www.dhmjournal.com/index.php/journals?id=295

Figure 3

Fremantle Hospital Hyperbaric Medicine Unit FH01 for monoplace chamber application; pressures are absolute pressures. The total time is 2 hours 40 minutes (160 minutes); compression rate 18 kPa·min^{-1}; decompression rate 8.4 kPa·min^{-1} from 284 to 200 kPa and 10 kPa·min^{-1} from 200 kPa to 'surface pressure'. BIBS – built in breathing system; kPa – kilopascals; min – minutes; msw – metres of seawater; O_2 – oxygen; Pt – patient

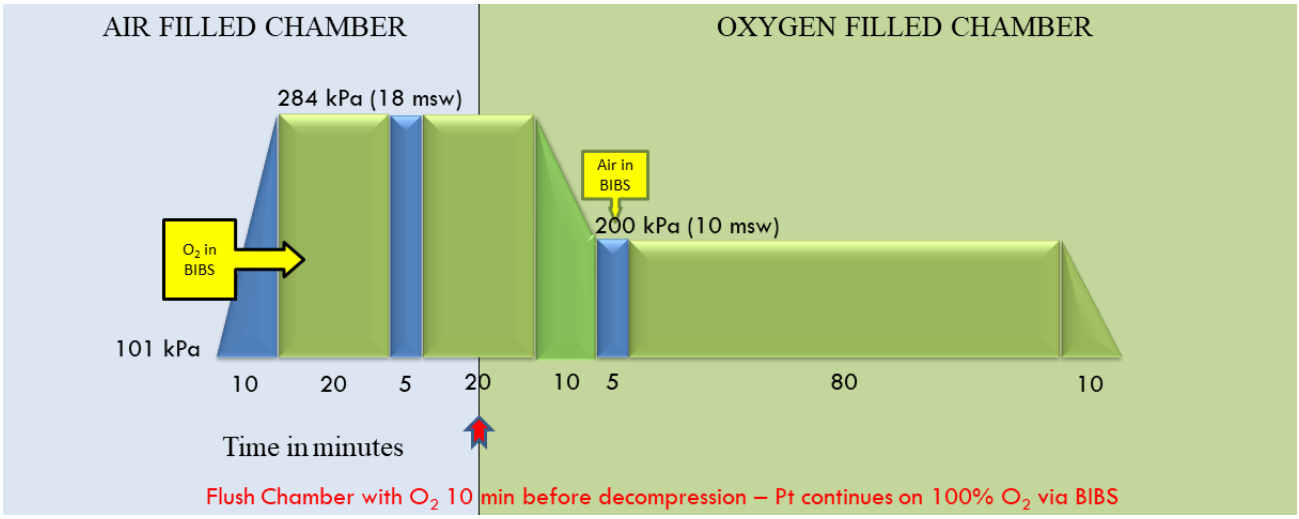
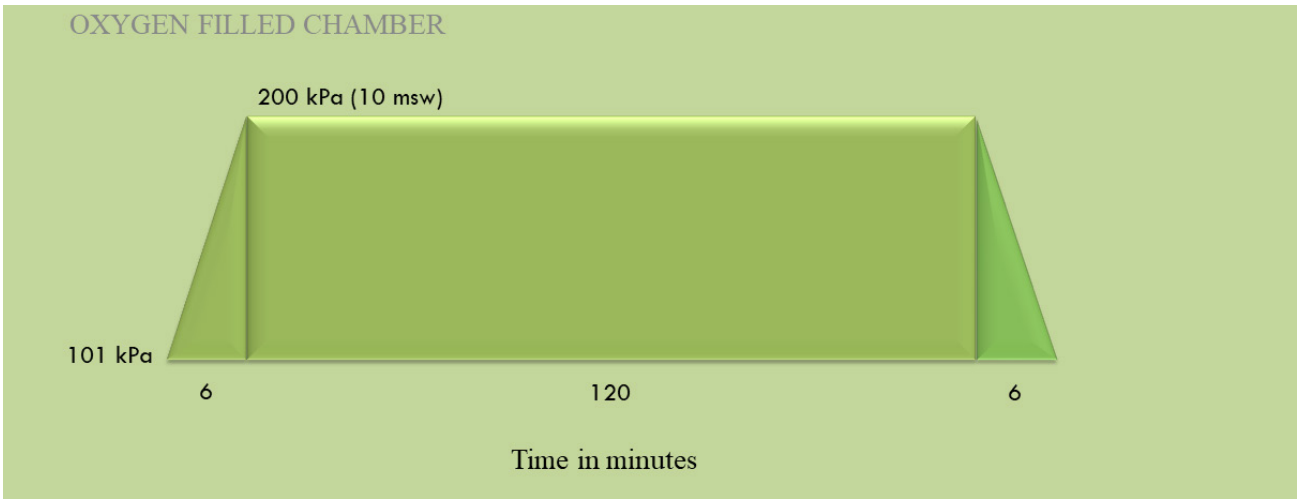


Figure 4

Fremantle Hospital Hyperbaric Medicine Unit Table 10:120:06 for monoplace chamber application; pressures are absolute pressures. The total time is 2 hours 12 minutes (132 minutes); compression rate 16.5 kPa min^{-1}; decompression rate 16.5 kPa/min^{-1}; kPa – kilopascals; msw – metres of seawater



open question. This study aimed to determine if divers with mild DCS could be effectively treated with a shorter initial chamber treatment table.

Methods

Ethical approval for this study was granted by the South Metropolitan Area Health Service Human Research Ethics Committee (HREC 10/477).

All patients presenting to the Fremantle Hospital Hyperbaric Medicine Unit with DCS were assessed to establish whether they met the criteria to be included in the trial. The primary outcome was the number of treatments required until resolution or plateau in recovery, with the secondary outcome being resolution of all symptoms after the initial recompression.

INCLUSION CRITERIA

Patients were included if they were 18 years or older, gave informed consent, and had one or more of the following manifestations: mild neurological symptoms, pain, lymphatic/skin, and constitutional/non-specific symptoms.

Pain was defined as musculoskeletal pain and specifically excluded girdle-type pain, a harbinger of spinal DCS. Further information of these manifestations is listed in *Appendix 1 (Groups 3–6 of the Divers Alert Network classification system). The one departure from this classification system was that patients with true vertigo were not included as this is not considered a mild symptom in contemporary practice.[6,7] Patients were excluded if they had serious neurological (including inner ear) or cardiopulmonary DCS, or any manifestation not in the inclusion criteria. The assessing physician decided on the diagnosis of 'mild DCS' based on the Appendix 1 table and was blinded to the treatment arm participants were then assigned to.

Participants were randomly allocated to receive recompression via either TT6m (Figure 1) or FH01 (Figure 3), in a Sechrist 3200 or 3600 monoplace chamber (Sechrist Industries Inc, Anaheim CA). The randomisation process was via a sealed opaque envelope system selected by the duty hyperbaric technician, with computer generated allocation. Participants were not informed into which arm of the trial they were assigned. Inspection of the TT6m and FH01 (Figures 1 and 3) tables used in this study show that they have identical profiles up to the end of the second O_2 period. At this point the assessing doctor, who was blinded to treatment table allocation, would make a decision as to whether the diver's symptoms had resolved sufficiently to allow completion of the table as allocated (> 75% symptom resolution), or to change the table and as such, define these participants as 'treatment failures' to allow an extended time of initial recompression treatment as a safety mechanism. For FH01 subjects this meant conversion to TT6m and for those already in TT6m arm, one or two extensions with further 20-minute O_2 breathing periods at 284 kPa.

ADJUNCTIVE THERAPY

All patients could receive normobaric oxygen whilst awaiting hyperbaric therapy where appropriate. One litre of fluid was advised to be given to all trial patients prior to recompression, either orally or as intravenous normal saline. The need for further oral or intravenous fluid and analgesia was decided by the referrer or by the assessing doctor according to clinical need. Analysis of the type and amount of adjunctive therapy was not performed.

INITIAL TREATMENT TABLE

Patients received either a TT6m or the shorter FH01 in a monoplace chamber. In this study the effect of initial treatment table (the independent variable of interest) upon both initial and eventual symptom resolution (complete or not) is reported.

FOLLOW-UP TREATMENT TABLE

All patients received a follow-up hyperbaric treatment unless they had become asymptomatic prior to the commencement of their initial recompression, (n = 1 in the TT6m arm), or did not re-attend, (n = 1 in the FH01 arm), as per our usual practice of treating to resolution of symptoms plus one. The decision as to whether a further treatment was required was made on further assessment immediately prior to commencing the next treatment. Follow-up treatments did not differ by the initial treatment arm. The protocol was that the patient would routinely receive a daily FH 200:120:06 table (120 minutes at 200 kPa [2.0 atm abs] with no air break, Figure 4) unless they had significant ongoing or recurrent symptoms where the treating clinician could opt for a TT5m (Figure 2). If there was no monoplace availability for a timely follow-up treatment, a participant could be given a 243 kPa (2.4 atm abs) treatment in the multiplace chamber (two 45-minute O_2 breathing periods separated by a 5-minute air break with a 24-minute decompression). Patients were treated to resolution of all symptoms plus one treatment or plateau (no change in symptoms after three treatments).

FOLLOW UP POST DISCHARGE

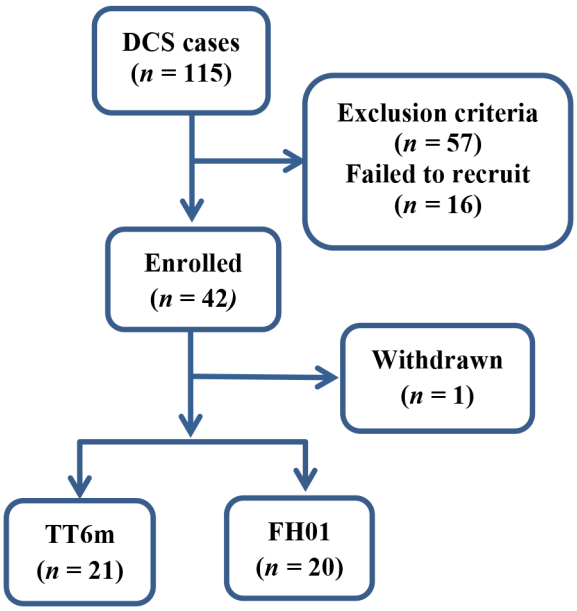
All patients were attempted to be contacted by telephone two weeks following their final treatment to assess their progress and presence of any residual or recurrent symptoms.

ANALYSIS

Data were stored in Microsoft Excel then imported in SAS (Cary, NC) version 9.4 for analysis. The initial power calculations were based upon a two-sided t-test, where the null hypothesis was that there would be no difference detected between protocols in the mean number of treatments before symptoms resolved or there was no further improvement (plateau). Asymptomatic 'plus one' treatments were not counted in this number. An initial sample size of 20 in each arm was decided as being achievable for recruitment into a study, based on the number of cases of DCS treated annually (approximately 30 per year). A sample of 20 patients in each arm would have a power of 87% to detect a mean difference of one treatment between arms. With the exception of reporting aggregated data for resolved cases, all values reported herein relate to the intention to treat (ITT) analysis. Any participants defined as failures to respond to two oxygen breathing periods at 284 kPa were included in the ITT.

Because the expected number of recompression treatments required was small, we anticipated the results would not be normally distributed, and planned an analysis to explore the differences between the median total number of treatments required in each group using a two-sided Wilcoxon rank-sum test (WRS). A Fisher's exact test was calculated when

Figure 5
Modified CONSORT flow diagram; FH01 – Fremantle Hospital Treatment Table 01; TT6m – United States Navy Treatment Table 6 (modified)



comparing the number of patients resolved after their initial treatment between treatment tables. Significance was accepted when $P < 0.05$.

Results

A total of 115 patients with suspected DCS presented during the study period (17 October 2010 to 5 July 2014). Of these, 41 patients diagnosed with mild DCS were included in the study, 21 allocated to TT6m and 20 to FH01 (Figure 5). There was no difference between allocation groups in patient age or sex. Two patients allocated to the FH01 arm showed < 75% symptom resolution at the end of the second 20-minute O_2 breathing period at 284 kPa (2.8 atm abs), and their treatment table was continued as a TT6m (though not added to the TT6m arm for analysis). Likewise, two TT6m patients required a single 20-minute O_2 breathing extension. Neither of the two participants that crossed to the TT6m arm required extensions to their TT6m.

One patient was treated on two occasions, just over two years apart, and was considered in the analysis as two separate cases. Thirty-seven cases (90%) were male, mean age (years) was 35.3 (SD 6.7) for females and 36.5 (SD 9.2) for males, 36.9 (SD 10.5) for FH01 and 35.8 (SD 7.5) for TT6m. The distribution of symptoms by treatment table is presented in Table 1. There were two subjects in each group that had a lengthy delay to recompression, both of whom had been diving overseas.

The median total number of treatments to achieve symptom resolution was one (IQR 1–1) for FH01 (range 1–3) and two (IQR 1–2) for TT6m (range 0–5), (WRS $Z = -2.67$, $P = 0.01$). Of the patients receiving FH01 initially, 17/20 (85%) showed complete symptom resolution after the initial treatment, versus 8/21 (38%) for TT6m ($P = 0.003$). At the completion of their final treatment, both FH01 and TT6m had similar overall outcomes, with 19/20 and 20/21 respectively asymptomatic ($P = 0.97$). Of the 'treatment failure' patients, one of those in the TT6m arm that required an extension had resolution of symptoms at the end of their initial extended TT6m, the other had full resolution after a single follow-up treatment. For the FH01 participants changed to TT6m, neither had complete resolution after their initial treatment but both were fully resolved after a single follow-up treatment.

Table 1
Distribution of symptom severity[5] by treatment table; [a] – denotes that more than one symptom group may be present; FH01 – Fremantle Hospital Treatment Table 01; TT6m – United States Navy Treatment Table 6 (modified)

Symptoms	TT6m *n* (%)[a]	FH01 *n* (%)[a]	Total *n* (%)[a]
Mild neurology	6 (29)	7 (35)	13 (32)
Pain	16 (76)	16 (80)	32 (78)
Lymphatic/skin	2 (10)	2 (10)	4 (10)
Constitutional/ non-specific	8 (38)	4 (20)	12 (29)

In one of the cases in the TT6m arm, symptoms persisted after two recompression treatments (TT6m then one 200:120:06 table) but further treatment was declined. This patient was nevertheless assigned two as the number of treatments for the primary outcome. One FH01 participant had resolution of symptoms after the first treatment but failed to return for a follow-up treatment. The participant remained asymptomatic at follow-up telephone contact. This patient was accordingly assigned 'one' as the number of treatments for the primary outcome. For the two patients who did not achieve resolution (one in each group), both had three treatments at plateau.

All subjects received the FH 200:120:06 table (Figure 4) as follow-up treatment, except for three receiving TT5m (nil in the FH01 and three in the TT6m groups respectively) and two receiving a 243 kPa multiplace treatment (one in the FH01 and one in the TT6m groups respectively). No distinction was made between different follow-up treatment tables in our analysis.

Of the 20 FH01 patients, 14 (70%) could be contacted at two weeks after their final treatment. All 14 had full resolution after their final treatment and remained asymptomatic at two weeks. Similarly, for the 21 TT6m patients, 12 (57%) could be contacted at two weeks after their final treatment, and all

had full resolution after their final treatment and remained asymptomatic at two weeks.

Discussion

This study found that the median total number of treatments to achieve resolution of symptoms was significantly fewer in the FH01 arm than in the TT6m arm, and that treatment table FH01 more frequently had complete symptom resolution after the initial treatment than TT6m. However, there was no difference in the number of patients achieving resolution at the completion of treatment.

There has previously only been one randomised controlled trial on the treatment of DCS completed: a trial of a non-steroidal anti-inflammatory drug as adjunctive therapy to recompression.[9,10] Another randomised controlled trial comparing oxygen and oxygen-helium in the treatment of air-diving decompression illness was reported as underway, but final results have never been published.[11,12] The present study is only the second completed randomised controlled trial published on the treatment of DCS, and the first to compare the outcomes of short and long oxygen recompression tables. Although there have not been other randomised trials, several studies have suggested the efficacy of short treatment tables. One compared enhanced treatment tables with a variety of regular treatment tables in a non-randomised multicentre study of 327 treated scuba divers.[13] A logistic regression analysis confirmed the shorter regular treatment tables had greater successful resolution of symptoms than the enhanced tables (63% vs. 48% respectively), though the authors highlighted a potential selection bias in the study design.[13] Another study reviewed the development of these short oxygen tables and their published outcomes as well as experience with using a short no-air-break table, and reported a 98% full recovery rate.[14] On the basis of the results of these retrospective reviews it was concluded that "…*this short oxygen protocol has proven highly effective for the type of patients presenting to our hospital, a major Divers Alert Network referral center, for decompression sickness*."[14]

A retrospective review of 292 cases of Type I DCS treated with either TT5 (208 cases) or TT6 (84 cases) showed similar (4.3% versus 3.6% $P > 0.10$) rates of symptom recurrence.[3]

A possible reason for the increased efficacy of the shorter table (FH01) could be that treated divers were exposed to much less exogenous nitrogen (10 minutes versus 45 minutes) during their initial recompression, owing to the differing length of air breaks in the respective treatment tables (Figures 1 and 3). It is conceivable that nitrogen in air breathed during air breaks may diffuse into residual bubbles and expand them. The fact that FH01 table is completed at 200 kPa rather than 190 kPa in the TT6m seems less likely to be a significant contributor to the outcome difference.

One case that was withdrawn and excluded from analysis was a 37-year-old man who presented with symptoms of musculoskeletal DCS, subsequent investigation of which determined the event to be factitious. Munchausen's Syndrome presenting with DCS symptoms has been previously described.[15–17]

Regarding the ITT analysis, the two patients who discontinued FH01 were thereafter treated with TT6m, but were not added to the TT6m arm. To have counted patients who were not responding to FH01 within the TT6m arm would have introduced a directional bias. Furthermore, the two patients who were discontinued from the TT6m were treated for the remainder of their initial treatment differently (an extra 20-minute O_2 period at 284 kPa / 2.8 atm abs) to the two patients moved from the FH01 arm (TT6m). Following their initial treatment however, follow-up treatments were equivalent for all four patients.

LIMITATIONS

This was a small study prone to both Type 1 and Type 2 errors. Nevertheless, based on the present results, at the least it seems very unlikely that choosing the shorter FH01 table to treat mild DCS would constitute an inferior approach when compared to a TT6.

Another limitation was that many patients could not be contacted for post treatment follow-up, therefore it is not known with certainty if the comparable outcomes between FH01 treatment and TT6m were lasting. Another limitation may have been a form of selection bias, with just 41 of 115 (36%) potentially eligible patients recruited, although, as indicated in Figure 5, 57 patients (50%) did not satisfy the eligibility criteria, plus allocation to the treatment arms was randomised.

Conclusion

We conclude that FH01 appears superior to TT6m for the treatment of mild DCS. Although the ultimate rate of recovery was not different, which is probably to be expected in mild DCS where the natural history is toward eventual recovery irrespective of treatment modality, divers treated with the shorter oxygen table required fewer recompression treatments and were more likely to be symptom-free after the first recompression.

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A survey of caustic cocktail events in rebreather divers

Peter Buzzacott[1], Grant Z Dong[2], Rhiannon J Brenner[2], Frauke Tillmans[2]

[1] *Prehospital, Resuscitation and Emergency Care Research Unit, Curtin School of Nursing, Curtin University, Western Australia*
[2] *Divers Alert Network, Durham, North Carolina, USA*

Corresponding author: *Dr Frauke Tillmans, Research Director, Divers Alert Network, 6 W Colony Place, Durham (NC) 27705, USA*
ftillmans@dan.org

Keywords
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Abstract
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Introduction: Closed-circuit rebreathers (CCRs) are designed to be watertight. Ingressing water may react with carbon-dioxide absorbent in the CCR, which may produce alkaline soda with a pH of 12–14, popularly referred to by CCR divers as a 'caustic cocktail'. This study aimed to explore divers' responses to caustic cocktail events and to investigate if CCR diving experience is associated with experiencing a caustic cocktail.
Methods: An online survey instrument was developed and an invitation to participate was extended to certified CCR divers aged ≥ 18 years. Relationships between number of caustic cocktail events and potential risk factors: age; hours of rebreather diving experience; and number of rebreather dives were explored.
Results: Of the 413 respondents, 394 (95%) identified as male, mean age was 46 years and median length of CCR certification was six years. Fifty-seven percent (n = 237) of respondents reported having experienced a caustic cocktail. The probability of self-reporting none, one, or more caustic cocktail events increased with experience. Divers reported a variety of first aid treatments for caustic cocktails, with ~80% citing their CCR instructor as a source of information.
Conclusions: The more hours or dives a CCR diver accrues, the more likely they will self-report having experienced one or more caustic cocktail events. The majority of CCR divers responded to a caustic cocktail by rinsing the oral cavity with water. A proportion of divers, however, responded by ingesting soda, dairy, juice, or a mildly acidic solution such as a mixture of vinegar and water. The recommendation to immediately flush with water needs reinforcing among rebreather divers.

Introduction

Closed-circuit rebreather (CCR) systems are designed to be watertight and airtight. Bubbles are rarely seen escaping a normally functioning rebreather when it is being used at a constant depth. An exception may be a semi-closed-circuit rebreather, where some of the breathing gas is routinely expelled. In fully-closed rebreathers, when bubbles are seen escaping a leak is indicated and water may be entering the breathing circuit. Ingressing water may mix with the substances packed into the rebreather that absorb carbon-dioxide (CO_2) and a by-product of the consequent reaction between water and the CO_2-absorbing agent is the production of extremely concentrated caustic soda, dissolved NaOH, with a pH between 12–14.[1,2] This mixture is popularly referred to by CCR divers as a 'caustic cocktail'.[3] If this enters the mouth and oropharynx, resulting pain and injury severity may vary from coughing, dyspnoea and dysphagia,[4] through to severe internal corrosive injury.[5]

The Divers Alert Network (DAN) diving incident reporting system (DIRS) collects incident reports from recreational divers, including CCR divers. If the divers supply contact details, then additional information is often sought by DAN in order to compile a more detailed version of events. The incidents are summarised each year in the DAN Annual Diving Report, and the first 500 incidents were recently reviewed.[6] Twenty-six of these (5%) involved rebreathers.[6] In speaking with some of these rebreather divers, it became apparent that there exists a range of home remedies for first-aid treatment after oral contact with caustic soda. These include rinsing with or drinking a mild acid[3] such as a carbonated drink or fruit juice, or to swallow milk or other dairy products.[5]

The accepted first-aid treatment for a oral exposure to caustic soda is to immediately flush repeatedly with water,[7] preferably freshwater but seawater is still effective if this happens to a diver in the sea. Harm will be minimised if the diver immediately removes the rebreather mouthpiece from the mouth and repeatedly flushes the oral cavity with water. However, one diver described waiting till he had exited the water to gargle with soda, reportedly because that is what he was taught during his rebreather diving class. He

also reported suffering burns to the inside of his mouth. In severe cases with the potential for internal corrosive injury, or if symptoms are not mild or improving, it is highly recommended the diver seek medical attention.[8]

It is not known who suffers caustic cocktail events, how soon they occur after rebreather certification, how long into the dive they occur, how frequently they occur, or how divers respond to these incidents. This study aimed to explore divers' responses to caustic cocktail events and to investigate if CCR diving experience is associated with experiencing a caustic cocktail.

Methods

Ethics approval was granted by the Institutional Review Board of Divers Alert Network, approval 023-18 dated 6 April 2018.

A survey instrument was developed and assessed for face and content validity, then hardcopies were pilot-trialled at Boston Sea Rovers, a large recreational diving trade show in the USA. Following this trial, an online version was developed, a second pilot trial undertaken, and minor revisions made. The invitation to participate was extended to certified CCR divers aged 18 years or older. A link to the survey instrument was published on the DAN website as an ongoing research project. The link was shared through DAN's social media outlets (Facebook, Instagram and Twitter) and launch of the study was advertised during four different webinars in the fall of 2020, targeting the recreational and technical diving community. The survey was online from 9 September 2020 to 1 March 2021. Participants were presented with a participant information page and required to anonymously indicate consent before proceeding to the survey. The survey instrument collected data on the divers' age and sex, CCR diving experience, the source of their knowledge of how to respond to a caustic cocktail event and each diver's experiences with caustic cocktail, whether personally experienced or witnessed. The structure of the survey is shown in Figure 1.

Figure 1
Online survey structure flowchart

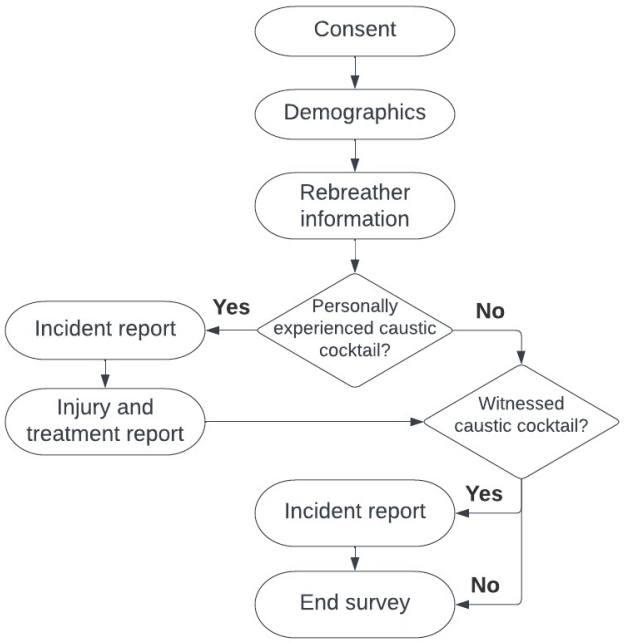
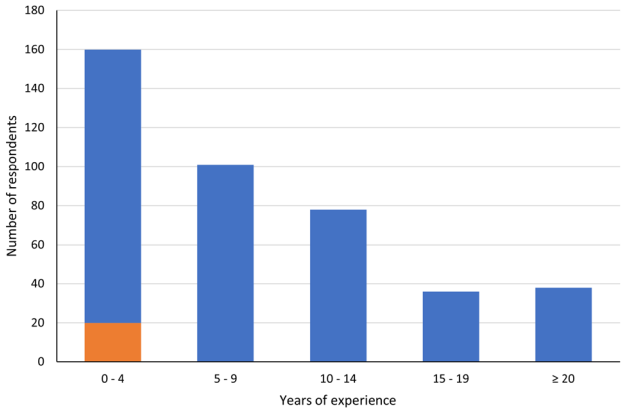


Figure 2
Number of years of experience diving with rebreathers among the 413 respondents; orange subsection represents divers with less than one year of experience



ANALYSIS

Data were stored in Microsoft Excel and analysed using SAS version 9.4 (SAS, Cary NC, USA). Frequencies are reported by counts and percentages. Normally distributed variables are described by means and standard deviations (SD), whereas variables with non-parametric distributions are described with medians and inter-quartile ranges (IQR). The relationships between number of caustic cocktails personally experienced and type of rebreather configuration preferred was explored using a chi-square test, with odds ratio (OR) and 95% confidence intervals (CI) estimated.

Self-reported pain scores were tested for normality using a Shapiro-Wilk test and, being non-normally distributed, the association between pain and seeking medical treatment was assessed using a logistic regression, which does not rely on a Gaussian distribution of residuals.

Potential risk factors (age, hours of rebreather diving experience, and number of rebreather dives) were explored for association with a caustic cocktail event using an ordinal logistic regression model, with four outcome levels, (0, 1, 2, ≥ 3 experiences). The model was parsimoniously optimised using backwards elimination, with the goodness of fit assessed using the log likelihood ratio test (LLRT). At each stage a chi-square score test tested the proportional odds assumption. Regression parameters were iteratively estimated using Fisher's scoring method. Significance was accepted at $P < 0.05$.

Table 1
Reported preferred rebreather configurations

Configuration	Frequency *n* (%)	Caustic cocktail *n* (%)
Back-mount	312 (76)	167 (53)
Side-mount	34 (8)	30 (88)
Chest-mount	32 (8)	20 (63)
Back-mount and side-mount	19 (5)	12 (63)
Back-mount and chest-mount	9 (2)	4 (44)
All three configurations	1 (0)	1 (100)
Missing	6 (1)	0 (0)

Table 2
Reported sources of advice for responding to a caustic cocktail; EMS – emergency medical services

Advice	*n* (%)
Instructor during training	322 (78)
Manufacturer	117 (28)
Dive team members / divers at dive site	109 (26)
Social media	55 (13)
Medical professional (diving physician / EMS)	51 (12)
Divers Alert Network	30 (7)

Table 3
Frequency of personally experienced caustic cocktails

Number of events	Age Mean (SD)	Number of dives Median (IQR)	Number of hours Median (IQR)	Total
0	45 (10)	200 (81–400)	250 (92–550)	175 (42)
1	48 (10)	200 (100–500)	300 (137–700)	174 (42)
2	46 (10)	442 (200–1,000)	600 (300–2,000)	46 (11)
≥ 3	49 (8)	700 (450–2,700)	1,000 (450–2,700)	17 (4)

Results

Of the 413 respondents, 394 (95%) identified as male and mean age was 46 years (SD 10). Respondents reported a total of 3,492 years of experience since first certified to dive rebreathers (median 6 years, IQR 3–12), 177,330 CCR dives and 278,279 CCR diving hours. The median number of self-reported dives was 200 (IQR 100–500) and the median reported hours of rebreather diving was 300 (IQR 120–750). Forty-four participants (11%) reported ≤ 50 hours experience. The range of years of experience is shown in Figure 2. The rebreather configurations used, and the respective proportion of users reporting a caustic cocktail are presented in Table 1.

After excluding multiple configuration sub-groups ($n = 29$ participants, < 1%) and the six missing configurations, compared with chest-mount, the odds of reporting having experienced a caustic cocktail event were lower in back-mount divers (OR 0.69, 95% CI 0.32, 1.45) and greater in side-mount divers (OR 4.50, 95% CI 1.27, 15.95), as shown in Table 1.

There were 37 manufacturer brands named by the participants. When asked if they self-pack their CO_2 scrubbers, 23 (6%) reported using pre-packed cartridges, and 389 (94%) reported refilling their own scrubbers, (one did not report their preference). It is worth noting that the ability of using pre-packed cartridges is determined by design of the rebreather, some models allow for both options.

CAUSTIC COCKTAIL EXPERIENCE

Regarding the participants' reported sources of advice for what to do in the event of experiencing a caustic cocktail, the various responses are shown in Table 2.

Other reported sources of advice for what to do in the event of a caustic cocktail included books, magazine articles, internet searches, and internet forums. Fifty-seven percent (n = 237) of respondents reported having personally experienced a caustic cocktail. One hundred and seventy-five participants (42%) reported not having experienced a caustic cocktail themselves. The frequency of personally experiencing a caustic cocktail among those 237 participants is presented in Table 3.

Fitting age, dives, and hours to the ordinal logistic regression model with reported number of caustic cocktails experienced as the outcome variable, age was removed first as least-significant (P = 0.09), and the fit of the model was not significantly worse off (LLRT $P > 0.05$). Next for removal

was the variable 'hours of experience on CCR', ($P = 0.07$) but this significantly worsened model fit (LLRT $P < 0.05$) therefore the optimised model shown in Equation 1 retained number of hours experience and number of dives experience. The proportional odds assumption held true at each stage of the model optimisation.

$$Ln\left(\frac{P_j}{1-P_j}\right) = \alpha_i + (0.000488\ Dives + (0.000192)\ Hours$$

Eq. 1

The modelled probability of outcome state j, (of 1, 2 or ≥ 3 caustic cocktails experience, compared with no caustic cocktail history), is P_j, where $\alpha_1 = 0.0108$, $\alpha_2 = -2.1017$ and $\alpha_3 = -3.6171$; *Dives* is the number of CCR dives; and *Hours* is the number of hours rebreather diving experience. In this sample of CCR divers, for every 100 additional dives, the odds of self-reporting an additional caustic cocktail increased by 5%, (OR 1.05, 95% CI 1.009, 1.093), and for every 100 additional hours of experience, the odds of self-reporting an additional caustic cocktail increased by 2% (OR 1.019, 95% CI 0.999, 1.041).

Regarding the most recent dive during which participants had experienced a caustic cocktail, the event occurred after a median of 40 minutes (IQR 10–60) into the dive. After the caustic cocktail occurred, the first thing the participants reported flushing their mouth with, drank, or ate in immediate response are presented in Table 4.

On a scale from 1 to 10, with 1 being minimal and 10 being maximal, the reported pain scores after experiencing the caustic cocktail are shown in Figure 3. Of the 237 divers (57%) who reported having experienced a caustic cocktail, $n = 34$ (14%) reported having sought medical advice, including 10 who contacted the DAN medical assistance helpline. The median pain score for participants who did not seek medical treatment was 2 (IQR 1–4) and the median score for participants who did was 5 (IQR 3–7), OR 1.4 (95% CI 1.2, 1.6). Twenty-two of the 237 divers (9%) reported taking medications as a result of the caustic cocktail.

Discussion

The proportion of respondents who identified as male is far higher than found during the Behavioural Risk Factor Surveillance System surveys of US divers,[9] 'Discover Scuba' participants worldwide,[10] or in other large surveys of recreational divers. Why such a high proportion of responding rebreather divers should be male is unknown, though this is a survey of divers with an interest in reporting a link to caustic cocktails, not a randomly sampled representative sub-set of CCR divers. The reported median of 200 dives over a median of six years is similar in scale to the estimated average of 30 dives per year per recreational rebreather diver made in a study of CCR fatalities.[11]

Figure 3
Distribution of pain scores after experiencing a caustic cocktail

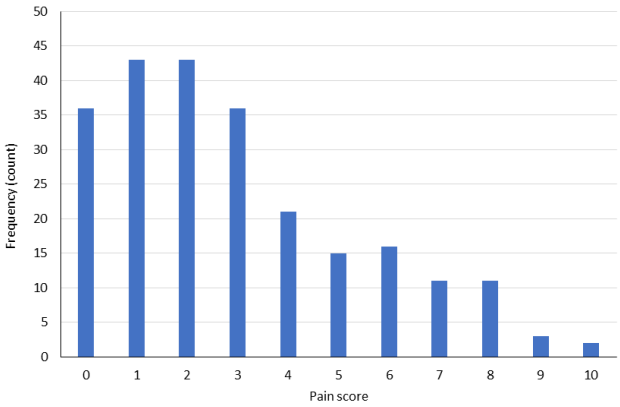


Table 4
First-aid treatment for most recent personally experienced caustic cocktail events

Treatment	*n* (%)
Water	186 (79)
Soda	19 (8)
None	10 (4)
Milk / yoghurt	6 (3)
Fruit juice	5 (2)
Mild acid	5 (2)
Other	4 (2)
Total	235 (100)

We cannot draw inference from Figure 2 regarding when a caustic cocktail may be experienced by CCR divers, other than to conclude they were reported by divers with less than one year of CCR experience through to divers with more than 20 years of experience. There appeared to be no difference in age between divers reporting 1, 2 or ≥ 3 caustic cocktail experiences. The number of experiences with caustic cocktails did appear associated with exposure, both in number of hours rebreather diving and number of rebreather dives. In this study, divers with 1–5 years of experience were the most frequent group to respond to our survey to report experiencing or witnessing caustic events. In short, it appears a caustic cocktail event can happen at any stage of a rebreather diver's CCR diving, but the more hours and more dives experience they accrue, then the more likely they will experience a caustic cocktail event, regardless of whether they have experienced one previously. The odds of reporting having experienced a caustic cocktail event were lower in back-mount divers than in chest-mount divers, and greatest in side-mount divers. Survey study designs cannot investigate causality however, so prospective research is needed to determine if any particular configuration is more prone to water ingress, bearing in mind that different configurations are used in different environments.

Many respondents reported low pain scores associated with caustic cocktail events, and a minority reported extremely high pain scores, but pain scores were missing for nearly half the participants. Perhaps unsurprisingly, among those who did report pain scores, (n = 237, 57%), there appeared to be an association between higher pain scores and the odds of seeking medical treatment.

LIMITATIONS

Future research should explore differences between rebreather divers who have experienced a caustic cocktail and rebreather divers who have not, preferably prospectively. This survey, as with surveys in general, suffers from many limitations such as non-random sampling, and the results may not be representative of rebreather divers in general. Even so, the relationships between self-reported variables may offer some insight into the caustic cocktail experience. To our knowledge, this is the largest online survey of rebreather divers, and the first subjected to peer-review.

Conclusions

Caustic cocktail events can occur at any time on the spectrum of CCR diving experience, but the more hours and/or the more dives a CCR diver accrues, the more likely they will self-report having experienced one or more caustic cocktail events. Where the response to a caustic cocktail event was reported, the majority of CCR divers responded by rinsing the oral cavity with water, having been advised to do so by their instructor during rebreather dive training. A proportion of CCR divers, however, responded by ingesting soda, dairy, juice, or a mildly acidic solution such as a mixture of vinegar and water (a treatment recommended in the 1970s).[3] The recommendation to immediately flush with water[7] needs reinforcing among rebreather divers and emphasis should be placed on educating rebreather instructors who, according to our findings (Table 2), are the primary source of advice for most rebreather divers.

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The effect of pressure changes during simulated diving on the shear bond strength of orthodontic brackets

Meenal N Gulve[1], Nitin D Gulve[2]

[1] *Department of Conservative Dentistry and Endodontics, M.G.V.'s K.B.H. Dental College and Hospital, Nashik, India*
[2] *Department of Orthodontics and Dentofacial Orthopedics, M.G.V.'s K.B.H. Dental College and Hospital, Nashik, India*

Corresponding author: *Dr Meenal N Gulve, Department of Conservative Dentistry and Endodontics, M.G.V.'s K.B.H. Dental College and Hospital, Nashik, India*
meenalgulve@gmail.com

Keywords
Barotrauma; Dental; Diving; Hyperbaric research; Scuba

Abstract
(Gulve MN, Gulve ND. The effect of pressure changes during simulated diving on the shear bond strength of orthodontic brackets. Diving and Hyperbaric Medicine. 2022 June 30;52(2):97–102. doi: 10.28920/dhm52.2.97-102. PMID: 35732281.)
Introduction: This study investigated the effect of pressure variations to which divers are subjected on shear bond strength of orthodontic brackets bonded to teeth with resin modified glass ionomer cement (RMGIC) or composite resin.
Methods: Eighty extracted premolars were randomly divided into two groups. Group 1: orthodontic brackets were bonded with RMGIC. Group 2: orthodontic brackets were bonded with composite resin. Each group was further divided into two subgroups. Subgroup A: The samples were kept at sea level pressure (101 kPa). Subgroup B: The samples were pressurised once from 101 kPa to 405 kPa for five minutes, then depressurised to 101 kPa. Shear bond strength was then measured.
Results: Shear bond strength of brackets bonded with RMGIC in the simulated diving group was significantly less than that of the sea level pressure group ($P = 0.019$), while no significant difference was found between the simulated diving group and sea level pressure group for brackets bonded with resin cement ($P = 0.935$). At sea level pressure, there was no significant difference between shear bond strength of brackets bonded with RMGIC and composite resin ($P = 0.83$). In simulated diving conditions, there was a statistically significant difference between shear bond strength of brackets bonded with the RMGIC and composite ($P = 0.009$).
Conclusions: Pressure changes during scuba diving may have an adverse effect on the retention of brackets bonded with RMGIC. Using composite resin for bonding brackets appears to be good strategy for patients such as divers who will be exposed to pressurised environments.

Introduction

In light of overwhelming popularity of scuba diving, general dental practitioners should be prepared to address complications arising as a result of diving and to provide patients with accurate information.[1] The relevant conditions for dentists who treat divers include diving-associated headache, sinus and middle ear barotrauma, trigeminal or facial nerve baroparesis (pressure-induced palsy), mouth piece associated herpes infection, pharyngeal gag reflex, temporomandibular joint disorder, barodontalgia (barometric-related dental pain) and barotrauma (barometric-related tooth injury).[2]

The changes in volume inside the body's gas-containing cavities associated with the changing ambient pressure, can cause several adverse effects, which are referred to as barotrauma.[1] Dental barotrauma refers to mechanical dental injuries related to barometric pressure changes. It can manifest as tooth fracture (also called barodontocrexis), restoration fracture, and dislodgement of crowns etc.[3] Other than a need for dental treatment, potential consequences include aspiration or swallowing of the dislodged restoration or dental fragment, and pain which may lead to incapacitation while diving and premature discontinuation of the planned dive.[4,5] Previous studies have reported that pressure changes can affect retention of restorations,[4,6] crowns,[7,8] orthodontic bands[9] and endodontic posts.[10–13]

With the increasing number of divers, it is inevitable that the dentist will have orthodontic patients who participate in diving.[14] Orthodontic treatment involves using fixed or removable appliances on teeth to correct their position. The success of a fixed dental appliance depends on the metal attachments (brackets and bands) being securely attached to the teeth so that they do not become loose during treatment. Brackets are usually attached to the incisors, canines and premolars, whereas bands are more commonly used on the molars. The most common adhesives used for attaching bands to teeth are conventional glass ionomer luting cement and resin modified glass ionomer luting cement.[15] To attach brackets to teeth, composite resin and resin modified glass ionomer cement are commonly used.[16]

It is important to be aware of the effect of pressure changes on orthodontic components in terms of retentive strength,

as the potential danger resulting from dislodgement of such components during a dive is obvious. One study assessed the effect of environmental pressure on the retentive strength of cements for orthodontic bands,[9] showing that strength of bands cemented with conventional glass ionomer luting cement is reduced after pressure cycling. Whether the pressure variations that divers are exposed to affect the retention of orthodontic brackets is still unknown.

The aim of the present study was to investigate the effect of pressure variations to which divers are subjected on shear bond strength of orthodontic brackets bonded with resin modified glass ionomer cement (RMGIC) or composite resin. The null hypothesis was that, regardless of the type of cement used, the shear bond strength of orthodontic brackets would not change after simulated dives.

Methods

Ethical approval was obtained prior to the study from our Institutional Ethics Committee (protocol ref no. 579/2021-22).

TEETH

Eighty extracted human premolars were used in the study. Tooth inclusion criteria included absence of endodontic treatment, carious lesions, restorations and enamel defects such as enamel hypoplasia, enamel hypomineralisation or visible cracks. The selected teeth were disinfected with 70% alcohol for 30 minutes. Soft tissue and calculus was removed by ultrasonic scaling. Teeth were stored in distilled water at room temperature and used within six months of extraction.

The teeth were embedded using autopolymerising acrylic blocks, with the buccal surface parallel to the load direction under shear bond strength testing. The facial surfaces of teeth were cleaned with a mixture of water and pumice. The teeth were rinsed thoroughly with water and dried with compressed air.

ORTHODONTIC BRACKETS

Eighty premolar brackets (0.022 MBT Preadjusted Gemini stainless steel, 3M Unitek, USA) were used. The average surface of the bracket base was 9.6 mm^2.

BONDING PROCEDURE

Teeth were randomly divided into two groups of 40 premolars.

Group 1: Brackets bonded with RMGIC (GC Fuji Ortho LC; GC International Corp., Tokyo, Japan). The enamel surface was etched with 37% phosphoric acid gel for 30 seconds, then rinsed with water spray for 20 seconds and left moist. Cement mixing was done according to manufacturer's instructions. On a mixing pad, one level large scoop of powder to two drops of liquid was dispensed. The powder was divided into two equal parts. The first portion was mixed with liquid for about 10 seconds. After this the remaining powder was incorporated and mixed thoroughly for 10 seconds. The mixture was placed on the bracket base. A bracket positioning gauge was used to place the bracket on the mid-buccal surfaces of the teeth at least 4 mm away from the buccal cusp ridges, while the bracket slot was perpendicular to the tooth coronal long axis. Using a force gauge, a 300 g compressive force was applied to each bracket to reduce and standardise the adhesive thickness. Excess cement was removed with a dental probe.

Group 2: Brackets bonded with composite resin (Transbond XT; 3M Unitek, St Paul, Minnesota, USA). The enamel surface was etched with 37% phosphoric acid gel for 30 seconds, then rinsed with water spray for 20 seconds and dried with oil-free compressed air for 20 seconds. According to manufacturer instruction, the primer (Transbond XT Primer; 3M Unitek, St Paul, Minnesota, USA) was applied to the etched surface. The single-component composite resin was then applied to the bracket base and placed on the tooth in a similar manner to group 1.

All the brackets of both groups were cured using an Ortholux LED Curing Light (3M Unitek, Monrovia, CA, USA) for 10 seconds each from the occlusal, mesial, distal and gingival aspects. After light curing, specimens were stored in distilled water at 37°C for 24 hours to allow complete polymerisation of the bonding material.

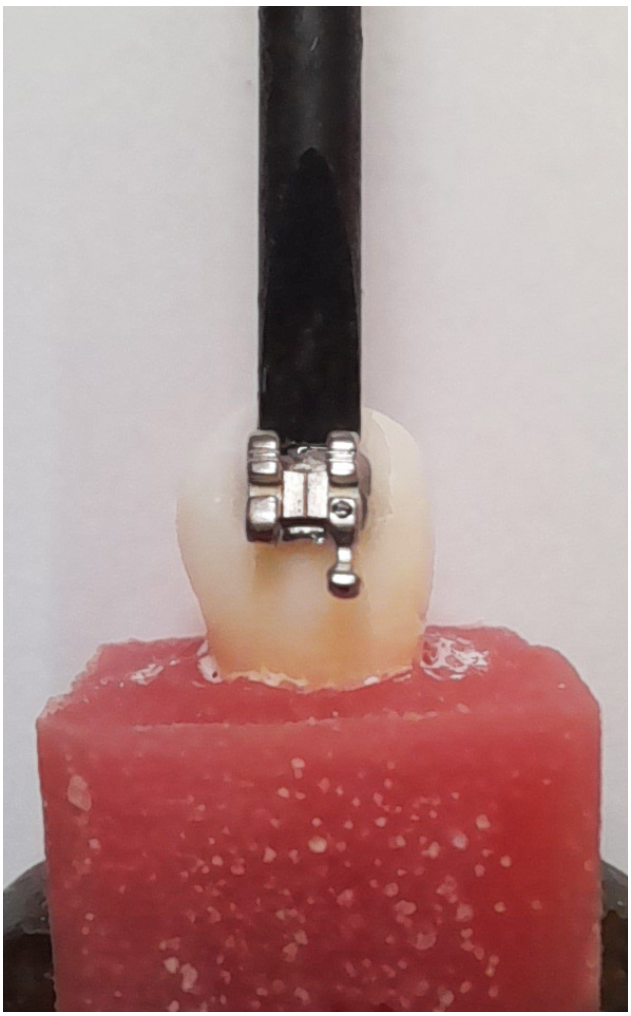
Each group was randomly divided into two subgroups A, B of 20 samples each.

- Subgroup A (sea level pressure). The samples were kept at normal atmospheric/sea level pressure (~101 kPa) and treated as a control.
- Subgroup B (simulated dive). The samples were exposed to pressure to simulate a dive. The simulator was a customised pressure chamber (Ashirwad Manufacturing, India) with a pressure controller programmed to change internal pressure between 101 to 405 kPa. The samples were placed in the pressure chamber in an open glass container soaked in distilled water. Compressed air was introduced to increase the pressure from 101 to 405 kPa at a rate of 101 $kPa \cdot min^{-1}$ to simulate a descent. Once the maximum pressure of 405 kPa was reached it was maintained for five minutes and then decreased back to 101 kPa at 101 $kPa \cdot min^{-1}$ to simulate ascent. This procedure was designed to simulate conditions that a recreational scuba diver might experience on a single dive to 30 metres depth.

SHEAR BOND STRENGTH TESTING

Each specimen was loaded into a universal testing machine (Five Star Manufacturing, India), with the long axis of the specimen kept perpendicular to the direction of the applied force. A knife-edge chisel was positioned in the occluso-gingival direction and in contact with the bonded specimen

Figure 1
Shear bond strength testing configuration



(Figure 1). Bond strength was determined in the shear mode at a crosshead speed of 0.5 mm·min^{-1} until fracture occurred. The values of failure loads in newtons (N) were recorded and converted into megapascals (MPa) by dividing the failure load (N) by the surface area of the bracket base.

STATISTICAL ANALYSIS

Descriptive statistics, including the mean, standard deviation, standard error, and minimum and maximum values, were calculated for each of the groups tested. The Kolmogorov-Smirnov test determined the data were normally distributed and parametric tests were therefore used. One-way analysis of variance (ANOVA) and Tukey multiple comparison tests were used to compare shear bond strength among the groups. Significance for all statistical tests was predetermined at $P < 0.05$.

Results

Descriptive statistics for the shear bond strength of all groups are presented in Table 1. Shear bond strength of brackets bonded with RMGIC was significantly less in the simulated diving group than the sea level pressure group ($P = 0.019$), while no significant difference was found between the simulated diving group and sea level pressure group for brackets bonded with resin cement ($P = 0.935$). In the sea level pressure group there was no significant difference between shear bond strength of brackets bonded with RMGIC and composite resin ($P = 0.83$). In the simulated diving group, there was a statistically significant difference between shear bond strength of brackets bonded with the RMGIC and composite ($P = 0.009$).

Discussion

With a growing number of divers, dentists will increasingly encounter oral complications of pressure changes and these would require careful attention.[17] These conditions potentially may cause distraction or incapacitation that could jeopardise diving safety.

Fixed orthodontics is a type of orthodontic appliance where brackets are bonded to teeth. The bond strength between the enamel surface and bracket must withstand the mechanical and thermal effects of the oral environment.[18] To best of our knowledge, this is the first investigation that has assessed the effect of pressure change on the bond strength of orthodontic brackets. In the present *in vitro* study, orthodontic brackets bonded with two different types of cement were subjected to a single simulated dive in a pressure chamber and the shear bond strength was investigated. RMGIC and composite resins were selected because they are the most frequently used bonding material in orthodontics.

In the constant sea level pressure condition, mean shear bond strength of RMGIC after acid etching of the enamel surface was similar to those of composite resin. This was consistent with previous studies.[19,20] However, after a simulated dive the brackets bonded with RMGIC showed significantly lower shear bond strength than the sea level pressure group. In contrast, in brackets bonded with composite resin, the shear bond strength was not affected by the simulated dive. The null hypothesis was therefore rejected.

During descent to depth gas-containing anatomic spaces will be compressed, and during ascent, any compressed gas introduced to these spaces will expand.[21] Problems arise when gas containing spaces cannot expand or contract to equalise internal and ambient pressures. Thus, bubbles and porosities in the cement or interfacial surfaces could be affected during pressure change. In diving, stress is induced when air contained in porosities in the cement layer attempts to compress. Conversely when returning to the surface, the enclosed gas expands inducing further stress. The accumulated stress of these compression- expansion cycles can cause cracks and/or propagation of existing cracks and flaws inside the cement layer and/or along the internal surface.[8] Each porous material might have blind pores, through pores (open porosity) and closed pores.

Table 1
Shear bond strength (MPa) comparisons between control (constant sea level pressure) and dive (simulated dive) sub-groups of orthodontic brackets bonded to teeth using composite resin or resin modified glass ionomer cement (RMGIC)

Cement	Constant sea level pressure (n = 20)		Simulated dive (n = 20)		P-value
	Mean (SD)	Range	Mean (SD)	Range	
RMGIC	11.35 (1.27)	8.8–13.2	10.03 (1.87)	5.5–12.4	0.019
Composite resin	11.72 (1.07)	9.1–13.7	11.46 (1.25)	8.6–13.6	0.935
P-value	0.83		0.009		

The blind pore terminates inside the material. The through pores pass through and through the material. Porosities that include closed pores are potentially most influential on the mechanical properties of the material.[12] The effects of pressure are expected to be less when porosity or air inclusion is lower.[11]

The formation of glass ionomer cement requires a chemical reaction between an acid and base reagent. The fluoraluminosilicate glass powder (base) and the polycarboxylic/water (acid) must be mechanically mixed prior to use.[22] It is recognised that such mixing methods may result in the incorporation of air porosity in the cement.[23–25] This may explain our findings that the hand-mixed RMGIC is affected by pressure exposure.

Light activated composite resin adhesives are single-component materials stored in opaque packages. Single-component resins are convenient because no mixing is required, thus there is less chances of incorporation of air porosities.[26] This could be the reason that composite resin was not affected by pressure exposure.

According to one study, the brittle cements are affected more by environmental pressure cycling.[8] Generally, resins are less brittle and more fracture-resistance than RMGIC.[26] This may be another reason that the shear bond strength of brackets bonded with composite resin was not significantly affected by pressure exposure.

There is no universally accepted minimum clinical bond strength for orthodontic attachments. However the strength should withstand normal orthodontic and masticatory forces (8–9 MPa).[27] On the other hand, adhesive forces should not be too strong in order to avoid enamel loss after debonding (40–50 MPa).[28] In the present study, the mean shear bond strength of brackets bonded with RMGIC in the simulated diving group was 10.03 MPa, ranging from 5.5 to 12.4 MPa. This indicates that some samples failed below optimal bond strength. Although the clinical condition and the forces applied to the teeth in the oral cavity are different from the design of this study, these numbers do have clinical significance.

A direct comparison between the results of the present study and those of others is somewhat difficult because of variety of dental components and material used. However, despite these variations, the present results may, at least in part, be compared with those of previous studies in which similar test methods and material were used. One study found that the retention of full cast crowns cemented with resin was not affected after pressure cycling.[7] Another investigated the effect of cyclic environment pressure changes on the retention of crowns on extracted teeth.[8] That study found that crowns cemented with either zinc phosphate cement or conventional glass ionomer cement had significantly reduced retention, whereas retention of crowns cemented with resin cement was unaffected by pressure cycling.

In the present study all the variables that could have an effect on shear bond strength such as pre-treatment of teeth, placement of light source, curing protocols and storage protocols of the prepared specimens were kept constant. Thus, the only variable affecting the shear bond strength in this study was the effect of pressure exposure on the bonding cement.

The studies which assessed the effect of pressure changes on the dental components, simulated a diving environment by using either hyperbaric chamber[11–13] or a customised pressure chamber.[6,29,30] In this study, a customised pressure chamber was used to simulate diving environment.

The clinical significance of this study should be tempered by its limitations. The oral cavity is a complex environment, with variations in temperature, stresses, humidity, acidity, and plaque. It is impossible to design a laboratory condition that fully reproduces the oral environment. Therefore, further clinical studies are needed to confirm these findings. This study aimed to recreate the conditions of a single simulated dive to 30 metres depth. Commercial and military divers dive more frequently and to greater depths than this. More research is needed to determine how these adhesives perform under higher pressures and for a greater number of pressure cycles.

Conclusions

Within the limitations of this study, it can be concluded that, pressure changes during diving may have an adverse effect on the retention of brackets bonded with RMGIC. Using composite resin for bonding brackets appears to be

good strategy for patients such as divers, who are likely to be exposed to pressurised environments.

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Quantifying drysuit seal pressures in non-immersed scuba divers

Derek B Covington[1], Matthew Spears[2], Richa Wardhan[2], Meghan Brennan[2], Yasmin Islam[3], Andrew D Pitkin[2]

[1] *Department of Anesthesiology, Center for Hyperbaric and Environmental Physiology, Duke University, Durham (NC), USA*
[2] *Department of Anesthesiology, University of Florida College of Medicine, Gainesville (FL), USA*
[3] *Department of Ophthalmology, University of Florida College of Medicine, Gainesville (FL), USA*

Corresponding author: *Dr Derek Covington, Department of Anesthesiology, Center for Hyperbaric and Environmental Physiology, Duke University, DUMC 3094, Durham, NC 27710, USA*
derek.covington@duke.edu

Keywords
Diving; Latex; Paresthesia; Silicone; Technical diving

Abstract
(Covington DB, Spears M, Wardhan R, Brennan M, Islam Y, Pitkin AD. Quantifying drysuit seal pressures in non-immersed scuba divers. Diving and Hyperbaric Medicine. 2022 June 30;52(2):103–107. doi: 10.28920/dhm52.2.103-107. PMID: 35732282.)
Introduction: Drysuits use flexible neck and wrist seals to maintain water-tight seals. However, if the seals exert too much pressure adverse physiological effects are possible, including dizziness, lightheadedness, syncope, and paresthesias in the hands. We aimed to quantify the seal pressures of neck and wrist seals in non-immersed divers.
Methods: We recruited 33 diving volunteers at two dive facilities in High Springs, Florida. After a history and physical exam, we measured vital signs as well as wrist and neck seal pressures using a manometer system.
Results: The mean (SD) seal pressure of the right wrist seals was found to be 38.8 (14.9) mmHg, while that of the left wrist seals was 37.6 (14.9) mmHg. The average neck seal pressure was 23.7 (9.4) mmHg. Subgroup analysis of seal material demonstrated higher mean sealing pressure with latex seals compared to silicone; however, this difference was not statistically significant.
Conclusions: Drysuit seal pressures are high enough to have vascular implications and even potentially cause peripheral nerve injury at the wrist. Divers should trim their seals appropriately and be vigilant regarding symptoms of excessive seal pressures. Further research may elucidate if seal material influences magnitude of seal pressure.

Introduction

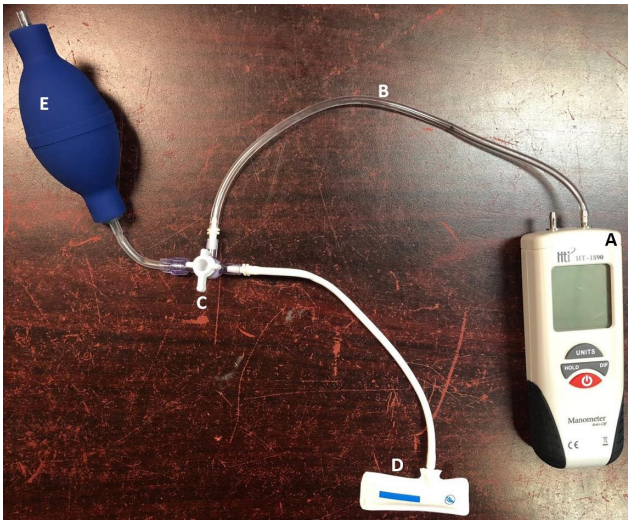
Proper exposure protection is vital for a variety of outdoor pursuits. Appropriate exposure protection not only provides comfort, but it also aids in preventing complications secondary to cold, such as decreased manual dexterity, altered mental status, and hypothermia. Cold environments have been historically associated with occupational exposures; however, more recently these are increasingly recreational in nature.[1]

For those participating in aquatic sports, exposure protection is even more important because water has a much greater conductive transfer of heat compared to that of air, which may lead to a rapid loss of body heat and increase the risk of hypothermia.[2] In addition to preventing hypothermia, individuals pursuing scuba diving seek thermal protection to maintain their manual dexterity, which is vital to safely retrieve equipment, perform underwater tasks, such as inflating a lift bag or adding breathing gas to a closed-circuit rebreather.

Furthermore, research has shown that thermal effects can influence the efficiency of decompression and that remaining warm during the decompression process significantly reduces the risk of decompression sickness (DCS).[3] It is postulated that vasoconstriction induced by cold body temperatures can increase the degree of gas bubble formation during a dive and increase the risk of the diver developing DCS.[4] Therefore, it is crucial that scuba divers don the proper exposure protection for the conditions in which they will be diving so they stay comfortable and warm as well as maintain manual dexterity and limit the risk of DCS.

For dives in temperate waters or short dives, wetsuits often provide adequate exposure protection. However, for longer dives and/or dives in cold water, drysuits are the preferred exposure protection. Drysuits are composed of durable, waterproof material with elastic seals at the wrist and neck that prevent water penetration into the suits. To facilitate a water-tight seal, drysuits apply pressure via these neck and wrist seals. The seals must be comfortable enough to dive and to use over long periods of time as well as durable enough to withstand abrasions and repeated use. Not surprisingly, if these seals exert too much pressure, they can have adverse physiological effects. For instance, neck seals that are too tight can induce dizziness, lightheadedness, and/or syncope due to pressure exerted on the carotid sinus. Similarly, divers with wrist seals that are too tight may experience paresthesias, weakness, or numbness in their

Figure 1
The device assembled to measure the drysuit seal pressures; A – manometer; B – plastic tubing; C – a three-way stop cock; D – disposable vinyl neonatal blood pressure cuff; E – bulb inflator



hands (likely due to radial nerve involvement), which may lead to decreased manual dexterity and inability to complete underwater tasks. Furthermore, these symptoms persisting after a dive may complicate the differential diagnosis of DCS.

Although the consequences of excessively restrictive drysuit seals are commonly encountered by drysuit divers, especially novice drysuit divers, the seal pressures of drysuit neck and wrist seals have yet to be quantified. The purpose of this study was to quantify the seal pressures of neck and wrist seals in non-immersed divers. We hypothesised that these seals exert pressures consistent with impedance of venous blood flow as suggested by the potential side effects of these seals, such as craniofacial vascular engorgement, syncope, and upper extremity paresthesias.

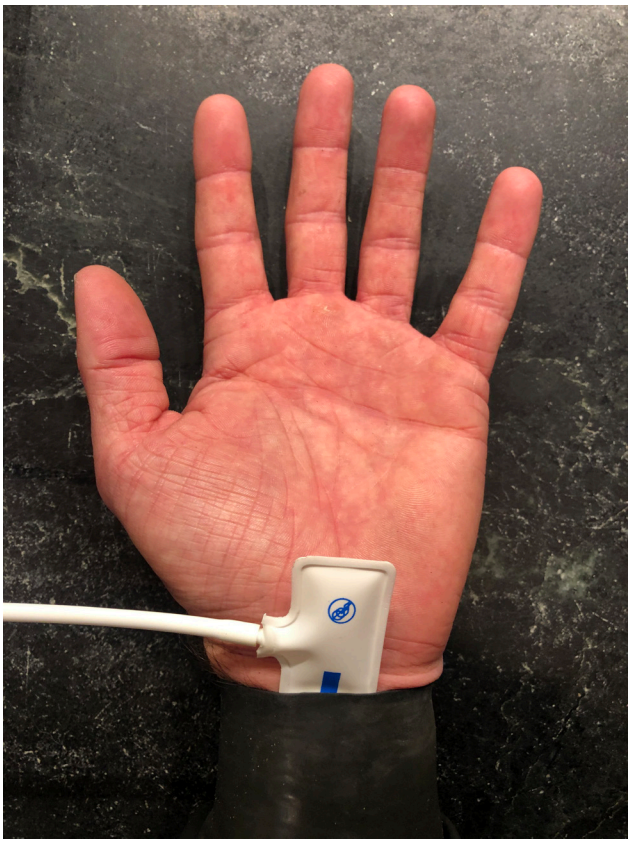
Methods

Institutional Review Board (IRB 201602349) approval at the University of Florida was obtained.

The study was performed from 0800 to 1700 over three days at two dive facilities in High Springs, Florida, using subjects recruited at those locations. All subjects denied significant cardiopulmonary disease, as well as any significant medical history. All subjects were certified drysuit divers who had at least two months of drysuit diving experience and at least 10 dives in the drysuit they used in the study. Exclusion criteria included historical or physical examination findings consistent with cardiopulmonary disease.

In order to satisfy ethics approval requirements, a brief health survey, diving history, and measurements of non-invasive blood pressure, oxygen saturation, and heart rate before and after drysuit donning were performed. Per the ethics approval committee, these assessments were to ensure subject safety.

Figure 2
The neonatal blood pressure cuff slightly inflated, zeroed, and advanced under the drysuit seal along the volar aspect of the wrist



To determine the seal pressure of the drysuit seals, a manometer (HT-1890, HT Instruments, Faenza, Italy) was connected to a three-way stopcock via clear, plastic pressure tubing as well as a neonatal non-invasive blood pressure cuff (Welch Allyn, Neonate #1, M1866A, Skaneateles Falls, New York, USA) and an inflation bulb (Figure 1). The pressure was zeroed by partially inflating the non-invasive cuff with the bulb and turning the stop cock to allow the cuff to communicate with the manometer. With the subject's arm supine, the non-invasive cuff was advanced under the seal on the medial volar surface of the wrist (Figure 2). The same procedure was followed for the contralateral wrist. After measurement of seal pressure on the wrists, the same cuff was placed underneath the drysuit neck seal in the right anterolateral and left anterolateral positions to obtain seal pressures. These two neck measurements were then averaged. The subject then doffed the drysuit and rested for 120 seconds before a final set of blood pressure, heart rate and oxygen saturation measurements were recorded as described above.

All statistical analyses were performed using JMP® Software (JMP® v15, Cary NC, USA). Summary statistics were

calculated for demographic data for study participants and seal pressures for each type of drysuit seal: latex, silicone, and neoprene. Two sample *t*-tests were used to compare seal pressure means between latex and silicone. Only one study participant used a neoprene drysuit; therefore, no comparisons were completed between neoprene drysuits and other types of drysuits. Statistical significance was set at $P < 0.05$ and pressure data are presented as mean (SD).

Results

This study involved 33 subjects comprising 24 males (73%) and 9 (27%) females. The mean age was 37.9 (SD 11.2) with a range from 19 to 69 years. The mean years of diving experience was 16.7 (9) with a range of 1 to 40 years. The mean total number of dives was 2,037 (1,854) with a range of 60 to 7,000 dives, while the mean time of diving in a drysuit was 10.45 (7) years with a range from 2 months to 27 years (Table 1).

Wrist and neck seal pressures are reported in Table 2. Subgroup analysis showed that the wrist seals made of latex had higher mean seal pressures than those made of silicone. There was no significant measurable difference between right and left seals. No subjects used neoprene wrist seals. Subgroup analysis of the latex neck seals also yielded higher mean seal pressures compared to those made of silicone and neoprene. One subject had a neoprene neck seal, which had a seal pressure of 22.7 mmHg (Table 3).

Although the latex seals exerted higher pressures compared to those made of silicone and neoprene, statistical analysis via unpaired *t*-tests did not support statistically significant differences in these pressures. The statistical analysis is presented in Table 4.

Discussion

We found that the average sealing pressures of the wrist and neck seals were substantial and likely of enough magnitude to induce physiological changes, such as paresthesias and syncope. These findings help explain the commonly reported symptoms of hand paresthesias and occasional syncopal episodes in divers using drysuits.

Previous research investigating pressure and nerve injuries has shown that pressures of 30 mmHg can limit axonal transport and result in nerve dysfunction as well as endoneurial oedema.[5] The same study also found that pressures of 50.25 mmHg applied to the carpal tunnel for 120 seconds can alter the structure of the myelin sheaths, leading to permanent nerve damage.[5] Our findings show that the average pressures generated by the wrist seals of drysuits exceed the pressure required to limit axonal transport and epineural blood flow. Furthermore, a large percentage of the subjects demonstrated seal pressures greater than that required to damage the myelin sheath, which could lead to permanent nerve damage.

Table 1
Subject demographics

Demographic	Mean (SD)	Median (range)
Age (years)	37.9 (11.2)	35 (19–69)
Height (cm)	175.2 (9.2)	178 (155–190)
Weight (kg)	79.7 (15.6)	77.3 (47.7–113.4)
Body mass index ($kg \cdot m^{-2}$)	25.7 (3.7)	26.1 (18.6–34.9)
Diving experience (years)	16.7 (9)	18 (1–40)
Total dives	2,037 (1,854)	1,500 (60–7,000)
Drysuit experience (years)	10.5 (7)	10 (0.2–27)

Table 2
Drysuit seal pressures (mmHg) measured in 33 subjects

Seal	Mean (SD)	Median (range)
Right wrist	38.8 (14.9)	34.8 (15.3–66.3)
Left wrist	37.6 (12.8)	37.7 (9.2–62.2)
Neck	23.7 (9.4)	22.5 (6.8–44.5)

Table 3
Drysuit seal pressures (mmHg) by seal material

Seal	Mean (SD)	Median (range)
Latex (*n* = 28)		
Right wrist	39.7 (14.7)	36.0 (15.3–66.3)
Left wrist	38.2 (12.8)	37.8 (9.2–62.2)
Neck	24.4 (10.8)	22.9 (7.3–43.6)
Silicone (*n* = 5)		
Right wrist	34.1 (16.8)	31.7 (17.7–57.5)
Left wrist	34.2 (14.1)	30.3 (18.9–56.7)
Neck (*n* = 4)	18.8 (10.7)	18.8 (5.7–31.9)
Neoprene (*n* = 1)		
Neck	22.7	22.7

Table 4
Unpaired *t*-tests assessing differences in exerted pressure (mm Hg) of latex and silicone drysuit seals

Comparison	Absolute mean difference (95% CI)	*P*-value
Right wrist latex vs silicone	5.6 (-9.2 to 20.4)	0.45
Left wrist latex vs silicone	4 (-8.8 to 16.8)	0.53
Neck latex vs silicone	7.43 (-2.64 to 17.5)	0.22

If only the latex seals are considered, the average seal pressures in this study were even higher. These high seal pressures and the fact that some subjects reported dive times exceeding 10 hours are concerning for nerve injury; however, there are no reports in the literature of upper extremity nerve injury due to drysuit wrist seals. It is possible that the seal pressures of the wrist seals may be lower during diving secondary to immersion physiology, which can be characterised by an increased shunting of blood to the central circulation, diuresis, and intravascular depletion.[6,7] It is also possible that these wrist seals induce subclinical neuronal damage.

Previous research investigating pressure and venous return found that initial venous narrowing of superficial and deep leg veins occurs between 30 and 40 mmHg when in a seated or standing position.[8] The same study found that complete occlusion of superficial and deep leg veins occurs at 20 to 25 mmHg when in the supine position and 50 to 60 mmHg when in the seated position.[8] In the aforementioned study, cuff pressures were gradually increased over 30 seconds while occlusion was observed via ultrasound. Our findings suggest that the pressure exerted by the drysuit seals on the neck are sufficient to occlude venous return from the head and neck. It is possible that this restriction of venous outflow could lead to increasing venous pressure and intracranial pressure, which can result in decreased cerebral perfusion pressure (CPP).[9] It is worth noting the latex seals are narrower than the venous tourniquet, which may lead to differences in pressures exerted into deeper tissues.

A study on syncope, cerebral perfusion, and oxygenation found that presyncopal symptoms coincided with an excessive reduction in mean middle cerebral artery blood flow velocity.[10] In the same study, it was noted that progressive drops in mean arterial pressure and CPP were observed when vasovagal syncope was induced under laboratory conditions. As the pressures exerted by the neck seals were found to be higher than what would be expected within many of the veins in the superficial neck, it is likely these seals are inhibiting craniofacial drainage, which could increase intracranial pressure and subsequently reduce CPP. These findings in the above-mentioned study and those in the present study lead us to believe that a decrease in CPP may explain the syncopal episodes reported by some divers using drysuits.

We also found that the average seal pressure was lower in divers with silicone seals, although these differences were not statistically significant. Thus, our research suggests that divers who are sensitive to the effects of seal pressures should consider the use silicone seals instead of the more commonly used latex seals. However, it is important to note that variances in seal design and material property could also impact seal pressures as well. Considering only one subject had neoprene seals, we cannot make conclusions regarding this material's seal pressure.

Limitations of this study include the relatively small number of subjects. In addition, this study included only five subjects with silicone wrist seals, four subjects with silicone neck seals, and one with a neoprene neck seal. All remaining seals were latex. In addition, as mentioned above, seal design and differences in material properties could also influence seal pressures. Furthermore, this study assessed only non-immersed divers. Thus, the described physiologic changes associated with drysuits when the diver is topside may not translate to the haemodynamic changes induced by diving and immersion. As immersion phenomena and activity underwater induce a variety of significant physiologic changes, such as increases in preload, decreases in heart rate, and others, it is likely that the blood flow through these seals would be different. Lastly, drysuit seals will stretch over time and with use. Thus, the seal pressures will likely decrease over time. Consequently, our results represent only a single point in time for each subject and for each seal.

Conclusions

Drysuit seals exert a significant amount of force to prevent water intrusion. Although the average seal pressure may vary slightly between divers and seal materials, the seal pressures are of a magnitude consistent with vascular implications and even possible neural injury, especially in the setting of latex wrist seals. Divers wishing to avoid the effects of these seals should be especially careful to trim their seals appropriately. Further work should focus upon the impact of seal material on seal pressure.

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A review of snorkelling and scuba diving fatalities in Queensland, Australia, 2000 to 2019

John Lippmann[1,2,3]

[1] *Australasian Diving Safety Foundation, Canterbury, Victoria, Australia*
[2] *Department of Public Health and Preventive Medicine, Monash University, Victoria, Australia*
[3] *The Royal Lifesaving Society – Australia, Sydney, Australia*

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

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Age; Breath-hold diving; Cardiac; Chain of events analysis; Diving deaths; Obesity

Abstract
(Lippmann J. A review of snorkelling and scuba diving fatalities in Queensland, Australia, 2000 to 2019. Diving and Hyperbaric Medicine. 2022 June 30;52(2):108–118. doi: 10.28920/dhm52.2.108-118. PMID: 35732283.)
Introduction: This study examined all known diving-related fatalities in Queensland, Australia, from 2000 to 2019 to determine likely causes and potential countermeasures.
Methods: Data were extracted from the Australasian Diving Safety Foundation fatality database, including previously published reports. The National Coronial Information System was searched to identify diving-related deaths in Queensland for 2014–2019 and data were extracted, analysed, and combined with previously published data covering the period 2000–2013. Descriptive statistics and parametric and non-parametric tests were used to analyse these data.
Results: There were 166 snorkelling and 41 scuba victims identified with median ages of 59 and 49 years respectively, and 83% of snorkel and 64% of scuba victims were males. One quarter of snorkel and 40% of scuba victims were obese. Two-thirds of the snorkellers and three quarters of scuba divers were overseas tourists. Contributory predisposing health conditions were identified in 61% of snorkel and 50% of scuba victims. Nine scuba victims died on their first dive.
Conclusions: The increase in snorkelling deaths likely reflects increased participation, higher age, and poorer health. The main disabling condition in both cohorts was cardiac-related. Pre-existing health conditions, poor skills, inexperience, poor planning, supervision shortcomings and lack of effective buddy systems featured in both cohorts, and apnoeic hypoxia in breath-hold divers. Suggested countermeasures include improved education on the importance of health and fitness for safe diving and snorkelling, increased emphasis on an honest and accurate pre-activity health declaration and subsequent implementation of appropriate risk mitigation strategies, improved supervision, better buddy pairing, and on-going education on the hazards of extended apnoea.

Introduction

Extending from just south of the Tropic of Capricorn towards the coastal waters of Papua New Guinea, the Great Barrier Reef (GBR) is approximately 2,300 km long and 72 km wide at its widest point, covering an area of almost 350,000 square kilometres off the Queensland coast. It is reportedly the largest system of coral reefs, mangroves and estuarine environments worldwide. The abundance and diversity of marine life is immense with some 400 species of coral and 1,500 species of fish.[1] As such, it has long been a mecca for scuba divers and snorkellers, both local, interstate, and international. Beyond the GBR is the Coral Sea with some spectacular dive sites, and, in addition to the tropical waters, southern Queensland hosts a variety of temperate water species and is popular with predominantly local divers.

Scuba diving and snorkelling are conducted in a hostile environment and some fatalities are inevitable, whether resulting from adverse conditions, inexperience, equipment issues, inadequate health and fitness, or attitudinal and oversight shortcomings. Diving-related tourism is an important income source for Queensland and, in 1992, a regulated Code of Practice (COP) for diving activities was introduced. This has been periodically updated, the latest version being released in 2018 with another version due for release in 2022.[2] The COP is regulated and overseen by a team of specialised diving inspectors from WorkSafe Queensland who investigate serious incidents occurring in a diving workplace, which includes commercial recreational snorkelling and scuba diving operations. Fatalities are also investigated by the police and subsequently the coroner.

Although fatalities occurring in Queensland are sometimes well-publicised and may appear to be common, given the amount of snorkelling and scuba diving that occurs there, especially on the GBR, the number of fatalities appears to be relatively low. In an earlier review, it was estimated that the fatality rate for international scuba divers in Queensland was considerably lower than estimates from a variety of

other locations and it was postulated that the existence of the COP may help to mitigate the risks.[3]

The aim of this research was to examine all known diving-related fatalities in Queensland waters from 2000 to 2019 to determine likely causes and potential countermeasures.

Methods

This represents a complete, or near-complete, case series of snorkelling and scuba diving fatalities that occurred in Queensland waters from 1 January 2000 to 31 December 2019. For inclusion, the scuba diver must have been reported to have been wearing a scuba set.

ETHICS APPROVAL

Ethics approvals for the collection and reporting of these data were received from the Victorian Department of Justice Human Research Ethics Committee to access the National Coronial Information System (NCIS; CF/21/18434)[4] as well as the Queensland State Coroner.

SEARCH

Historical data (1970–2000) were obtained from the Australasian Diving Safety Foundation (ADSF) diving fatality database and Project Stickybeak reports.[5–8] Information gathered during previous published investigations for 2000 to 2013[9,10] were reviewed and relevant further data extracted from these, and, where necessary the underpinning coronial documents.

A comprehensive keyword search was made of the NCIS for scuba diving-related deaths in Queensland for the period 1 January 2014 to 31 December 2019. Keywords included scuba, compressed air, compressed gas and div*, snorkel*, breath-hold and div*, and underwater fishing. Data obtained from the NCIS was matched with those held on the ADSF fatality database. Additional reports were obtained directly from the Queensland State Coroner.

REVIEW PROCEDURE AND OUTCOME MEASURES

The investigator reviewed all datasets. Data 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, training and experience, origin of victims, dive location and conditions, buddy circumstances and oversight, dive purpose and depth, equipment used and resuscitation factors.

ANALYSIS

A chain of events analysis (CEA) was performed for each case using existing templates.[10,11] Descriptive analyses based on means and standard deviations (SD) or medians and interquartile ranges (IQR), and Mann-Whitney U tests for comparisons of age or body mass index (BMI), as appropriate, were conducted using SPSS® Version 25 (IBM Armonk, NY; 2017). The level of statistical significance assumed was $P = 0.05$. Annual fatality rates and 95% confidence intervals were calculated based on an exact binomial method as implemented in the binomial test in the R statistical package.[12]

TOURISM RESEARCH AUSTRALIA DATA

Since 2005, Tourism Research Australia has conducted annual surveys of international and national tourists who have visited various Australian states and territories. The International Visitor Survey samples 40,000 departing, short-term international visitors over 15 years of age annually. It is conducted in the departure lounges of major international airports and utilises computer-assisted personal interviewing. The survey results are weighted to data on international visitor numbers over the period.[13]

While these data can measure overseas visitors to Queensland who dived on their trip to Australia, they are not sufficiently detailed to determine if these activities were done in Queensland. However, it was evident from the data that (depending on the year) people who had visited Queensland accounted for 80–90% of snorkellers and scuba divers. Based on Tourism Research Australia advice the denominator used to calculate death rates was therefore reduced by a commensurate amount.

Results

HISTORICAL

Thirty-nine percent of all snorkelling and scuba diving deaths in Australian waters from 1970 to 2019, inclusive, occurred in Queensland, comprising 55% of the total snorkelling and 24% of the scuba fatalities. The proportion of scuba deaths in Queensland remained relatively stable over the period. However, the proportion of snorkelling deaths occurring in Queensland waters was subject to a variety of peaks and troughs, likely related to rises and falls in tourist numbers and increased snorkelling activity elsewhere. Snorkel and scuba diving fatalities in Queensland and Australia as a whole from 1970–2019 are displayed in Figure 1.

During this extended period there were a total of 352 diving-related deaths in Queensland, including 102 in scuba divers, 235 in snorkellers/breath-hold divers, and 15 in divers using surface-supplied breathing apparatus (the latter are not addressed in this report). While the average annual deaths of scuba divers remained stable over time, there was a substantial increase in annual snorkelling deaths over the period. There was also an increase in the ages of both snorkel and scuba victims (Table 1).

Figure 1
Snorkel (SN) and scuba (SC) diving fatalities in Queensland and Australia as a whole from 1970–2019

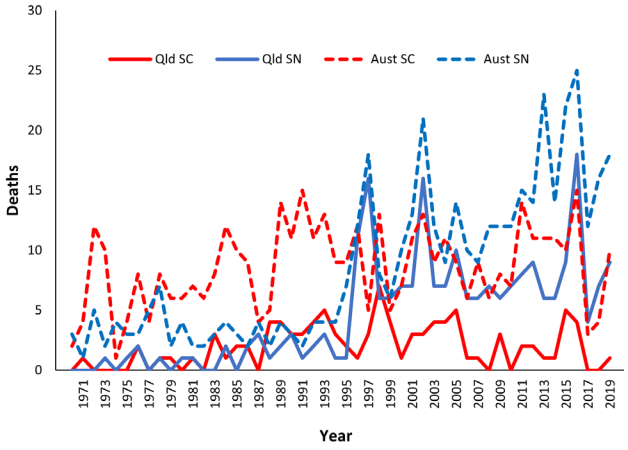


Figure 2
Body mass index categories of snorkel and scuba victims of diving fatalities in Queensland 2000–2019

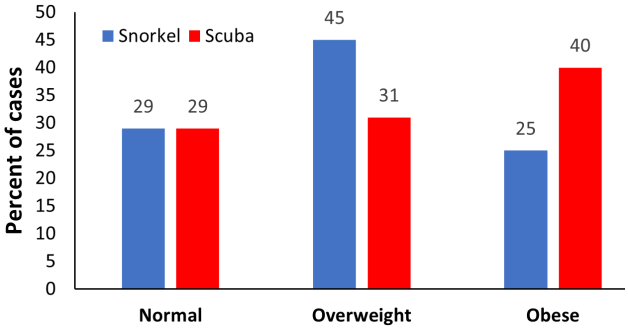


Table 1
Deaths and demographics of snorkel and scuba victims of diving fatalities in Queensland for 1970–1999 and 2000–2019

Parameter	1970–1999		2000–2019	
	Snorkel	Scuba	Snorkel	Scuba
Deaths / Deaths per year	69 / 2.3	61 / 2	166 / 8.3	41 / 2.1
Age, median (IQR)	42 (28, 66)	41 (33, 59)	59 (37, 69)	49 (33, 59)
Sex (% male)	70	75	83	63

STUDY PERIOD 2000 TO 2019

Data were available for 166 snorkelling/breath-hold and 41 identified scuba diving fatality victims in Queensland during this period. These represented 84% of the snorkelling and 23% of the scuba fatalities throughout Australia.

Demographics

The median (IQR) age of the deceased snorkellers in Queensland was 59 (37, 69) which was significantly higher than snorkel victims in other states and territories where the median age was 39 years ($P < 0.001$). The range was 16 to 83 years.

The median (IQR) age of the scuba divers was 48 (32, 57) with a range of 20 to 71 years. Although the median age of scuba victims elsewhere in Australia was lower, at 46 years, this difference was not significant ($P = 0.63$). The snorkel victims in Queensland were significantly older than the scuba victims ($P = 0.02$). Eighty-three percent of the snorkel victims and 64% of scuba victims were males.

The BMI was available for 138 snorkellers, including 117 males and 21 females, with a mean (SD) of 27.6 (5.3) $kg.m^{-2}$ and range of 17.4 to 50.0 $kg.m^{-2}$. Seventy percent of the combined snorkeller group were overweight (45%) or obese (25%) and there was no significant difference between the sexes ($P = 0.87$). Similarly, the BMI was available for 35 of the scuba victims, including 23 males and 12 females. The mean (SD) BMI being 28.0 (4.6) $kg.m^{-2}$ with a range of 20.0 to 37.6 $kg.m^{-2}$. Thirty-one percent were overweight and 40% obese (Figure 2). There was no significant difference between the sexes ($P = 0.92$).

Experience and certification

Thirteen of the snorkellers were documented to have had some formal diving certification. One was a free diving instructor, two were scuba instructors, another two were dive masters and at least eight others were scuba certified. Forty-two percent of the snorkellers had little or no prior experience, and 27% were reported to have been 'experienced'. No relevant information was available for the remaining cases. One half of the experienced snorkellers were diving solo, and most were spearfishing or practicing breath-holding.

Seven of the scuba victims were participating in a 'resort dive' (i.e., a non-certification scuba experience), 16 were certified as open water divers, four as advanced open water

divers, one was an instructor, and another commercially certified. Certification status was unreported in seven cases. Nine of the scuba victims died on their first dive, 12 were novices (0–30 dives post-certification), 14 were 'experienced' (31–200 dives) and at least five 'very experienced' (> 200 dives). No indication of experience was available for one victim. Four of the 'very experienced' victims were over 50 years of age and their deaths appeared to have been cardiac-related.

Location and setting

One hundred and fifty-nine (96%) of the snorkelling incidents occurred in the sea, five in a pool, and one each in a lake and mineshaft. Of those which occurred in the sea, 142 (89%) were on the GBR. Ninety (54%) of the snorkelling incidents occurred in a 'commercial' (mainly supervised recreational diving) setting and 75 (46%) in a private setting. All but three of those in a commercial setting occurred on the GBR. Fifty-nine incidents in a private setting occurred on the GBR and the remaining 16 occurred further south.

All the scuba incidents occurred in the sea with 33 (83%) on the GBR and the remainder further south. Thirty-four (83%) of the scuba incidents occurred in a commercial setting and the balance occurred during private diving activities.

Origin of victims

Thirty-three (20%) snorkelling victims were Queenslanders, 19 (11%) were interstate visitors, and 111 (67%) were overseas tourists. The final three were from overseas, two of whom were working in Australia and the other studying.

Eight of the scuba victims were from Queensland, two from interstate and 31 were overseas tourists. The origins of all overseas victims are shown in Table 2.

Supervision and buddy / group situation

At least 58% of the snorkellers were under supervision and at least one third had set off unsupervised. However, the level of supervision varied greatly, from a one-to-one in-water guide to a single lookout for 100 guests in commercial settings, to one lifeguard for more than 200 swimmers in a public setting. Serious issues in supervision were evident in some incidents as discussed later.

Eight of the snorkellers collapsed on returning to the boat or pontoon so their buddy situation is excluded. Forty-six (28%) set out solo and without any supervision. Of the remaining 112 snorkellers who set out with a buddy or amongst a group, 45 were still together when the incident occurred, while seven separated during the incident. Twenty-one victims were amongst (sometimes loosely) supervised groups in a commercial setting but were essentially snorkelling solo as they were not allocated to buddy pairs.

Table 2
Origin of overseas tourists involved in snorkel and scuba diving fatalities in Queensland 2000–2019

Region	Snorkel *n* (%)	Scuba *n* (%)
Asia	37 (32)	6 (19)
North America	32 (28)	16 (52)
Europe	22 (19)	3 (10)
United Kingdom	17 (15)	5 (16)
New Zealand	4 (4)	0 (0)
Africa	1 (1)	0 (0)
Unknown	1 (1)	1 (3)

Five of the scuba divers set out solo, 18 became separated before the incident and another seven during their incident. Only 14 divers were still with a buddy or buddies.

Dive purpose

The vast majority (137, 83%) of snorkellers were sightseeing, 21 (13%) were spearfishing, four (2%) were practicing breath-holding, and the remainder were work-related. Similarly, the vast majority (29, 71%) of the scuba divers were sightseeing, seven were participating in resort dives, one was under training, two were working, and the activity of one was unknown.

Depth of incident

At least two-thirds of the snorkellers were likely surface snorkellers and at least one quarter were breath-hold diving to some extent. One hundred and twenty-one (73%) of snorkelling incidents occurred on the water surface, 13 underwater, and eight after exiting the water. The remainder were unknown. Twelve of the scuba incidents occurred at the surface and 13 at depths up to 10 metres of seawater (msw). Another 10 incidents occurred between 10 and 30 msw. Two divers collapsed on the boat post dive and, in four cases the incident depth was unknown.

Swimming skills (snorkellers), buoyancy aids and weights

There was no information about the swimming ability of 60 snorkelling victims. Of the remainder, 80 (48%) were reported to have been competent swimmers and 25 (15%) weak or non-swimmers. Only 14 of the weak/non-swimmers were wearing a floatation aid. Most of the snorkellers (122, 75%) were wearing fins. However, at least 21 (13%) were not and, of these, at least five were reported to have been weak swimmers. Seventeen of the snorkellers were reported to have been wearing weights, all but one of whom were breath-hold diving. Only three of these had ditched their weights before being found. There were several reports where a single person on a tender was unable to

lift a (generally overweight or obese) snorkeller aboard, so delaying the rescue and reducing the likelihood of successful resuscitation.

In two scuba cases no body was recovered, four victims collapsed after boarding the boat or platform, 22 were 'rescued' (16 on the surface and six underwater), and 12 divers were 'recovered' from underwater after a search and associated delay. Thirty-five of the scuba divers were still wearing their weights when found, 20 had uninflated buoyancy control devices (BCDs) and half were found both wearing weights and with uninflated BCDs.

Resuscitation

In water rescue breathing was performed to some extent on at least 16 of the snorkellers (in one case using a scuba demand valve to provide ventilations) and on five of the scuba divers. Airway management complications from regurgitation, water, froth, pulmonary oedema fluids, clenched teeth and poor positioning were reported in at least 70 (42%) of the snorkel and 22 (54%) of the scuba incidents but likely occurred in more as relevant details are usually not sought or included in the reports.

Basic life support (BLS) was performed in at least 138 (83%) of the snorkelling and 37 (90%) of the scuba incidents. In most of the others it was inappropriate due to the long delay in body recovery or absence of a body. In most of the commercial scenarios, resuscitation was commenced by trained staff, sometimes assisted by bystander medical professionals. Supplemental oxygen was reported to have been provided during initial resuscitation in 75 (46%) of the snorkelling and at least 26 (62%) of scuba incidents. However, it was not available when required in 22 snorkelling and seven scuba incidents (five of the latter being in a private setting). Supplemental oxygen was not applicable in 24 snorkel and three scuba incidents, and there was no information about oxygen administration in 46 snorkelling and six scuba cases.

An automated external defibrillator (AED) was available at or near the site and used onsite in at least 66 (40%) of the snorkelling and 10 of the scuba incidents. In two-thirds of the snorkelling incidents (and in all the scuba cases) the victim was under the direct supervision of a commercial operator. Most of the others were at sites such as island resorts or public beaches where the individuals were snorkelling independently. Shocks (from one to seven) were given in 19 of the snorkelling cases, no shock in 43, and it was unclear in the remaining four cases. No shock was given in six scuba cases, with one to four shocks delivered in the remainder. In most cases, there was no clear indication of the time from likely cardiac arrest to AED attachment. However, in only 18 cases it appears that attachment could have occurred within 10 minutes or less. Pre-shock delays of 10 to 20 minutes and sometimes far longer were the norm.

CHAIN OF EVENTS ANALYSIS

Predisposing factors

Two hundred and thirty-four likely or possible predisposing factors were identified in 160 of the 166 snorkelling incidents, and 59 were identified in 38 of the 42 scuba cases. The most frequent of these were health-related, which likely influenced the outcome in 102 (61%) of the snorkel and one half of the scuba victims. The most common were ischaemic heart disease (IHD), obesity and hypertension, in both groups. In snorkellers, a variety of other health factors such as a history of cardiac arrhythmias, diabetes, epilepsy, and the presence of alcohol were implicated. Autopsies often revealed undiagnosed IHD, cardiomegaly and left ventricular hypertrophy, all of which predispose to cardiac arrhythmias.

Lack of skills and experience were identified as contributing factors in at least 50 of the snorkelling and 12 scuba incidents although they may well have been a factor in others. In snorkellers, they were most often associated with a primary drowning. Nine of the scuba victims were uncertified – seven participating in organised resort dives and two on their first dive supervised by a friend. Three were certified with very few or no subsequent dives for at least one year, and another two had done few dives since training. One diver who had trained in a dam was doing their first ocean dive which was in a strong current. The final victim was very experienced but had not dived for more than two years. At least nine of these scuba deaths were associated with primary drowning or cerebral arterial gas embolism (CAGE).

Poor planning decisions were implicated as contributing to 55 of the snorkelling and seven of the scuba fatalities. Most of the snorkelling cases involved the decision to snorkel or breath-hold solo and usually unsupervised. Other factors included setting off in conditions that were obviously beyond the victims' skill levels. Five of the scuba deaths resulted from decisions to dive in adverse conditions; two of these involved resort dive participants who became separated from their instructors in poor visibility. Another two involved non-instructor-certified divers teaching friends at unsuitable sites. The other involved an instructor taking a certified, albeit inexperienced diver into a strong current without having a pre-agreed separation plan.

Activity-related predisposing factors were evident in 27 snorkelling/breath-hold incidents. Twenty-two involved extended breath-hold diving, five in a pool. Pre-dive hyperventilation was either witnessed or probable in at least seven cases. Seventeen of the victims had set out solo, four had separated before or during the incident, and only one was still with a buddy. Four of the other five deaths with activity-related predisposing factors involved spearfishing in areas with large sharks or crocodiles. The final incident involved a large stingray.

Unsafe supervision was identified as a factor in 18 snorkelling and seven scuba incidents, of which 12 snorkel and all the scuba occurred in a commercial setting. Seven of the snorkel cases involved a failure of the lookout(s) to notice that the victim was missing until a post-dive head count or notification by others. Others involved poor selection of suitable conditions for inexperienced and/or elderly snorkellers due to current or chop, and/or the area to be supervised being too large to be effectively monitored. Two involved poor supervision of an inexperienced snorkeller/weak swimmer by more experienced buddies. Others involved inexperience and distraction of lookouts. Four of the scuba cases involved poor in-water supervision of uncertified or very inexperienced divers. Another involved a dive operator's failure to provide a guide to oversee a novice on their first open water dive and in difficult conditions. One case was associated with poor surface supervision of a solo diver.

Organisational shortcomings were identified in at least 15 snorkel and five scuba incidents, a likely underestimate. Inadequate training of snorkeller lookouts, too few lookouts for the number of snorkellers or the size of the snorkel area, poor selection of snorkelling area due to prevailing or likely evolving conditions, and briefing inadequacies were identified. In one case, a staff member gave poor advice about the relevance of a medical condition apparently with adverse consequences. Three scuba incidents involved resort dives which were conducted in poor visibility, and which resulted in the victims separating from the instructors. In at least two of these, the instructors were swimming in front of the group and facing ahead. In one, it was noted that the divers had not been briefed on weight belt ditching or separation procedure. One incident resulted from a poorly organised commercial dive where there was inadequate functional equipment from the outset. The other involved poor maintenance of, and procedures for, the use of a dive club's compressor which led to serious air contamination. Other problems included faulty, or lack of readily available oxygen equipment or AEDs.

Equipment inadequacies were identified as contributing factors in 17 snorkelling and four scuba incidents, at least 13 of which resulted in primary drowning. With snorkellers, these mainly involved the lack of fins and/or personal floatation devices in weak or non-swimmers, an overly tight floatation device, an overly tight wetsuit, and obvious overweighting in at least one breath-hold diver. The scuba incidents included the occupational dive mentioned above, a faulty pressure gauge, and contaminated cylinder air in two cases.

Triggers

In all, 201 likely or possible triggers were identified from 148 of the snorkelling incidents and 56 triggers were identified from 37 of the scuba incidents. Various environmental factors triggered 93 snorkelling and 25 scuba fatalities. Sixty-eight of these environmental triggers in snorkellers and 14 of those in scuba divers appear to have arisen from the direct effects of immersion which redistributes circulation and can impact cardiac function and lead to cardiac arrhythmias in susceptible persons. Adverse surface conditions, current and poor visibility were implicated in at least 24 snorkel and 11 scuba incidents, in some cases compounding the cardiac effects of immersion by increasing exertion and anxiety. Seven other environmental triggers involved snorkellers' encounters with dangerous marine creatures (two sharks, two crocodiles, two Irukandji, one stingray). Anxiety (reported by witnesses) was identified as a probable trigger in at least six snorkel and 10 scuba cases but very likely contributed to others. Water aspiration through the snorkels of novices was identified as the probable trigger in 45 snorkelling incidents but was likely to have occurred in more. Extended apnoea, with or without hyperventilation, was the trigger in 21 fatal breath-hold incidents.

There were four gas supply-related triggers in scuba divers which involved two divers who ran out of air, one who became nauseated from oil contamination and one diver whose air was severely contaminated with both carbon monoxide and carbon dioxide. Other scuba incident triggers included trauma and inadequate decompression.

Disabling agents

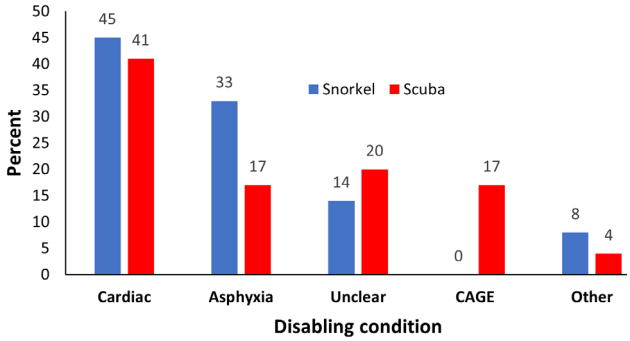
Disabling agents (i.e., actions or circumstances associated with the triggers that caused injury or illness) were identified in 148 of the snorkelling fatalities, the majority (95, 64%) being medical-related, predominantly IHD. Pre-existing cardiac arrhythmias were implicated in five deaths, epilepsy in two. Immersion pulmonary oedema was identified as the likely disabling agent in two snorkelling incidents but may well have been present in more. Apnoeic hypoxia was the likely disabling agent in 21 of the 22 deaths involving extended hypoxia (the other possibly associated with IHD). Other likely disabling agents in the snorkel incidents were laryngospasm from water aspiration through snorkels (17), environmental (10), and buoyancy-related (3).

Thirty-six disabling agents were identified in the scuba incidents with insufficient information to determine likely agents in six cases. Half of the disabling agents were medical-related, all but one with cardiac disease or abnormality. The other likely disabling agents identified were ascent-related (7), buoyancy-related (5) and gas supply-related (3).

Disabling conditions (Figure 3)

The disabling condition directly responsible for death or incapacitation followed by death from drowning, was identified in 142 of the snorkelling fatalities but was unclear in the remaining 24, including seven where no body was found. The disabling condition in the other 'unclear' cases

Figure 3
Disabling conditions for snorkel and scuba victims of diving fatalities in Queensland 2000–2019



was either asphyxia or cardiac as there were indications of both. The most prevalent disabling conditions were cardiac-related (74), asphyxia (primary drowning) (55) and trauma (five). Immersion pulmonary oedema was the likely disabling condition in two cases but was identified as a possibility in five. Of the 65 snorkellers with a cardiac disabling condition for which the BMI was known, 26 (40%) were obese. By comparison, only 2 (4%) of the 46 asphyxia victims with known BMIs were obese.

The likely disabling condition was identified in 33 of the scuba divers but was unclear in the remaining eight. In three, it was difficult to determine whether the disabling condition was cardiac or CAGE as there were indications of both. In one of these, immersion pulmonary oedema was another possible differential diagnosis. There were 17 cases where the disabling condition appeared to have been cardiac-related. Of these, eight of the divers were known to have been under medical care for some related condition, although the extent of the predominantly heart disease was likely unknown. Five of these had declared their condition to the dive operator, two having produced medical clearances, albeit from doctors without dive medical training. None were under special observation during the dive. Twelve scuba divers had not declared any medical conditions, and in another two cases, it was unclear. Five victims were not under any medical care and there was no indicative information in four cases. One scuba death resulted from carbon monoxide poisoning and one diver died from fulminant decompression sickness.

ACTIVITY AND FATALITY RATES

Based on its annual visitor surveys, Tourism Research Australia data estimated that the average annual number of international visitors who snorkelled in Queensland between 2005–2019 inclusive was 429,849 (95% CI: 410,076–449,622). (Smith D, personal communication, 2022) Over that period, there were 81 deaths in snorkellers from overseas so the average annual fatality rate for overseas visitors in Queensland was 1.25 deaths per 100,000 snorkellers (95%CI: 1.00–1.56).

Similarly, the estimated average annual number of international visitors who went scuba diving in Queensland during that period was 192,403 (95% CI: 178,742–206,064) and there were 19 deaths among this group. This yields an annual fatality rate of 0.66 deaths per 100,000 international scuba divers (95% CI: 0.39–1.02). Note: This rate is higher than estimated in an earlier report[3] as the denominator provided by Tourism Research Australia had been subsequently reduced to provide a more accurate estimate.

Discussion

The ages of both the snorkel and scuba victims during the study period were substantially higher than over the previous three decades, likely reflecting the increasing age of diving participants generally.[14,15] The more than threefold increase in annual snorkel deaths between the periods seems largely reflective of the increased number of participants and their health status and probable lesser aquatic experience and skills. Many of the victims, especially the snorkellers, were older overseas tourists with pre-existing medical conditions which contributed to their demise. More than 80% of the incidents occurred on the GBR, with one half of the snorkelling and the vast majority of the scuba deaths occurring in a commercial setting. One third of the snorkellers had set off solo and many others were snorkelling without a designated buddy in a large group. In addition, many snorkellers and scuba divers who set off with a buddy became separated before their incident. Although the majority of snorkel victims were under supervision, the efficacy of this varied greatly as a result of pre-activity screening, sea conditions, and supervisors' ratios and experience. Many victims were inexperienced, and some died on their first snorkel or scuba experience. Fatalities in experienced breath-hold divers were mainly attributable to apnoeic hypoxia.

Resuscitation was attempted in most cases but was often belated due to delays in the recognition of the incident and subsequent rescue or recovery of the victim.

DEMOGRAPHICS

The substantially higher age of the snorkellers in Queensland is likely a reflection of older tourists particularly from overseas visiting the GBR and snorkelling. Worldwide there has been an increasing incidence of scuba fatalities in older divers.[9,16–18] The high prevalence of health-related conditions identified in both cohorts of victims is consistent with the increased likelihood of adverse health conditions in an older demographic.[19] Some conditions increase the risk of an incident in the water, whether snorkelling or scuba diving.

The high proportion of obesity in victims, especially the scuba divers, is cause for concern given its association with significant health conditions including sudden cardiac death.[20,21] Obesity has been implicated as a potential risk factor for a scuba diving fatality.[9,22]

PRE-EXISTING HEALTH CONDITIONS

The finding that pre-existing health conditions likely contributed to such a high proportion of both scuba and snorkelling fatalities is sobering, highlighting the need for participants to be sufficiently fit and healthy to participate in relative safety. Existing or potential divers and snorkellers with chronic medical conditions may require assessment at regular intervals. With the mix of circulatory changes associated with immersion, exertion, anxiety, exhilaration, saltwater aspiration and breathing resistance, snorkelling and scuba diving involve an array of potential triggers to a cardiac event in a susceptible person.[23] Some of the victims were under treatment for relevant medical conditions although relatively few had declared a pre-existing condition. Some individuals may have intentionally withheld information for fear of not being allowed to participate, while others might not have realised the reality of the potential risks and the importance of notifying the operators to enable risk mitigation strategies to be implemented. Many victims had undiagnosed heart disease and appeared to be reasonably healthy. Obesity could be used as a precautionary signal to trigger closer observation, especially in scuba divers.

EXPERIENCE

Many of the snorkel victims were inexperienced and some had very poor aquatic skills. A leaking mask or water aspiration through the snorkel can readily trigger panic and, in some cases, laryngospasm and subsequent unconsciousness and drowning. It is important that snorkel operators carefully screen prospective snorkellers and provide training, buoyancy support and close supervision where indicated. The use of well-fitting fins should be actively encouraged. The COP requires that "*all at risk snorkellers should be directed to wear and/or use a flotation or other device which is able to support the wearer in a relaxed state*." However, despite these measures being implemented by compliant operators, some deaths remained difficult to prevent due to pre-existing health issues and logistical challenges.

Whereas experience improves diving-related skills and environmental understanding, it can also breed complacency. Many of the more experienced snorkel victims were diving solo or with an intentionally loose 'buddy system'. A large proportion of these succumbed to apnoeic hypoxia after extended breath-holding with, or without, hyperventilation. The likelihood of blackout varies between dives and pushing one's breath-hold limits without a capable and ready rescuer is precarious. Despite this information being available for a long time, many breath-hold divers remain falsely confident that it won't happen to them.

The deaths of seven scuba divers during resort dives is concerning. However, three of these were associated with undeclared and possibly undiagnosed cardiac disease so might not have been easily avoidable. Four of the incidents (including one of the cardiac deaths) involved poor planning and/or supervision which led to separation of the victim and the instructor, likely resulting in panic and subsequent drowning in three divers. It is essential that, in such activities, the instructor very carefully assesses the existing and potential conditions and adjusts ratios or abandons the activity accordingly, as well as positioning themselves to maximise oversight of all participants.[2,24] The victim of the final resort dive incident panicked when their mask flooded, and, despite the efforts of the instructor, made a rapid breath-hold ascent which resulted in pulmonary barotrauma.

Many highly experienced scuba divers have been diving for long periods and often belong to the older cohort of divers who are more likely to have pre-existing disease, often cardiac-related.[14] It is recommended that all divers aged 45 years or over undergo a medical assessment with a focus on cardiovascular evaluation, preferably by a doctor trained in diving medicine to monitor their on-going fitness to dive.[25]

BUDDY SITUATION AND SUPERVISION

As in other reports, many of the deceased snorkellers and some of the scuba divers had set out solo or separated prior to their incident.[9,10,22,26,27] Others snorkelled alone within a large group. In such scenarios, if serious problems arise, they often go unnoticed for an extended period making survival unlikely. Even if unable to perform a rescue, a vigilant buddy can often alert others and set a rescue in motion.

Operators should ensure that pre-snorkel briefings include strong advice to set out and remain with a buddy and the benefits of doing so. Participants should be assisted with buddy selection, if required. Despite this, it is inevitable that some individuals will choose to set off alone or separate, intentionally, or otherwise.

Problems with supervision included inadequate identification or monitoring of weak swimmers or inexperienced snorkellers, poor site selection due to conditions and/or size and ineffective lookouts. In any setting, particularly commercial, it is important to assess a person's skills and experience and to have a system to readily identify at risk participants so that they can be more closely monitored. Many operators in Queensland have introduced risk mitigation strategies including encouraging the use of personal floatation devices or other floatation aids and colour coding on snorkels to indicate an increased risk and in-water supervision. This is to be applauded and should be encouraged elsewhere.

The COP requires that in commercial settings, dive site risk assessments are conducted considering the conditions as well as all aspects of the conduct of the dive operation, including entries and exits, risk of separation, searches for divers, rescues, and evacuations.

With large groups of snorkellers to observe, it can be difficult for a lookout to recognise a problem, especially if they are relatively inexperienced, tired, or distracted. There is a need to ensure that there are sufficient lookouts to effectively supervise an area, considering the size, shape and geographical features of the site, the prevailing conditions, the number of snorkellers and the effectiveness of the vantage point. These lookouts should be adequately trained in observation and monitoring techniques, always remain vigilant and be relieved at regular intervals to avoid fatigue or complacency.

Prompt identification of a distressed or unconscious snorkeller or diver, together with rapid rescue, will maximise the chances of survival. However, substantial delays in recognition do occur as it can sometimes be difficult to determine whether a motionless snorkeller is unconscious or just quietly observing the scenery below. Many of these deaths are silent and signs of distress absent or overlooked. It is better to have a high index of suspicion and run the risk of over-reacting. In addition, suitable rescue techniques need to be identified and practiced ensuring that they can be done swiftly and effectively when needed.

BRIEFING

In some cases in commercial settings it was reported that the victims did not attend or did not pay attention to the briefing and so might have missed important information that could have prevented their incident. A thorough pre-snorkel or dive briefing is an important safety and risk mitigation tool which is especially necessary for the inexperienced, but also may provide valuable insights and local knowledge for experienced divers and snorkellers. Such a brief should be located and timed to minimise distractions. It should inform participants of the potential risks associated with certain health conditions and highlight the importance of honestly declaring these to the operator to enable them to implement safety processes. Similarly, participants should be encouraged to declare their swimming ability and snorkelling experience. The brief should also highlight the importance of the buddy system, of staying in the designated area, the likely site conditions (e.g., currents) and marine life, what to do if they need assistance and the timing and recall procedure, among other things.

Confusion arising from language issues can create a problem with briefings. Some operators have staff who provide briefs or translation in key languages. To assist with this, WorkSafe Queensland has published an informative dive and snorkelling guide, currently available in 14 languages, which should be made readily available to non-English speaking participants.[28]

RESCUE AND FIRST AID

It is very important for a diver who is likely to become unconscious underwater to initiate self-rescue and try to attain positive buoyancy to reach the surface where they will generally be more easily located. As in other series, many of the scuba victims were still wearing their weights and had uninflated BCDs.[9,22,27] The COP now requires that resort dive participants are taught how to inflate and deflate their buoyancy devices on the surface.

It is also important for breath-hold divers to adjust their buoyancy to be positively buoyant in the last few metres to the surface. In that way they will be more likely to rise to the surface if unconscious.

The problem of a single person trying to drag an unconscious or semi-conscious person onto a small vessel is not uncommon in both commercial and private settings. Particularly where assistants are not readily available, a carefully prepared plan, appropriate equipment and practice may reduce the difficulty and associated delay.

In the commercial setting, once resuscitation was commenced it generally appeared to have been done with reasonable efficiency by appropriately trained staff. Supplemental oxygen was reported to have been provided in almost half of the cases, substantially higher that the 16% documented nationwide (under-reporting may well have occurred).[29] This is very likely a result of the requirements under the COP, coupled with better reporting, although under-reporting is still likely.

The increasing availably of AEDs, as required under the COP, is a positive development to be encouraged in other jurisdictions, especially considering an ageing diver population and the increasing prevalence of cardiac-related incidents. Unfortunately, their success to date in the diving setting has been rather limited, partly because of the significant delays from cardiac arrest to AED attachment in these environments. Improved supervision and efficient rescue can reduce this interval, increase the likelihood of the victim having a shockable rhythm and enhance the chances of survival. Appropriate supervision and rescue training and practice are essential to reduce delays.

FATALITY RATE

There appears to be a dearth of reasonably reliable and accessible information on international snorkelling fatality rates, so it is difficult to compare the snorkelling annual fatality rate from this study. However, the estimated rate for overseas scuba divers in Queensland is considerably lower than other published rates[3,30] suggesting that scuba diving in Queensland may be comparatively safe. This could be due in part to often more favourable conditions and easier diving when compared to more temperate environments. However, it is interesting to note that there was no significant increase in the average annual number of scuba deaths in Queensland between the periods 1970–1999 and 2000–2019 despite what was likely an increase in diving activity and an increase in the average age of divers with its associated risks. The more

stringent oversight and better management of diving because of the COP may have contributed to this.

CODE OF PRACTICE

The latest COP has incorporated possible mitigation strategies to most of the issues identified in this investigation, and, if conscientiously implemented together with the additional recommendations herein, are likely to prevent some future incidents. However, as mentioned earlier, some diving-related morbidity and mortality is inevitable despite all efforts.

DATA COLLECTION

Data collection and reporting for diving-related fatalities varies between various places, often depending on the familiarity and interest of the initial (usually local police) investigators with scuba diving or snorkelling and any follow-up systems in place. However, unless key questions are included in the incident proforma used, valuable information which could be used to improve safety can easily be missed. An example of a template for data collection for a scuba fatality can be found at: https://adsf.cdn.prismic.io/adsf/b198f7ef-9afa-4f0b-91b9-f70ef481595f_Data-Collection-1+%281%29.pdf.

LIMITATIONS

As with any uncontrolled case series, the collection and analysis of fatality data are subject to inevitable limitations associated with the incident investigations. Given that many incidents were unwitnessed, assertions in the reports are sometimes speculative. Important information may not be available, which rendered chain of events data incomplete and limiting conclusions that can be drawn.

The results of the international visitor survey are based on samples, rather than a census and therefore subject to sampling error. However, with relative standard errors for the number of participants at around 1.5% (snorkellers) and 3.3% (scuba) sampling error was not a major barrier to their use.

Comparisons between annual fatality rate estimates from different data sources can be unreliable due to a variety of factors including accuracy of denominator (and sometimes numerator) data.

Conclusions

While scuba diving deaths remained stable, there was a substantial increase in the number of snorkelling deaths in Queensland over the past two decades. This is likely a reflection of the increased number of participants, their higher ages and poorer health. However, considering the number of overseas participants the estimated fatality rates appear to be relatively low, which may in part be due to the existence and enforcement of a COP and better oversight and management.

Issues identified included pre-existing medical conditions, poor skills, inexperience, poor planning, supervision shortcomings and lack of effective buddy systems in both cohorts, and apnoeic hypoxia in breath-hold divers. The main disabling condition in both snorkellers and scuba divers was cardiac-related, and a high proportion of victims, especially scuba divers, were obese.

Potential countermeasures include increased education of the importance of health and fitness for safe diving and snorkelling, fitness-to-dive assessments for older divers and those with chronic health conditions, improved pre-travel health screening for tourists planning to snorkel, increased emphasis on the importance of an accurate pre-activity health declaration and subsequent implementation of appropriate risk mitigation strategies, improved supervision with higher supervision-to-participant ratios when appropriate, better buddy pairing, and continued and strengthened education on the hazards of extended apnoea for breath-hold divers.

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Blood pressure in rats selectively bred for their resistance to decompression sickness

Emmanuel Dugrenot[1,2], Jérémy Orsat[1], François Guerrero[1]

[1] *Univ Brest, ORPHY EA4324, IBSAM, 6 avenue Le Gorgeu, 29200 Brest, France*
[2] *TEK diving SAS, F-29200, Brest, France*

Corresponding author: *Dr François Guerrero, EA4324 ORPHY, 6 Av. Le Gorgeu CS 93837, 29238 BREST Cedex 3, France*
francois.guerrero@univ-brest.fr

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Abstract
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Introduction: Susceptibility to decompression sickness (DCS) is characterised by a wide inter-individual variability whose origins are still poorly understood. This hampers reliable prediction of DCS by decompression algorithms. We previously selectively bred rats with a 3-fold greater resistance to DCS than standard rats. Based on its previously reported relation with decompression outcomes, we assessed whether modification in vascular function is associated with resistance to DCS.
Methods: The arterial pressure response to intravenous administration of acetylcholine (ACh, 5 $\mu g.kg^{-1}$) and adrenaline (5 and 10 $\mu g.kg^{-1}$) was compared in anaesthetised DCS-resistant rats (seven females, seven males) and standard Wistar rats (seven females, 10 males) aged 14–15 weeks. None of these rats had previously undergone hyperbaric exposure.
Results: There was a non-significant tendency for a lower diastolic (DBP) and mean blood pressure (MBP) in DCS-resistant rats. After ACh administration, MBP was significantly lower in resistant rats, for both males ($P = 0.007$) and females ($P = 0.034$). After administration of adrenaline 10 $\mu g.kg^{-1}$, DCS-resistant rats exhibited lower maximal DBP ($P = 0.016$) and MBP ($P = 0.038$). Systolic and pulse blood pressure changes did not differ between groups in any of the experiments.
Conclusions: Resistance to DCS in rats is associated to a trend towards a lower vascular tone but not blood pressure reactivity. Whether these differences are a component of the susceptibility to DCS remains to be confirmed.

Introduction

Susceptibility to decompression sickness (DCS) is characterised by a wide interindividual variability in humans. This is documented both by empirical data which have shown that multiple divers can execute the exact same dive profile but not all of them will experience symptoms[1] and by experiments employing animal models of DCS which provide many examples of this huge inter-individual variability for the occurrence of DCS.[2,3]

This could be partly explained by interindividual variability in post-dive venous gas emboli (VGE) formation.[4,5] However, although the occurrence of DCS correlates with VGE detected post-dive,[1] this correlation is weak. These observations clearly show that for the same hyperbaric exposition, the probability of DCS depends on many factors which drive the formation of VGE and/or modulate their power to trigger DCS. Indeed, for a given dive profile the risk of DCS is influenced by many individual factors including body composition,[6] the presence of right-to-left shunts (such as patent foramen ovale)[7] or, in animal models of DCS, hydration.[8] A more complete overview of DCS risk factors is provided elsewhere.[9] Physiological variables including inflammation,[10,11] coagulation,[12,13] oxidative stress,[14] and vascular dysfunction,[14,15] have also been claimed to modulate susceptibility for DCS. However, little consensus has been reached and the primary physiological variables that drive resistance to DCS remain to be specified. A consequence is that not all DCS can be predicted by decompression algorithms based on theoretical models of saturation and bubble formation in divers.[6]

There is a body of data which suggest that the vascular system also might influence both the amount of VGE formed after a dive and the probability of DCS. Indeed, one *in vitro* study showed that bubbles can form at active hydrophobic spots located at the surface of the endothelium.[16] Administration of nitric oxide (NO) donors decreases both the number of VGE detected after a dive[17] and the risk of DCS in animal models,[18,19] whereas inhibition of NO synthase increases it.[20,21] Chronic administration of angiotensin converting enzyme inhibitor before the dive reduces the occurrence of DCS in rats,[22] consistent with a post-dive decrease of angiotensin II in animals with no symptoms of DCS but not those with DCS.[14] Lastly, one study reported significant differences in basal total arterial compliance and stable metabolites of NO in the plasma between divers with low and high bubble grades.[23] Taken together, these data suggest that the viscoelastic properties of the vascular system might influence the susceptibility to DCS.

Our group initiated a large-scale artificial selection program with Wistar rats based on their resistance to DCS, and reported a threefold decrease in DCS occurrence.[24] This selection program now provides a population with significantly increased spontaneous resistance to DCS. First investigations showed that, when compared to standard Wistar rats, these animals exhibit increased leukocyte counts, lower coagulability and lower mitochondrial basal oxygen consumption,[25] as well as modifications of the gut microbiome.[26] At the vascular level, we observed decreased *in vitro* vasorelaxation of the aorta in response to NO donor administration, and no differences in vasoconstriction elicited by phenylephrine or KCl.[25]

Based on the previously reported association of vascular function with decompression outcomes and the apparent contradiction with the lower vasorelaxation capacity observed in our selected animals, we assessed whether increased resistance to DCS is associated with *in vivo* modification in vascular function. To this end we compared arterial pressure response to acetylcholine (ACh) and adrenaline administration in DCS-resistant and standard Wistar rats.

Methods

ETHICAL APPROVAL

The protocol described in this study was conducted in accordance with the Directive 2010/63/EU of the European Parliament and of the council on the Protection of animals used for scientific purposes, and with the French national laws R214-87 to R214-137 of the Rural Code and subsequent modifications. It followed the 3Rs and was approved by the Ethics Committee of the Université de Bretagne Occidentale for Animal Experimentation (approval no. APAFIS#10838-2017072817299340v1 and APAFIS#15628-2018061516233394v3).

ANIMALS

Fourteen DCS-resistant animals (seven females and seven males), aged 14–15 weeks old, bred at the university animal house, were used in this study. They were compared to 17 age-matched standard Wistar rats (seven females and 10 males), i.e., the same as those we used for the founding stock, obtained from the same breeder (Janvier Labs, St Genêts, France). Because the aim was to assess any difference in cardiovascular function associated with resistance to DCS independently of persistent physiological modifications induced by diving itself,[27,28] none of these rats were previously exposed to hyperbaric conditions. The standard rats were acclimated with the facility for at least two weeks. All animals were housed three per cage under controlled temperature (21 ± 1°C) and lighting (12 h of light per day, 0600–1800) at the university animal housing facility until the day of the experiment. They were fed standard rat chow and water *ad libitum*.

ARTERIAL PRESSURE

Following anaesthesia, a temperature probe was inserted rectally and animals were placed in supine position on a warming pad (Z31SY, Ascon tecnologic, Italy) to maintain central body temperature in a normal range (37.5 ± 0.5°C). A 2 cm cervical incision was performed, followed by a tracheostomy (2 mm diameter polyethylene tube). An arterial catheter (Leader Flex 22 G, 0.7 × 40 mm, Vygon, France) was inserted in the right carotid allowing continuous intra-arterial pressure monitoring. A venous catheter was inserted in the left jugular vein (Leader Flex 22 G, 0.7 × 40 mm, Vygon, France) for infusion of drugs. Vital signs (heart rate, invasive arterial pressure and body temperature) were continuously recorded during the procedure (MP35, BIOPAC Systems, Inc. Varna, Bulgaria).

Acetylcholine (5 $\mu g \cdot kg^{-1}$, Sigma, A6625-25G) was first administered. After blood pressure returned to basal values, adrenaline (5 $\mu g \cdot kg^{-1}$, Sigma, E4250-1G) was administered, followed by a 10 $\mu g \cdot kg^{-1}$ dose when a stable blood pressure was reached again. All traces were displayed on a personal computer using Biopac Student Lab Pro 3.7.1 (BIOPAC Systems, Inc. Goleta CA, USA) and stored for later analysis. Diastolic (DBP) and systolic (SBP) blood pressure were measured before administration of each drug and after injection. For each point, mean blood pressure (MBP) was calculated according to the formula MBP = DBP + 1/3 (SBP – DBP). Maximal changes in systolic, diastolic, pulse and mean pressure were determined for each drug and dose.

STATISTICAL ANALYSIS

All data were analysed using Statistica™ software (v. 13, StatSoft France, 2017). Because results were not all parametrically distributed, as assessed with a Shapiro-Wilk test, we used a Kruskal-Wallis ANOVA by ranks test on four independent groups: standard females (StF), standard males (StM), resistant females (ReF) and resistant males (ReM). When a significant difference was detected between groups a Mann-Whitney U post-hoc analysis was run. Differences were considered significant at $P < 0.05$. Data were reported as median (interquartile range [IQR]).

Results

ACETYLCHOLINE

Blood pressure values before and after administration of ACh are presented in Table 1.

Before administration of ACh, no statistically significant difference between groups was detected for SBP and pulse pressure (PP). There was a tendency for a lower DBP and MBP in DCS-resistant than in standard rats, although the differences between groups did not reach statistically significant threshold either.

Table 1

Changes in median (IQR) blood pressure (mmHg) elicited by acetylcholine; DBP – diastolic blood pressure; Max – maximum; MBP – mean blood pressure; PP – pulse pressure; ReF – female DCS-resistant Wistar rats; ReM – male DCS-resistant Wistar rats; SBP – systolic blood pressure; StF – female standard Wistar rats; StM – male standard Wistar rats

Pressure measure	StF		StM		ReF		ReM		*P*-value	
	Basal	ACh	Basal	ACh	Basal	ACh	Basal	ACh	Basal	ACh
DBP	83.7 (65.1–91.6)	38.7 (37.1–41.1)	83.2 (71.9–104.9)	39.2 (37.0–40.5)	59.9 (59.3–90.4)	31.8 (28.3–37.0)	69.5 (63.2–78.1)	34.8 (31.1–41.5)	0.055	0.050
SBP	108.7 (93.4–114.9)	71.1 (70.3–71.5)	108.7 (97.1–133.6)	72.2 (66.5–80.0)	88.6 (85.9–116.6)	65.1 (59.5–70.4)	99.9 (88.2–107.9)	69.5 (57.4–73.9)	0.186	0.128
MBP	92.0 (74.5–99.3)	49.2 (48.3–49.5)	91.7 (80.3–118.6)	48.5 (47.5–53.9)	69.5 (65.0–99.2)	42.8 (39.5–48.1)	77.5 (74.3–88.0)	45.4 (43.4–46.4)	0.066	0.006
PP	25.0 (23.3–28.3)	33.5 (32.4–34.5)	25.2 (24.0–30.5)	30.0 (27.8–38.1)	26.6 (24.6–28.7)	33.1 (32.5–33.5)	30.0 (14.9–33.3)	34.7 (19.9–42.8)	0.875	0.877

Intravenous administration of ACh 5μg.kg^{-1} elicited hypotension in all groups. The Kruskal-Wallis analysis indicated that minimal MBP values after the administration of ACh were significantly different between groups. Post-hoc comparisons indicated that the post-ACh MBP values were significantly lower in resistant than standard animals, for both males (P = 0.007) and females (P = 0.034). The other blood pressure parameters after administration of ACh were not different between groups, although there was a non-statistically significant trend for lower DPB in DCS-resistant individuals than in standard rats. Nevertheless, for all blood pressures, the differences between the basal values and those measured after ACh administration were not different.

ADRENALINE

Kruskal-Wallis analysis indicated significant differences between groups for values of DBP, SBP and MBP obtained both before and after administration of adrenaline 5 μg.kg^{-1}, but not for PP (Table 2).

No differences were detected between groups for arterial pressures before administration of adrenaline 10 μg.kg^{-1}, whereas there were statistically significant differences between groups after injection of the drug for DBP and MBP but not SBP and PP (Table 3). Post-hoc testing indicated that maximum DBP was significantly lower in females rats resistant to DCS than in standard rats (P = 0.030). However, as was the case for ACh administration, the differences in blood pressures between the basal values and those measured after adrenaline administrations were not different between groups.

Discussion

We found lower MBP values after administration of ACh in DCS-resistant than standard rats of both sexes. In contrast, after administration of 10 μg.kg^{-1} adrenaline the hypertensive response was weaker in DCS-resistant than standard rats, as indicated by lower maximum values of DBP and MBP, which was more evident in females. However, the amplitude of the responses to both ACh and adrenaline were not different between resistant and standard animals. This was probably because of a trend (although non-significant) to lower basal pressures in resistant animals.

Susceptibility to DCS is characterised by substantial interindividual variability, which is particularly well documented in animal models.[2,3] Such variability also exists in divers[29] and is one of the causes of so-called 'undeserved' DCS since current decompression algorithms cannot take it into account. Indeed, one study reported that 97.5% of the DCS cases recorded in the DAN DSL database occurred without violation of the algorithm recommendations.[6] This 'probabilistic' character of the susceptibility to DCS also hampers studies of its determinants. To overcome this limitation we selectively bred Wistar rats based on their resistance to DCS. Indeed, the ratio of asymptomatic animals

Table 2

Changes in median (IQR) blood pressure (mmHg) elicited by adrenaline 5 μg.kg^{-1}; DBP – diastolic blood pressure; Max – maximum; MBP – mean blood pressure; PP – pulse pressure; ReF – female DCS-resistant Wistar rats; ReM – male DCS-resistant Wistar rats; SBP – systolic blood pressure; StF – female standard Wistar rats; StM – male standard Wistar rats

Pressure measure	StF		StM		ReF		ReM		*P*-value	
	Basal	Max	Basal	Max	Basal	Max	Basal	Max	Basal	Max
DBP	79.4 (70.7–87.5)	136.7 (135.0–165.0)	89.9 (77.9–95.7)	148.9 (144.7–152.7)	65.3 (48.6–70.1)	121.2 (98.0–123.2)	80.3 (58.8–104)	129.9 (117.6–142.6)	0.026	0.010
SBP	101.3 (94.7–111.1)	199.4 (197.4–201.6)	119.2 (111.5–131.2)	196.1 (185.8–208.8)	91.1 (79.0–94.7)	168.0 (130.7–170.6)	102.1 (96.1–108.4)	171.3 (162.8–185.8)	0.088	0.002
MBP	86.0 (80.0–95.4)	159.1 (156.2–177.2)	99.7 (88.1–107.6)	167.1 (161.9–171.0)	74.6 (58.8–77.0)	137.6 (108.9–143.1)	85.6 (71.3–94.8)	145.6 (135.6–155.3)	0.018	0.001
PP	23.5 (20.5–27.8)	57.5 (36.6–61.7)	29.3 (24.3–34.3)	51.4 (34.2–64.4)	26.7 (21.7–28.0)	33.0 (29.6–49.4)	25.8 (15.8–31.3)	38.6 (29.0–54.2)	0.651	0.516

Table 3

Changes in median (IQR) blood pressure (mmHg) elicited by adrenalin 10 μg.kg^{-1}; DBP – diastolic blood pressure; Max – maximum; MBP – mean blood pressure; PP – pulse pressure; ReF – female DCS-resistant Wistar rats; ReM – male DCS-resistant Wistar rats; SBP – systolic blood pressure; StF – female standard Wistar rats; StM – male standard Wistar rats

Pressure measure	StF		StM		ReF		ReM		*P*-value	
	Basal	Max	Basal	Max	Basal	Max	Basal	Max	Basal	Max
DBP	108.7 (83.7–111.2)	151.4 (145.5–159.5)	75.4 (73.9–98.7)	162.1 (159.0–166.0)	96.3 (63.7–114.7)	136.9 (129.4–141.8)	85.8 (57.7–107.9)	148.7 (133.0–166.4)	0.242	0.016
SBP	137.6 (109.4–150.9)	210.4 (204.6–223.7)	103.5 (99.0–129.7)	211.7 (193.4–222.3)	128.5 (107.5–148.4)	205.2 (200.8–215.3)	110.4 (96.4–126.3)	192.2 (177.8–204.4)	0.243	0.306
MBP	118.3 (88.3–124.4)	173.8 (169.1–178.2)	83.4 (82.4–109.1)	176.0 (175.1–185.8)	107.1 (78.3–128.9)	160.7 (157.5–161.5)	94.0 (70.6–114.0)	166.2 (157.0–175.0)	0.253	0.038
PP	33.7 (25.7–35.3)	57.4 (27.6–70.7)	28.6 (24.6–31.4)	41.7 (36.4–62.7)	32.9 (29.9–42.5)	59.9 (59.0–76.2)	29.9 (18.4–38.7)	37.0 (25.8–57.1)	0.545	0.509

rose from 35% in the non-selected Wistar rats to 80% and 72% in selected females and males, respectively.[25] Now that we have a population that is significantly different from normal in its resistance to DCS, our objective is to investigate the physiological characteristics of these individuals that may drive this resistance.

It is now well accepted that the risk of DCS depends not only on the amount of VGE formed during and after decompression but also the ability to cope with them, both being influenced by individual factors. Vascular function is one of a number of physiological risk factors proposed.[10–14] For instance, one study found that divers with lower bubble grades after a dive also had lower SBP and PP before the dive.[23] In keeping with these previous data, although the difference between groups did not reach statistical significance in the present experiment, we also observed that before any intervention (i.e., before administration of ACh) the DCS-resistant rats tended to have lower diastolic and mean blood pressure than the standard rats.

We found both greater hypotension in response to ACh and weaker adrenaline-induced hypertension in the rats resistant to DCS. Moreover, we observed these differences for DBP and MBP only, and not for SBP or PP. Since the changes between basal and post-infusion blood pressures were not different, it seems plausible that resistance to DCS could be associated with a general trend towards lower total peripheral vascular resistance but not vascular reactivity. One study reported that mean arterial blood pressure was increased in anaesthetised rats during a simulated air dive at 600 kPa, which was due to an increase in total peripheral vascular resistance which developed within five minutes.[30] This hypertensive response to hyperbaric exposure is confluent with an earlier study which reported decreased blood flow in skeletal muscles of Wistar rats exposed to 500 kPa He-N_2-O_2.[31] It is therefore plausible that the shift in the blood pressure observed in our DCS-resistant animals would at least partially counteract the hypertensive effect of diving by limiting the maximal total peripheral vascular resistance at depth. This is still to be confirmed and, even if so, whether this represents an advantage for the resistance to DCS remain to be determined. However, we showed previously that chronic treatment with nifedipine, which lowers arterial pressure, before the dive did not influence the risk of DCS in rats.[22] This suggests that factors that affect blood pressure, rather than the blood pressure itself, may influence resistance to DCS.

This hypothesis agrees with the pre-dive higher plasma concentration of NO metabolites previously reported in divers who produce lower grade bubbles after the dive.[23] It is also confluent with previous studies showing that the administration of NO donors decreases both the amount of VGE detected in humans after a dive[17] and the risk of DCS in animal models,[18,19] whereas inhibition of the NO synthase increases it.[20,21] Similarly, chronic administration of angiotensin converting enzyme inhibitor, but not angiotensin receptor antagonists, before the dive reduces occurrence of DCS in rats.[22] This result is confluent with the post-dive decrease of angiotensin II in animals with no symptoms of DCS but not those with DCS[14] and with the decreased plasma concentrations of adrenaline and noradrenaline in humans after a dive.[32] Unfortunately, we did not measure circulating concentrations of NO, angiotensin II or adrenaline in this study. However, we previously reported decreased coagulation tendency, a function influenced by both NO and angiotensin II, in male rats selected for their resistance to DCS.[25] This remains to be confirmed.

LIMITATIONS

In this study, we used standard Wistar rats obtained from an approved provider as control rats. Even if the DCS-resistant animals were derived from animals of the same Wistar strain obtained from the same provider, the standard and resistant animals used for this study were not bred in the same conditions since their birth. This might have influenced physiological parameters independently from the resistance to DCS. However, standard rats were kept for two weeks before the experiments which probably limited this potential bias. Additionally, our previous experiments showed that it is unlikely that our breeding conditions alone induced such a resistance.[25] Another limitation arises from our approach which compared animals of differing resistance to DCS but which were not exposed to a simulated dive. It is therefore possible that the differences we found between these groups may represent collateral modifications only. To experimentally question the relationship between these alterations of the vascular function and resistance to DCS is the subject of continued investigation by our research group and others.

Conclusion

This study revealed a possible shift towards lower basal blood pressure in rats animals bred to be resistant to DCS with no difference in responses to hypo- and hypertensive drugs when compared to standard rats. These differences are compatible with differences in vasoactive circulating factors and might represent a possible mechanism of DCS-resistance.

Currently-used decompression procedures based on calculated algorithms are presently considered to be relatively safe. Nevertheless, the fact that DCS still occurs even without violation of the algorithm recommendations[6] indicates that, for at least a proportion of the diver population, current algorithms are not conservative enough. It is now well recognised that improvements in decompression algorithms based primarily on biophysical models, may be possible by identifying and modifying a diver's individual risk factors.

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Efficacy and safety of hyperbaric oxygen treatment to treat COVID-19 pneumonia: a living systematic review update

Sylvain Boet[1,2,3], Cole Etherington[2], Nibras Ghanmi[4], Paul Ioudovski[4], Andrea C Tricco[5,6,7], Lindsey Sikora[8], Rita Katznelson[9]

[1] *Department of Anesthesiology and Pain Medicine, The Ottawa Hospital, Ottawa, Canada*
[2] *Clinical Epidemiology Program, The Ottawa Hospital Research Institute, Ottawa, Canada*
[3] *Francophone Affairs, Faculty of Medicine, University of Ottawa, Ottawa, Canada*
[4] *Department of Public Health Sciences, Faculty of Health Sciences, Queens University, Ontario, Canada*
[5] *Li Ka Shing Knowledge Institute, St Michael's Hospital, Unity Health Toronto, Toronto, Ontario, Canada*
[6] *Epidemiology Division and Institute of Health Policy, Management, and Evaluation, Dalla Lana School of Public Health, University of Toronto, Toronto, Ontario, Canada*
[7] *Queen's Collaboration for Health Care Quality: A JBI Centre of Excellence, Ontario, Canada*
[8] *Health Sciences Library, University of Ottawa, Ottawa, Canada*
[9] *Department of Anesthesia and Pain Management, University Health Network, Toronto, Ontario, Canada*

Corresponding author: *Dr Sylvain Boet, Department of Anesthesiology and Pain Medicine, Hyperbaric Medicine Unit, The Ottawa Hospital, 501 Smyth Rd, Critical Care Wing 1401, Ottawa, K1H 8L6, Ontario, Canada*
sboet@toh.ca

Keywords
Hyperbaric medicine; Hypoxia; Infection; SARS-CoV-2

Abstract
(Boet S, Etherington C, Ghanmi N, Ioudovski P, Tricco AC, Sikora L, Katznelson R. Efficacy and safety of hyperbaric oxygen treatment to treat COVID-19 pneumonia: a living systematic review update. Diving and Hyperbaric Medicine. 2022 June 30;52(2):126–135. doi: 10.28920/dhm52.2.126-135. PMID: 35732285.)
Introduction: As the COVID-19 pandemic evolves, new effective treatment options are essential for reducing morbidity and mortality as well as the strain placed on the healthcare system. Since publication of our initial review on hyperbaric oxygen treatment (HBOT) for hypoxaemic COVID-19 patients, interest in HBOT for COVID-19 has grown and additional studies have been published.
Methods: For this living systematic review update the previously published search strategy (excluding Google Scholar) was adopted with an extension from 1 February 2021 to 1 April 2022. Study inclusion criteria, data extraction, risk of bias estimation and dispute resolution methods were repeated.
Results: Two new studies enrolling 127 patients were included in this update, taking the total to eight studies with 224 patients. Both new studies were randomised controlled trials, one at moderate and one at high risk of bias. Across these eight studies, 114 patients were treated with HBOT. All reported improved clinical outcomes without observation of any serious adverse events. Meta-analysis remained unjustified given the high heterogeneity between studies and incomplete reporting.
Conclusions: This updated living systematic review provides further evidence on the safety and effectiveness of HBOT to treat acute hypoxaemic COVID-19 patients.

Introduction

More than two years following the first reported case of COVID-19, the SARS-CoV-2 virus has infected over 482 million individuals worldwide, causing over 6.1 million deaths as of 28 March 2022.[1] While global vaccination efforts are underway, there are varying rates of both access to and compliance with COVID-19 vaccines across the globe,[2,3] and the efficacy of the vaccines for new variants of concern remains unclear.[4,5] Even if COVID-19 eventually becomes endemic, morbidity levels, death rates, and the susceptible proportion of the population are unpredictable.[6] Endemic infections can still cause disruptive waves as variants emerge.[6] The clinical experience to date suggests that 15 to 20% of COVID-19 patients require oxygen supplementation, and the mortality rate is 20 to 25% of patients requiring intubation and ventilation.[7–11] Finding treatments to help patients avoid extended hospital stays and intensive care unit (ICU) admission can also help the healthcare system to maintain capacity and recover from surgical and procedural backlogs incurred from the progression of the pandemic. To improve our global efforts to combat COVID-19, there is significant value in assessing novel treatment modalities that show promise in improving clinical outcomes and that could benefit patients in the future.[6,12]

In 2021, we published a systematic review of the efficacy and safety of hyperbaric oxygen treatment (HBOT) for

COVID-19 patients.[13] Based on the limited available literature at the time, it was concluded that emerging data may suggest "*HBOT is safe and may be a promising intervention to optimise treatment and outcomes in hypoxaemic COVID-19 patients*".[13] Interest in HBOT for COVID-19 has continued to grow and further clinical evidence is emerging. Given the importance of providing up-to-date evidence to clinicians, policymakers, and patients, particularly in the context of a global pandemic, the original systematic review has been transitioned to a living review. A living systematic review is "*a systematic review which is continually updated, incorporating relevant new evidence as it becomes available*."[14] Active monitoring of the evidence through monthly searches, followed by incorporation and dissemination of any new information that is identified, facilitates timely and up-to-date guidance to clinicians and decision-makers.[14] This report is the first update of the original review.

This living systematic review aims to provide an up-to-date synthesis of the available evidence on the efficacy and safety of HBOT for COVID-19 patients to inform clinical decision-making.

Methods

PROTOCOL

The protocols for the original systematic review (CRD42020209933) and for the current living systematic review (CRD42022309553) were registered with the International Prospective Register of Systematic Reviews (PROSPERO). This update is reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 Checklist.[15]

LIVING SYSTEMATIC REVIEW

We followed the same methods used in the original systematic review.[13] These are briefly summarised in Box 1. The search strategies are provided in *Appendix 1. This update repeated the search strategy

Box 1

Summary of systematic review methods

ELIGIBILITY CRITERIA

Population: Studies involving patients of any age with confirmed positive or suspected acute COVID-19

Intervention: Hyperbaric oxygen therapy (HBOT) administered with the intention of treating acute COVID-19 (minimum oxygen pressure of 1.4 atmosphere absolute [ATA])

Control: Standard of care or no treatment/comparator

Outcome: At least one clinical outcome (e.g., mortality; need for intubation), measured at any time point after HBOT initiated

Design: Randomised or non-randomised trial, case control, cross-sectional, case series, case reports, letters or abstracts presenting study data

Language of publication: Any language

Date of publication: Since December 2019, when the first human case of COVID-19 was reported

SEARCH STRATEGY

Developed by information specialist in collaboration with research team and peer-reviewed by second information specialist

INFORMATION SOURCES

MEDLINE via Ovid, EMBASE via Ovid, Scopus, relevant grey literature sources (e.g., World Health Organization, clinicaltrials.gov)

STUDY SELECTION AND DATA EXTRACTION

Conducted by pairs of independent reviewers in duplicate using Covidence systematic review software (Covidence, Melbourne, Australia); disagreements resolved through consensus, or third reviewer as needed

RISK OF BIAS AND CERTAINTY OF EVIDENCE

Conducted by two independent reviewers in duplicate; disagreements resolved through agreement or a third reviewer as needed; the Cochrane Risk of Bias 2 (RoB2) tool was used for RCTs.[16] The GRADE framework[17] was used to assess the certainty of the evidence for each study.

DATA SYNTHESIS

Descriptive summary

LIVING REVIEW AND LITERATURE SURVEILLANCE

Monthly surveillance and updates, submitted for publication when new literature changes conclusions or certainty of evidence, or when data obtained on additional outcomes

Footnote: *Appendices 1–5 are available on DHM Journal's website: https://www.dhmjournal.com/index.php/journals?id=296

as previously published (excluding Google Scholar), but updated to 1 April 2022.

For included randomised controlled trials, the Cochrane Risk of Bias 2 (RoB2) tool was used.[16] RoB2 assesses whether an individual study has a lower or higher risk of bias according to five domains: bias arising from the randomisation process, bias due to deviations from intended interventions, bias due to missing outcome data, bias in measurement of the outcome, and bias in selection of the reported results.[16] The tool also provides an overall risk of bias judgement of low/high/some concerns.[16]

The certainty of the evidence for included comparative studies was assessed using the GRADE framework.[17] GRADE considers five domains (risk of bias, indirectness, inconsistency, imprecision, and publication bias), and rates the certainty of the evidence as high, moderate, low or very low.[17]

Results

STUDY SELECTION

The updated literature search identified 80 potential studies for inclusion, of which 13 were duplicates, and two met inclusion criteria after abstract and full-text screening (Figure 1). A total of eight studies were included in this review (six from the previous review and two from this update).

STUDY AND PATIENT CHARACTERISTICS

An overview of studies included and patient characteristics from both the initial and current updated review is presented in Table 1 and *Appendices 2 and 3. Further details on the included studies in this update are available in Table 2. Both the studies identified in this update were conducted outside of North America: one in in Argentina and the other in Russia.

Figure 1
PRISMA Flow Diagram

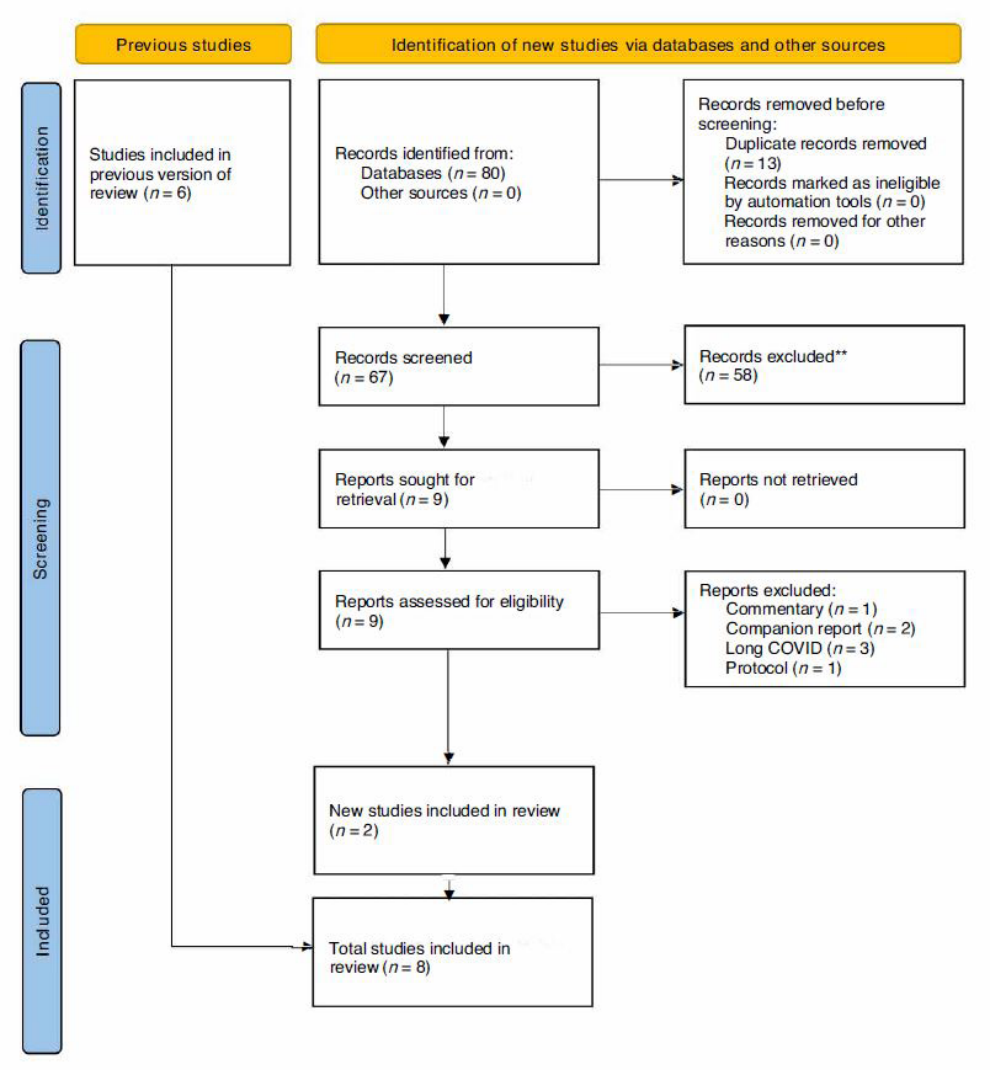


Table 1
Comparison of study and patient characteristics, initial review to current update; atm abs – atmospheres absolute; HBOT – hyperbaric oxygen treatment

Parameter	Initial review	Current update	Total
Studies (*n*)	6	2	8
Study design			
Case report (*n*)	2	0	2
Case series (*n*)	3	0	3
Cohort study (*n*)	1	0	1
Randomised controlled trial (*n*)	0	2	2
Patient characteristics			
Total patients (*n*)	97	127	224
Patients treated with HBOT (*n*)	37	77	114
Female (%)	12 (12.4)	58 (45.7)	70 (31.3)
Age range (years)	24–87	NR	24–87
Intervention details			
Length of sessions (minutes)	60–100	40–90	40–100
Mean number of sessions	1–7	4–6	1–7
Pressure range kPa / atm abs	152–203 / 1.5–2.0	141–162 / 1.4–1.6	141–203 / 1.4–2.0

Table 2
Characteristics of new studies included in this update; HBOT – hyperbaric oxygen treatment; ICU – intensive care unit; PCR – polymerase chain reaction; RCT – randomised controlled trial

Reference	Study design	Inclusion criteria	Exclusion criteria	Intervention	Control
Cannellotto[18]	RCT, *n* = 40 (20 per group), three centres	"*Patients in emergency department or ICU, >18 years of age, with confirmed diagnosis of COVID-19 by PCR or nasal swab, with pneumonia with oxygen dependence and no previous hospitalisation within the last 6 months*."	"*Patients unable to give consent, were pregnant or breast feeding, required mechanical ventilation, were unable to maintain prolonged sitting position (≥ 2 h) or had contraindications for HBOT*."	Monoplace 147 kPa 90 minutes ≥ 5 sessions Once daily	Standard of care
Petrikov[19]	RCT, *n* = 87 (57 HBOT, 30 control), single centre Two HBOT subgroups based on start of HBOT after admission: Group 1 (≤ 7 days): *n* = 28 Group 2 (> 7 days): *n* = 24	Patient admitted to hospital and clinical diagnosis of COVID-19	Not reported	Monoplace 142–162 kPa 40 minutes Number of sessions and frequency not reported	Standard of care

Table 3

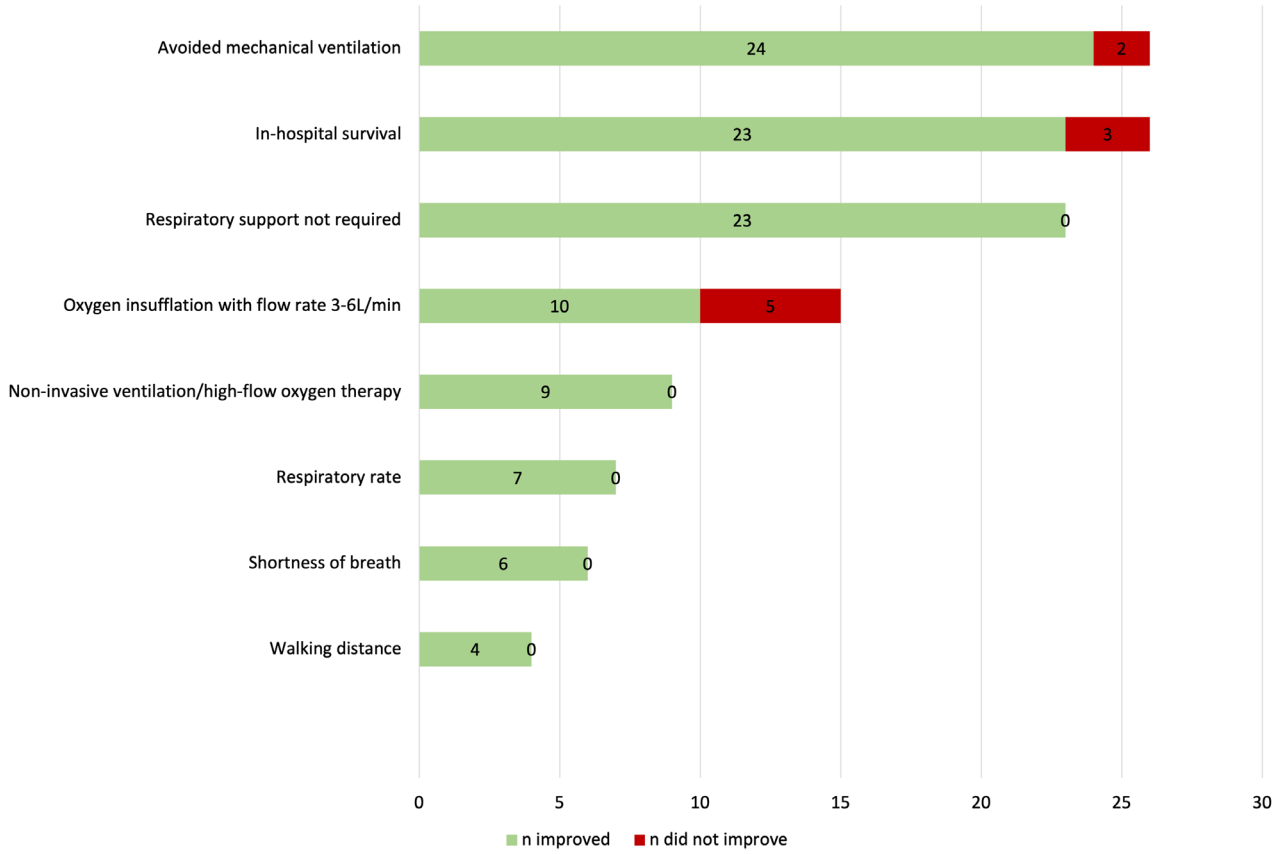
Effect of HBOT on COVID-19 patient outcomes reported by two new studies included in this update; HBOT – hyperbaric oxygen treatment; IQR – interquartile range; SpO_2 – peripheral oxygen saturation

Reference	Patients (*n*)	Timing of outcome measurement	HBOT sessions Mean (SD)	Biological outcomes	Imaging outcomes	Safety outcomes
Cannellotto[18]	40 (20 per group)	Within 30 days after admission	6.2 (1.2)	Nil	Nil	Ear discomfort (*n* = 1)
	Clinical outcomes	**Primary outcome - proportion of patients that recovered from hypoxaemia (SpO_2 ≥ 93%)** Control group: Day 3, 1 (8%); Day 5, 8 (40%); Day 10, 13 (65%); Day 15, 16 (80%) HBOT group: Day 3, 11 (55%); Day 5, 19 (95%); Day 10, 20 (100%); Day 15, 20 (100%) *Odds ratio (OR) of recovery from hypoxaemia (SpO_2 ≥ 93%) for HBOT vs. control group* Day 3, 23.2 (95% CI 1.6 to 329.6, *P* = 0.001); Day 5, 28.5 (95% CI 1.8 to 447.4, *P* < 0.001) **Co-primary outcome - median time to recovery** HBOT group: median (IQR) 3 (1.0–4.5) days; Control group: 9 (5.5–12.5) days (*P* < 0.010) **Secondary outcomes** *Acute respiratory distress* Control group: 3 (15%); HBOT group: 3 (15%) (*P* = 0.61) *Mechanical ventilation* Control group: 3 (15%); HBOT group: 1 (5%) (*P* = 0.61) *Death* Control group: 1 (5%), HBOT group: 1 (5%) (*P* = 1.00)				
Petrikov[19]	87 total 57 randomised to HBOT 30 randomised to control HBOT group divided into two subgroups based on start of HBOT after admission: Group 1 (≤ 7 days), *n* = 28 Group 2 (> 7 days), *n* = 24	Assessed over course of HBOT	Subgroup 1: 5.1 (2.5) Subgroup 2: 4.2 (2.0)	**Blood malone dialdehyde** decreased (HBOT group) from mean (SD) 4.34 (0.52) µmol·l^{-1} prior to HBOT to 3.98 (0.48) µmol·l^{-1} at day 7 **Total antioxidant activity** decreased (HBOT group) from 1.26 (0.28) mmol·l^{-1} to 1.21 (0.05) mmol·l^{-1} **Open circuit potential of platinum electrode** decreased (HBOT group) from -22.78 (24.58) mV to -30.45 (15.32) mV **Apoptotic lymphocytes** showed no significant change	Nil	Claustrophobia (*n* = 1) Pain in the ears (*n* = 4)

Table 3 continued

Petrikov[19] continued.	**Clinical outcomes**	**Oxygen saturation (SpO_2),** Mean (SD) Control group: Day 1, 92.6 (4.2); Day 3, 90.4 (4.9); Day 7, 91.3 (4.8); Day 14, 93.4 (4.7) HBOT group 1: Day 1, 91.9 (5.3); Day 3, 91.4 (3.8); Day 7, 92.1 (3.9); Day 14*, 96.1 +/- 2.8 (*$P < 0.05$ vs. baseline and vs. control) **National Early Warning Score (NEWS2),** Mean (SD) Control group: Day 4, 4.7 (2.3) points; Day 10, 4.0 (2.3) points HBOT group 1: Before HBOT, 4.4 (2.2) points; After HBOT, 1.2 (1.7) points ($P < 0.05$ for difference from the baseline in the HBOT group and from the control group value at day 10) **Ordinal scale for clinical improvement score,** Mean (SD) Control group: Day 4, 4.1 (0.7) points; Day 10, 3.9 (0.8) points HBOT group 1: Before HBOT, 4.2 (0.7) points; After HBOT, 3.0 (0.6) points ($P < 0.05$ for difference from the baseline in the HBOT group and from the control group value at day 10) **Respiratory support** (defined as oxygen supplementation through nasal cannulae or face mask with a flow of 3–6 $l \cdot min^{-1}$, in severe cases using high-flow oxygen therapy or non-invasive lung ventilation) *Non-invasive ventilation/high-flow oxygen therapy* (*P* not reported) Control group: Day 4, 10 (33.3%); Day 10, 8 (26.7%) HBOT group 1: Before HBOT, 9 (32.1%); After HBOT, 0 (0%) *Oxygen supplementation with flow rate 3–6 $l \cdot min^{-1}$* (*P* not reported) Control group: Day 4, 14 (46.7%); Day 10, 13 (43.3%) HBOT group 1: Before HBOT, 15 (53.6%); After HBOT, 5 (17.9%) *No respiratory support required* (*P* not reported) Control group: Day 4, 6 (20%); Day 10, 9 (30%) HBOT group 1: Before HBOT, 4 (14.3%); After HBOT, 23 (82.1%)

Figure 2
Summary of clinical outcomes for COVID-19 patients treated with HBOT, when reported, across all eight reviewed studies



This update identified two randomised controlled trials, one which is single centre[19] and the other which is multicentre.[18] Across all eight included studies, there were 224 patients (initial review: $n = 97$; update: $n = 127$). Of these, 114 were treated with HBOT (initial review: $n = 37$; update: $n = 77$). HBOT sessions ranged from 40 to 100 minutes, and the number of sessions ranged from one to seven. The pressure used ranged from 141–203 kPa (1.4–2.0 atmospheres absolute [atm abs]).

RISK OF BIAS

In the multicentre randomised controlled trial,[18] risk of bias was found to be low across all domains but one ("*risk of bias due to deviations from the intended interventions*") rendering an overall risk of bias assessment of "*some concerns*" with high certainty of evidence. In the single centre randomised controlled trial,[19] risk of bias was either found to be of some or high concern across each domain except for the domain "*risk of bias due to missing outcome data*", which was deemed low risk. Overall, this study[19] was rated as high risk of bias with moderate certainty of evidence. The risk of bias assessment for each study is provided in *Appendix 4.

EFFECTIVENESS OF HBOT FOR COVID-19

The two studies in this update assessed clinical outcomes (Table 3). Petrikov's study also assessed certain biological outcomes.[19] Improvements in all outcomes assessed for patients who were treated with HBOT compared to the control group were observed by both studies (Table 3). Across all eight studies, improvements were observed for a number of clinical outcomes for HBOT at pressures anywhere between 141 and 203 kPa (1.4 and 2.0 atm abs), including: in-hospital survival, median days to recovery, oxygen saturation, respiratory rate, shortness of breath, need for respiratory support, and walking distance (*Appendix 5). Five studies[18–22] reported patients treated with HBOT were able to avoid mechanical ventilation and one study reported improvement in the ordinal clinical outcomes scale.[19] Figure 2 summarises the number of patients who improved versus did not improve for each outcome, where data were available.

Discussion

This living systematic review update identified two new studies published since the completion of the original

review. As of this update, there are now eight studies which have assessed the efficacy and safety of HBOT for treating patients with COVID-19. Of note, the two new studies included in this update are the first randomised controlled trials published. Although continued investigation through rigorously conducted multicentre randomised controlled trials is still needed to draw definitive conclusions, the available evidence suggests HBOT may be an effective adjunctive treatment for COVID-19. It is difficult to comment definitively on safety on the basis of treating 114 patients but no serious adverse events have been reported in the reviewed studies.

In addition to the risk of bias present in the available studies, another challenge when assessing the effectiveness of HBOT for acute hypoxaemic COVID-19 patients is the lack of consensus in outcomes selection in the existing literature. We found a wide range of observed clinical outcomes, such as improved oxygen saturation, respiratory rate, walking distance, and in-hospital survival as well as avoiding the need for mechanical ventilation. The hyperbaric medicine community should develop a minimum set of core outcomes to be used in every study on HBOT for acute COVID-19. This could incorporate the World Health Organization ordinal COVID-19 scale that captures the main patient-centred outcomes into a single tool.[23,24] Consistent reporting of individual patient data across studies would also be beneficial for supporting future meta-analyses.

Based on published evidence, HBOT is a promising therapeutic option that could contribute to reduce the strain new variants continue to place on the healthcare system based on the ability to improve oxygenation without the need for intubation or mechanical ventilation. The published evidence reports a positive clinical effect of HBOT for acute hypoxaemic COVID-19 patients regardless of their specific HBOT regimen. Importantly, most studies found this positive clinical effect after just a few days, typically less than seven days. Interestingly, a number of reports suggest the mechanisms of action of HBOT in COVID-19 patients may include immunoregulatory effects in addition to correcting the oxygen debt.[25–29] These suggestions may have implications for other septic conditions for future research.

Even if the incidence of Omicron, the current dominant variant reduces, it is likely that new variants will continue emerge and their potential impact is unpredictable.[30] There are varying rates of both access to and compliance with COVID-19 vaccines across the globe,[2,3] and vaccine efficacy has been shown to wane over time.[31] Although current vaccines offer a certain immunity against new variants of concern, the protection level varies.[4,5] Despite intense research and some therapeutic progress, more low-cost, safe, effective and scalable treatment options are needed.[32] HBOT is an already approved drug/intervention for non-COVID-19 indications that is minimally invasive. It can be employed across a wide range of case-severity, unlike other interventions, which may be limited to narrow patient subgroups or time frames.[33–39] HBOT would not be subject to supply chain disruptions and product shortages, which have been observed throughout the pandemic for pharmaceutical interventions.[40] Of course, HBOT has some limitations such as chamber availability and requirement for transfer to a chamber from the hospital ward which should be acknowledged.

This living systematic review will be updated as required following monthly repetition of our search strategy. Our narrative review will be modified and meta-analysis will be performed as appropriate. The updated review will be submitted for publication when new literature changes the conclusions and/or certainty of evidence or when data are obtained on additional outcomes.

LIMITATIONS

This review is subject to several limitations. First, there are discrepancies in the quality of reporting between studies. Designing reporting guidelines specific to hyperbaric medicine is paramount to improvement in the quality of publication. Secondly, findings may be subject to various degrees of bias found in this review. In addition, a few studies that are likely to be relevant to our review are completed but not yet published according to registration databases.

Conclusions

This updated living systematic review provides further evidence on the promising effectiveness of HBOT to treat hypoxaemic acute COVID-19 patients.

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Review article

Perspective on ultrasound bioeffects and possible implications for continuous post-dive monitoring safety

Erica P McCune[1], David Q Le[1], Peter Lindholm[2], Kathryn R Nightingale[3], Paul A Dayton[1], Virginie Papadopoulou[1]

[1] *Joint Department of Biomedical Engineering, The University of North Carolina at Chapel Hill and North Carolina State University, Chapel Hill, NC, USA*
[2] *Department of Emergency Medicine, School of Medicine, University of California, San Diego, La Jolla, CA, USA*
[3] *Department of Biomedical Engineering, Duke University, Durham, NC, USA*

Corresponding author: *Dr Virginie Papadopolou, 9004 Mary Ellen Jones Building, 116 Manning Drive, Chapel Hill, NC 27599-7575, USA*
virginiepapadopoulou@gmail.com

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Abstract

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Ultrasound monitoring, both in the form of Doppler and 2D echocardiography, has been used post-dive to detect decompression bubbles circulating in the bloodstream. With large variability in both bubble time course and loads, it has been hypothesised that shorter periods between imaging, or even continuous imaging, could provide more accurate post-dive assessments. However, while considering applications of ultrasound imaging post-decompression, it may also be prudent to consider the possibility of ultrasound-induced bioeffects. Clinical ultrasound studies using microbubble contrast agents have shown bioeffect generation with acoustic powers much lower than those used in post-dive monitoring. However, to date no studies have specifically investigated potential bioeffect generation from continuous post-dive echocardiography. This review discusses what can be drawn from the current ultrasound and diving literature on the safety of bubble sonication and highlights areas where more studies are needed. An overview of the ultrasound-bubble mechanisms that lead to bioeffects and analyses of ultrasound contrast agent studies on bioeffect generation in the pulmonary and cardiovascular systems are provided to illustrate how bubbles under ultrasound can cause damage within the body. Along with clinical ultrasound studies, studies investigating the effects of decompression bubbles under ultrasound are analysed and open questions regarding continuous post-dive monitoring safety are discussed.

Introduction

Decompression sickness (DCS) is a condition caused by the formation and growth of bubbles from dissolved inert gases in the tissues when the body experiences decompression. The effects of DCS vary from symptoms such as skin itching, joint pain, numbness, and dizziness,[1,2] to rare but severe outcomes, such as coma or even death.[3] In the case of scuba diving, divers breathe gas at ambient pressure throughout the dive. As pressure increases with depth, so do the partial pressures of the inert gases breathed. This results in a pressure gradient from the inspired gas in the lungs to the rest of the body's tissues, which are saturated at sea level. During ascent, the pressure gradient reverses, and supersaturation can drive gas out of solution, resulting in bubbles in the tissues and bloodstream during and after decompression. Bubbles continue to appear in the venous blood for two to three hours post-dive and may cause problems by blocking blood vessels, mechanically distorting tissues, and inducing inflammatory cascades.[1]

Ultrasound monitoring, both in the form of Doppler and 2D echocardiography, has been used post-dive to detect decompression bubbles in the bloodstream, termed 'venous gas emboli' or VGE. Doppler ultrasound was first used in 1968 to detect intravascular decompression bubbles and became the predominant method for detecting VGE in divers.[4] In this case, VGE are detected aurally by employing continuous-wave Doppler detection with a single-element transducer with a separate transducer used as a receiver. This high-frequency sound is reflected by moving intravascular decompression bubbles and results in received chirp-like signals in the auditory range, which can be detected by a trained listener and used to provide a bubble grade.[5]

Figure 1
Venous gas emboli circulating post-dive can be detected using ultrasound via Doppler precordial or subclavian recording (audio) and precordial apical 4-four chamber view echocardiography (video). Note the differing probe placement for the two detection methods

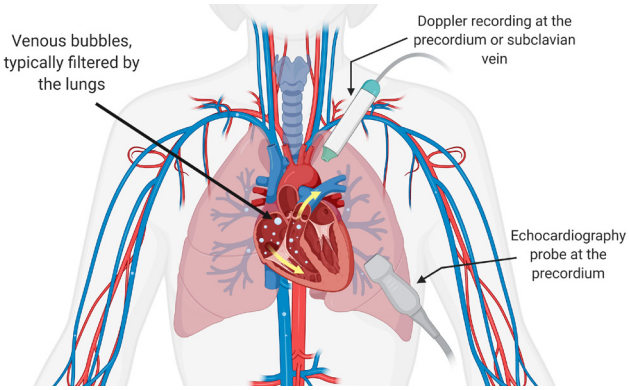
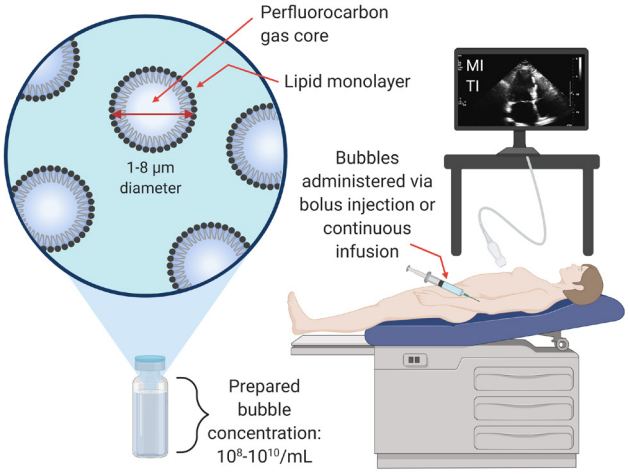


Figure 2
Clinical use and properties of ultrasound microbubble contrast agents



More recently, 2D echocardiography using a transducer array has been employed to visualise VGE in the heart. As with Doppler, the evaluation of these cardiac images allows raters to score circulating VGE and provide either a bubble grade or, more recently, employ frame-based bubble counting to evaluate VGE load.[6] Although data acquisition is more difficult, training for 2D echocardiography image evaluation is relatively quick,[6,7] unlike training for Doppler VGE detection, and this ease of training has shown 2D echocardiography to be a more economical form of evaluation compared to Doppler.[7] As a result, 2D echocardiography has quickly grown in popularity for post-dive decompression bubble analysis. These two methods of VGE detection are illustrated in Figure 1.

Post-dive VGE analysis is used as a tool for evaluating a diver's likelihood for developing decompression sickness. While VGE analysis cannot be used on its own to determine whether a diver will develop DCS, a lack of VGE is a good indication a diver will not develop DCS.[8] Also, despite the low specificity of VGE analysis, there is a definite positive association between VGE load and DCS incidence, with higher VGE grades corresponding to an increase in DCS risk.[9] Thus, ultrasound imaging provides a method for screening divers for DCS risk and can be used both for diving physiology research and in the development of decompression schedules for specific diving profiles. From the early use of Doppler in the 1970s to more recent echocardiography studies, it is well-established that there exists large variability in VGE loads not only for different dive profiles but also between subjects and for the same subject undergoing the same controlled dive profile.[10–12] Additionally, the time course of VGE varies significantly post-dive, so that regular monitoring intervals are paramount for correct quantification.[12–14] As such, continuous ultrasound monitoring could provide a more accurate post-dive assessment. The development of smaller, more portable echocardiography devices has increased the feasibility of this continuous monitoring. Continuous in-suit Doppler has been employed by NASA for bubble detection,[15] but this method has not yet been used for 2D echocardiography.

The increasing popularity of 2D echocardiography for post-dive monitoring and a push towards shorter intervals between image acquisitions or even continuous monitoring demands an evaluation of the safety of these methods. Ultrasound is considered the safest imaging modality to date; however, precautions still need to be taken when seeking to increase sonication time under abnormal imaging conditions, such as in the presence of bubbles in the tissues and bloodstream. In the realm of clinical ultrasound, established guidelines have resulted in the thermal index (TI) to avoid tissue heating and the mechanical index (MI) to avoid mechanical effects of ultrasonic waves on tissues. An MI safety limit of 1.9 is imposed during normal ultrasonic conditions, but more recent studies have shown that, in the presence of microbubble ultrasound vascular contrast agents, bioeffects, such as microvascular leakage, petechiae, cardiomyocyte death, and premature ventricular contraction, occur at much lower MIs.[16]

Microbubble ultrasound vascular contrast agents are small bubbles with an outer lipid shell and an inner gas core. Clinically, they are injected intravenously and most often used as an echogenic source to provide high contrast ultrasonic images of organ structure or blood volume and perfusion to an organ of interest. Studies have also investigated their use for gas transport, such as oxygen delivery to tissues,[17–19] and gas scavenging.[20] Free, unencapsulated bubbles have also been used clinically as contrast agents. For example, agitated saline is used in echocardiography to detect patent foramen ovale (PFO).[21] The properties and clinical use of encapsulated microbubbles can be seen in Figure 2.

Contrast agent manufacturer guidelines recommend setting the default MI to below 0.4 (Sonovue™)[22] or below 0.8

Figure 3
Microbubble behavior in a blood vessel under ultrasound sonication. Four mechanical effects of microbubbles are illustrated: microbubbles experience a push in the direction of ultrasound propagation (primary radiation force); can undergo cavitation depending on the sonication parameters (frequency match to their resonance diameter, transmit amplitude) which shrinks and expands the bubble; this oscillation creates local flow disturbances (microstreaming); and bubbles can coalesce (secondary radiation force)

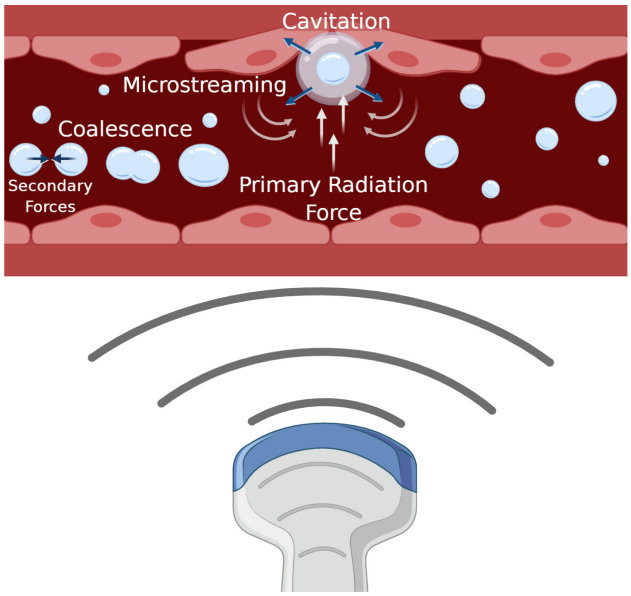
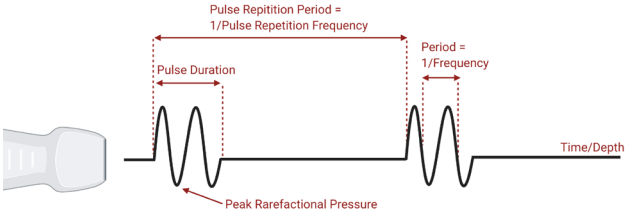


Figure 4
Properties of an ultrasonic wave. Note that horizontal distance represents time. Peak rarefactional pressure, pulse duration, pulse repetition period, and period are illustrated



(Definity™, Optison™)[23,24] for the safe use of microbubble contrast agents. Nevertheless, physicians still occasionally utilise a short sonication pulse at a higher MI (> 1.0) to momentarily break microbubbles in the field of view, before returning to low MI imaging (destruction-reperfusion technique for perfusion quantification).[25] To date, significant bioeffects from contrast imaging in humans have not been observed; however, due to bioeffects observed in some preclinical studies, the World Federation for Ultrasound in Medicine and Biology (WFUMB) has proposed that contrast imaging should be performed at an MI of less than 0.4 when possible to reduce the likelihood of bioeffects.[26]

2D echocardiography post-dive typically uses continuous imaging at > 1.2 MI to achieve higher quality images, which is significantly higher than the proposed 0.4 MI suggestion for imaging ultrasound contrast microbubbles. The properties of decompression bubbles, such as bloodstream concentrations and diameter distributions, are largely unknown and still debated, making direct comparisons between contrast agent microbubbles and VGE difficult; however, previous research showing the activation of gas bodies with ultrasound provides a reason to approach the sonication of gas-containing tissues with caution.[27] No studies to date have investigated potential mechanically induced bioeffects at the MIs used for post-dive evaluation.

This review aims to discuss what can be drawn from the current ultrasound and diving literature on this topic, and identify areas where more studies are needed. First, we provide an overview of ultrasound safety, introduce microbubble vascular contrast agents and summarise their dynamics under ultrasound that can lead to bioeffects. Next, we review the ultrasound bioeffects literature, focusing on the pulmonary and cardiovascular systems of special interest to diving physiology. Finally, we consider previous studies combining diving and low-frequency ultrasound and discuss open questions regarding the safety of post-dive echocardiography.

Ultrasound bioeffect mechanisms

HOW BUBBLES BEHAVE UNDER ULTRASOUND

Bubbles under ultrasound experience different mechanical effects depending on the surrounding environment and the ultrasound parameters used. The main mechanical effects of bubbles under ultrasound are described below, along with the type of bioeffects each may generate. These are also graphically depicted in an idealised blood vessel schematic in Figure 3.

1. Cavitation

Ultrasound imaging employs sound, in the form of pressure waves, to produce images. Pressure waves emitted from a transducer propagate and, when reflected off interfaces with different acoustic impedance, are received by the same transducer to form an image. The body is composed of tissues and water, which are incompressible. Gas, however, is compressible, and bubbles excited with a pressure wave will shrink during periods of increased pressure and expand during periods of rarefaction. Since VGE are bubbles in blood, surrounded by incompressible liquid, small VGE can expand and shrink under ultrasound. The properties of sound waves including the definition of various acoustic parameters can be seen in Figure 4.

Acoustic cavitation is the expansion and contraction of a gas bubble within a sound field. When a bubble in liquid is exposed to an acoustic field, that bubble will oscillate

around an equilibrium radius. Two types of oscillation can occur depending on the acoustic field insonifying the bubble: stable (non-inertial) cavitation and inertial cavitation. Under stable cavitation, a bubble undergoes repetitive oscillation over multiple acoustic cycles. When the acoustic amplitude is increased, oscillating bubbles reach a point where there is greater bubble expansion than there is contraction. This leads to the rapid growth and then violent collapse of the bubble (with the bubble fragmenting and gas dissolving into the surrounding fluid) in a process known as inertial cavitation. Both forms of cavitation can result in bioeffects; in some cases these effects can have harmful unintended consequences, but they may also be purposefully elicited in therapeutic settings. During stable cavitation, oscillating bubbles produce heat and cause localised shear stress or microstreaming of fluid near the bubble.[28] While sometimes undesirable, the physical effects from stable cavitation are utilised in therapeutic settings to produce pores in membranes for transporting of genetic material in a process called sonoporation[29] or to lyse blood clots.[30] Sustained stable cavitation, in the absence of unstable cavitation, is also used to temporarily open the blood-brain barrier[31] and the amount of stable cavitation has also been shown to correlate with the concentration of therapeutic agents delivered via focused ultrasound blood-brain barrier opening.[32] The collapse associated with inertial cavitation produces violent effects such as localised but extreme temperature rises and high-velocity liquid jets that cause mechanical damage.[33] Inertial cavitation can produce harmful effects such as micro-vessel rupture[34,35] and blood cell rupture.[36] As with stable cavitation, however, the effects of inertial cavitation are used therapeutically. Inertial cavitation can be used to fractionate tissue,[37,38] with applications in tumor ablation, open the blood-brain barrier with some bubble diameters,[39] release drugs from micelles,[40] and can be precisely controlled for sustained sonoporation.[41]

Although the above studies deal with encapsulated microbubbles, it should be noted that the lipid layer of the bubble is not what enables cavitation or other bubble mechanics. While sonication of free bubbles, such as saline, does not cause bioeffects,[42] it is not the bubbles themselves but rather their size away from resonance and timescale that prevent bioeffect generation. Free bubbles are capable of cavitation at even lower pressures than stiff-shelled encapsulated bubbles.[43] When not under supersaturated conditions, however, these unencapsulated bubbles have half-lives of only a few seconds.[44]

2. Microstreaming

As bubbles rapidly expand and contract during stable cavitation, fluid flow can be generated near the bubble in a process known as microstreaming. This flow around the oscillating bubble can impose shear stress on surrounding surfaces and result in cell death.[45,46] The stress exerted via microstreaming can also be used therapeutically; for example, to open membrane pores for therapeutic agent delivery via sonoporation.[47]

3. Radiation forces and coalescence

As ultrasound waves propagate through a medium, they have an associated momentum that can be imparted onto objects in their path. If an object in the beam's path is free to move, the imparted momentum will result in the translation of the object in the direction of the beam.[48] This imparted force is known as the primary radiation force. Bubbles pushed hard enough with this force may attain high speeds, and collisions with these high-speed bubbles have been proposed to be the cause of cell lysis[49] and clot lysis.[30] The pushing of microbubbles can also be used to localise and concentrate contrast agents near vessel walls to assist in the delivery of targeted agents.[50]

As microbubbles oscillate, they act as a secondary source of sound.[48] This source of sound is associated with another radiation force referred to as the secondary radiation force, which can cause attraction between nearby microbubbles or even other nearby particles. When two bubbles are close enough to one another via the primary and secondary radiation forces, they may fuse together as a single bubble in a process known as coalescence. The coalescence of microbubbles occurs because of the thinning of the bubble film. As encapsulated bubbles expand under an ultrasonic field, the flow between the bubbles creates a pressure reduction, and the two bubbles will move closer towards each other.[33] Once the bubbles are adjacent, their expansion will cause the pressure in the film between them to increase, which results in the thinning and flattening of the bubble surfaces.[51] The continued bubble expansion leads to the drainage of the film until it reaches a critical thickness.[51] At this point, the film ruptures and the bubbles coalesce into a single bubble. Whereas free bubbles coalesce more readily during collisions without the use of ultrasound, the resulting radiation forces from ultrasound make the coalescence of encapsulated bubbles much more likely.[52] The use of the secondary radiation force may allow for the combination of therapeutic agents encapsulated in microbubbles or may be used to aid in concentrating agents to a targeted area.[50]

RELEVANCE OF THE MECHANICAL INDEX

The mechanical index is used to infer the risk of nonthermal mechanical effects during diagnostic ultrasound. Apfel and Holland developed this metric using theoretical and experimental observations to determine the acoustic pressure amplitude required to cause an optimally sized bubble to undergo inertial cavitation.[53] In their work, a threshold level of 0.7 MI was reported for initiating inertial cavitation. Interestingly, because the FDA guidelines are based upon the acoustic output in use commercially prior to the 1976 FDA Medical Device Amendments (by law), although the MI computation is derived from Apfel and Holland, the

FDA MI guideline is 1.9. It is important to note that the MI calculation is based only on the threshold for generating inertial cavitation for free bubbles and not on the severity of effects resulting from inertial cavitation.[54]

The FDA defines the MI as the ratio of the peak rarefactional negative pressure (in MPa) adjusted for tissue attenuation (derated by 0.3 dB.MHz-cm^{-1} and the square root of the center frequency of the wave (in megahertz (MHz)), thus MI = $\frac{p_{r,0.3}}{\sqrt{f}}$.

From this equation, at a set peak rarefactional negative pressure, lower frequencies lead to higher MI values, indicating a higher possibility of inertial cavitation. This is because cavitation is more likely under long wavelength stimulation (low frequencies) when bubbles have more time to expand and is less likely under short wavelength stimulation (high frequencies) when sufficient time is not provided for bubble growth.[33]

It is important to note the conditions under which the MI was developed. First, there is an assumption of pre-existing microscopic gas nuclei in the body.[53] While this is an accurate assumption for gas containing bodies such as the lungs and intestine, the use of microbubble contrast agents, and potentially even the case of circulating decompression bubbles, it proves to be less applicable for tissues not known to contain gas,[55] such as most soft tissues including muscle, fat, and cardiac tissue. Second, the MI assumes the existence of optimally sized bubbles *in vivo*.[53] In some situations, this may be a reasonable assumption, such as in the case of contrast agents where the bubble size distribution is at least known initially. Most tissues, however, do not contain these pre-existing, optimally sized bubbles, meaning that the MI is not necessarily a good predictor of *in vivo* cavitation.[56] In the case of decompression sickness, bubbles are present, but their size is debated. VGE with diameters above 20–30 µm have been detected using 2D echocardiography, and theoretical calculations and new imaging techniques, such as a dual-frequency system for detecting and sizing bubbles,[57] also predict the presence of smaller bubbles < 10 µm.[58] One study, for example, detected microbubbles in the 1–10 µm diameter range in swine following hyperbaric chamber dives.[59] Third, the MI was developed assuming only a single acoustic period of sonication typical of traditional imaging schemes. This is not the case for some forms of ultrasound imaging, such as Doppler and acoustic radiation force impulse imaging, that employ several hundred acoustic periods.

There is debate about the validity of using the MI as a predictor of cavitation. This metric only accounts for the onset of inertial cavitation and does not include other cavitation events such as subharmonic emissions from stable cavitation, and it is a poor predictor of ultrasound contrast agent rupture.[60] As a result, other cavitation metrics have been proposed, the most notable being the cavitation index $I_{CAV} = \frac{P_r}{f}$.

This seeks to describe the cavitation process as a whole.[60] Under this metric, the likelihood of ultrasound contrast agent rupture increases for $I_{CAV} > 0.02$.[60] Aside from the issue of the MI not accounting for other cavitation events and tissues without pre-existing, optimally sized bubbles, this measurement system also only considers peak rarefactional pressure and frequency. It is important to note that other factors, such as sonication time, pulse duration, pulse repetition frequency (PRF), and even the waveform shape, also contribute to the likelihood of cavitation and the occurrence and severity of bioeffects.[54] Computational studies have been conducted investigating the effect of increased pulse durations on the inertial cavitation threshold. Church found that under the sonication of liquids, such as urine, water, or blood, increased pulse durations reduced the cavitation threshold as much as 6–24%, although the effect on tissue was minor.[56] Compared to some experimental data, inertial cavitation thresholds generated under the MI method do not always agree with the frequency response.[61] From this disagreement, some alternative methods have been proposed such as modifying the frequency exponent in the MI equation[56] or adopting a two-criterion model that considers both the inertial cavitation and also a fixed value for the maximum radius a bubble may attain during expansion.[61]

Despite its inaccuracies and over-simplifications, the MI remains a useful metric for evaluating the threshold for inertial cavitation and bioeffect production in certain scenarios. For example, when diagnostic B-mode imaging, which employs only a few acoustic periods, is used on gas containing bodies, the MI may provide a useful way to indicate frequency and acoustic pressure combinations that are more likely to lead to cavitation-induced bioeffects. Guidelines have been released advising caution using MIs above 0.4 for diagnostic imaging of tissues with gas-containing bodies,[26] which is significantly below the FDA's 1.9 MI guideline and the commonly used 1.2 MI for post-dive echocardiography. In the following discussion of experimental studies evaluating organ bioeffects under diagnostic imaging, the MI will be strongly considered, although other sonication factors that play a role in bioeffect production, such as sonication duration, will also be discussed.

Pulmonary and cardiovascular bioeffects

Although the papers discussed in this section do not focus on ultrasound as it relates to scuba diving, the pulmonary and cardiovascular systems are directly sonicated during post-dive echocardiography, making it useful to understand how they may be affected by ultrasound sonication. It is important to note, however, that the pulmonary and cardiovascular systems are not the only systems that suffer

from ultrasound-induced bioeffects. Although they will not be discussed in detail here, the intestines, kidneys, bones, and even nervous system experience unique effects under ultrasound.[28] Since this review is focused on post-dive echocardiography, however, the discussion below will be kept to the two most relevant systems. It should be noted that while this section discusses negative effects the pulmonary and cardiovascular systems may experience under ultrasound, overall ultrasound is considered the safest imaging modality. The effects described below serve as a cautionary tale for the use of continuous ultrasound without prior safety investigations, as they demonstrate harm from unusual sonication circumstances (i.e., in the presence of gas bodies, at high pressures, etc).

PULMONARY BIOEFFECTS

Although the lungs are not the focus of post-dive 2D echocardiography, they can receive exposure as the beam passes through the chest wall to the heart. Whereas the cardiovascular system contains circulating VGE post-dive that provide a potential source of gas cavitation under ultrasound, the lungs are comprised of pre-existing gas bodies. This makes it important to consult the literature on the potential for bioeffect generation in the lungs, especially when considering extending the duration of post-dive echocardiography.

Many murine studies have found that lung haemorrhage is possible under diagnostically relevant levels of pulsed ultrasound sonication, with typical thresholds between 0.4 and 1.4 MPa peak rarefactional pressure, frequencies from 1.1 MHz to 12.0 MHz, and an MI range of 0.37–1.0.[62–66] This pulmonary capillary haemorrhaging resulting from sonication has been shown to correlate with the length of comet-tail artifacts,[64,65] suggesting that these artifacts may be used to indicate developing damage during imaging. These results illustrate the potential for lung haemorrhage to occur in rats and mice at MIs much lower than the 1.9 MI FDA guideline. The sonication frequency, however, does not appear to be a strong factor in determining the haemorrhage threshold, making the MI a poor predictor for damage.[65,67] Despite haemorrhage occurring at low sonication pressures in murine models, some researchers have speculated that the mouse is a poor model for damage that could occur during human diagnostic imaging.[68,69] This is substantiated by cross-species studies that have found less damage occurring in larger animals, such as rabbits and pigs, compared to rats and mice at the same sonication parameters.[68,70] Zachary and O'Brien concluded that a species' sensitivity to ultrasound is likely determined by anatomical and physical properties such as alveolar diameter, thickness of alveolar septa, lung compliance, and pleural thickness,[70] which all differ significantly between humans and rodents. It is also important to note that these studies investigating lung haemorrhage thresholds focus ultrasound directly on the lungs, whereas lung ultrasound exposure during echocardiography is more incidental (and currently of short duration).

To determine whether the results of small animal studies are applicable to humans, researchers have investigated the effects of diagnostic imaging on both human and monkey lungs. Damage has been shown to be possible with clinical diagnostic settings in monkeys, but only minimal damage was found using the maximum diagnostic ultrasound settings.[71] A study on 50 human subjects undergoing clinical echocardiography at 1.3 MI found no lung damage, leading the authors to conclude that human lungs are not as sensitive as those of animals.[72]

The mechanism by which ultrasound causes lung haemorrhage is not well understood. The interaction of ultrasound and alveolar gas is likely the primary cause of lung damage, as determined by the low sensitivity of fetal swine lungs to ultrasound compared to adult lungs since fetal lungs contain no gas.[73] Although the interaction with gas is the likely cause, inertial cavitation is not believed to be the mechanism by which gas causes damage. Evidence for this includes the lack of frequency dependence on the haemorrhage threshold,[65] the lack of difference in lung damage due to positive or negative peak pressures (the use of negative peak pressures should lead to more damage if inertial cavitation was the mechanism),[74] and the lack of effect of hydrostatic pressure on damage.[75] Although the exact form of gas body activation leading to haemorrhage remains unknown,[76] hypothesised mechanisms include the acoustic radiation surface pressure at the tissue-air interface.[77]

Although it appears that lung damage due to human echocardiography under typical clinical conditions is unlikely, the effect of increasing sonication time should be considered. Murine studies have found that increased exposure duration increases the surface area of lung lesions resulting from ultrasound.[78,79] Even with the same total sonication on-time, longer exposure durations can lead to greater haemorrhage and a lower sonication threshold.[79] The effect of exposure duration is so significant in determining the occurrence and extent of lung damage that its inclusion into the MI equation for lung sonication has been suggested.[67] Still, it should be noted that the previously mentioned human clinical diagnostic study performed echocardiography for as long as 50 minutes and still found no lung haemorrhage.[72] Overall, diagnostic echocardiography in humans seems unlikely to cause lung damage using clinical settings, but it may be wise to exercise caution when implementing long exposure durations.

CARDIOVASCULAR BIOEFFECTS

Although there is a lack of research regarding the interaction of decompression bubbles and echocardiography, there is extensive research on an interesting parallel: the use of

microbubble contrast agents during echocardiography. In contrast echocardiography, microbubbles are introduced into the bloodstream, where they are confined to the vasculature, as an echogenic source to provide higher quality images. When sonicated, these contrast agents have the potential to cavitate and induce bioeffects through the mechanisms previously described. To better understand the effects of cavitating bubbles in the cardiovascular system, this section will provide a literature review of the bioeffects elicited under diagnostic ultrasound conditions in both the heart and bloodstream along with a discussion of the safety of contrast echocardiography.

1. Cardiac bioeffects

Human and animal studies have revealed the production of many cardiac bioeffects when exposing contrast agents to diagnostic imaging conditions. Examples of generated bioeffects include capillary rupture,[35,80,81] premature ventricular contraction,[80,82–85] ventricular damage,[34] cardiac bio-marker release,[86,87] and mortality.[84] There is great variation in the settings that elicit these bioeffects, however. Mortality, for example, occurred only in extreme conditions far removed from traditional echocardiography: continuous ultrasound focused on the heart at a low frequency, maximum MI, a continuous bolus injection of contrast agents, and a sonication duration of over 9 minutes.[84] Unlike the production of pulmonary capillary haemorrhage, many cardiac studies have found a strong MI dependence on cardiac bioeffect production. One study, for example, found a strong damage dependence on the MI in rats where damage occurred slightly below 0.4 MI and increased with increasing MI.[35] Similar low MI thresholds have been found in contrast echocardiography rat studies: microvascular leakage occurred with exposure above 0.3 MI,[80] higher rates of mortality occurred with pressures above 0.6 MPa at 1.3 MHz (above 0.53 MI),[84] and premature ventricular contraction occurred with thresholds between 0.3 and 0.77 MI.[80,83,84] Larger animal and human studies, however, have found higher thresholds required for generating bioeffects. In an open-heart canine model, capillary rupture occurred with both 1.0 and 1.8 MI, although significantly more damage was produced with 1.8 MI.[81] *Ex vivo* rabbit heart sonication with microbubbles showed damage occurring with an MI greater than 0.8 and more damage occurred when using a lower frequency,[34] an outcome the MI model predicts. Human models show even greater thresholds. In one human clinical contrast echocardiography study, an MI of 1.5 elicited premature ventricular contraction whereas a 1.1 MI did not.[85] Another study found increased release of the cardiac bio-markers troponin I, creatine kinase myocardial band (CK-MB), and myoglobin in the coronary sinus, suggesting microscale damage to cardiomyocytes, when imaging at 1.5 MI in triggered second harmonic mode but not with a mode that implemented an alternating low-high combination where 0.2 MI was interrupted with 10 images at 1.7 MI every minute.[87]

The MI is not the only relevant setting in relation to damage, however. Some studies have shown that sonication time impacts the amount of damage produced. At high pressures, mortality has been shown to gradually increase as the sonication time increases from nine to 30 minutes[84] and bio-marker release increases with time up to 15 minutes.[87] Bioeffect generation during contrast echocardiography also depends on the concentration and infusion rate of microbubbles. Increased infusion rates are associated with greater premature ventricular contraction[82,85] and greater microbubble dosages are known to produce more capillary leakage.[80]

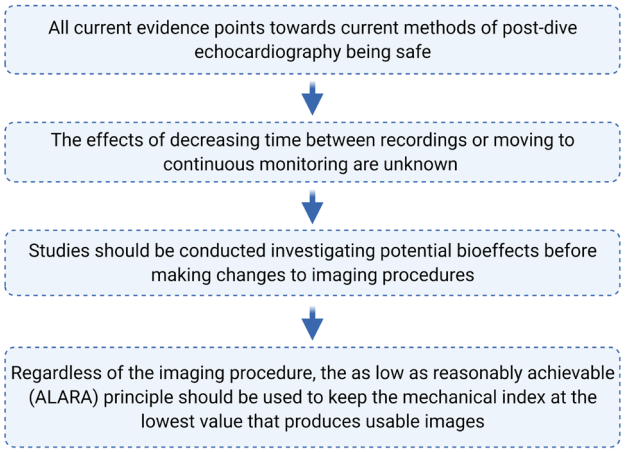
Despite the above studies that found bioeffect production with contrast echocardiography, human[88] and animal[86] studies have found no negative impacts from intermittent ECG-triggered contrast echocardiography at MIs around 1.0, and multiple reviews have concluded that contrast echocardiography has been shown to be safe in regards to the fairly insignificant findings of many studies.[25,89] Several major retrospective studies have found no increased risk of negative effects from the use of contrast agents during echocardiography.[90–93] The above studies also have several limitations that hinder their applicability to clinical settings. First, several of the studies employ contrast dosages much higher than those used clinically.[35,80,84,86] Many studies are also conducted on small animals or *ex vivo* organs,[34,35,80,81,86] meaning the studied hearts likely received greater ultrasound organ coverage or less tissue attenuation than would be present in clinical human use. Lastly, the studies on human subjects concede that the study population is more likely to experience arrythmias than healthy individuals,[82] potentially skewing results. Even so, contrast agent product inserts warn of potential arrythmia generation with MIs above 0.8[22–24] and caution has been recommended when using moderate and high MIs in contrast echocardiography.[25]

2. Vascular bioeffects

The use of contrast agents in vasculature provides an interesting parallel to sonication of circulating decompression bubbles. Since microbubble contrast agents travel through the bloodstream after injection, it is important to consider the potential interaction of these bubbles with blood cells under ultrasound sonication. Haemolysis, the destruction of blood cells, has been found with the sonication of microbubble-containing vasculature. Animal studies with contrast agents have found inertial cavitation to be the primary mechanism for haemolysis, as indicated by the strong correlation between the amount of haemolysis and the amount of inertial cavitation recorded using a cavitation detection system.[36,94,95] Increasing the dissolved oxygen (in normobaric conditions) in the blood, introducing more cavitation nuclei, also leads to greater inertial cavitation and greater haemolysis,[96] supporting inertial cavitation as the mechanism causing haemolysis. The amount of haemolysis occurring also shows a strong frequency effect where lower frequencies produce

greater haemolysis, and the amount of haemolysis increases with increasing MI.[95,97] Despite this, even when sonicating *in vitro* blood at MIs > 1.9, much greater than what would be used clinically, the levels of haemolysis produced are less than 5%[97,98] or almost indistinguishable from sham treatments.[36] Other studies have simply found no evidence of haemolysis even at maximum diagnostic settings.[99] The high thresholds necessary to invoke even minimal red blood cell destruction with contrast agents suggests that harmful levels of haemolysis are unlikely during diagnostic conditions.[28,36,76,97,98]

Figure 5
Current conclusions regarding post-dive monitoring safety



Relevance to diving

PREVIOUS STUDIES

Sonar and diagnostic ultrasound use vastly different parameters and are not comparable exposures (but we include this section for completeness). Of particular note, sonar typically uses frequencies in the kilohertz (kHz) range, which is much lower than the MHz frequencies used in diagnostic ultrasound. Sonar often transmits long or continuous signals, whereas diagnostic ultrasound most often uses pulsed sequences. The exposure in the studies in this section also occur during the dive bottom time, instead of post-dive. Despite the differences from post-dive echocardiography, the discussion of sonar exposure to divers still offers interesting insights into the potential interactions of decompression bubbles and ultrasonic waves. Several experimental studies have investigated the potential for decompression bubbles to grow under sonar. A computational study investigated the potential for bubbles of 1–10 μm initial radius in dissolved gas concentrations of 100–223% to grow under low-frequency ultrasound.[100] They found that under these conditions, sound pressures greater than 210 dB re 1 μPa (31.6 kPa) resulted in rapid bubble growth to sizes large enough to block capillaries and other small blood vessels, but that pressures below 190 dB re 1 μPa (3.16 kPa) were unlikely to result in bubble growth.[100] Supporting the conclusions of this study, several animal or *ex vivo* tissue studies under simulated dives found the potential for bubble growth under sufficiently high sound pressures. Prawns in 203 kPa hyperbaric conditions exposed to sound at 37 kHz and 1.4–2.8 MPa during a 10-minute bottom time presented bubbles for a longer period of time and with higher mean volumes than those not exposed to sound.[101] Bubble growth was also found in supersaturated *ex-vivo* blood and tissues when exposed to 37 kHz sound at pressures above 50 kPa.[102] Even sound pressures below 3.16 kPa have been found to elicit bubble growth in supersaturated conditions. Rats experiencing a simulated diving profile in a hyperbaric chamber that were exposed to 1.7 kPa sound at 37 kHz for the 60-minute bottom time produced larger bubbles and higher bubble densities than those with no sound exposure.[103]

Not only has sonar been shown to increase the amount or size of decompression bubbles, it has also led to increased damage or mortality in some studies. Immersed explanted pig lungs exposed to 22 and 36 kHz at 1 kPa and 0.8 kPa, respectively, incurred pulmonary microhaemorrhages.[104] A recent study found that rats exposed to diving profiles and 8 kHz sound experienced 20% mortality (vs. no deaths in the diving control group) and rats exposed to 8 kHz and to 15 kHz sound experienced higher rates of neurological decompression sickness.[105]

Few human studies exist to compare to the findings of animal and *ex vivo* tissue studies. Two case studies of divers exposed to continuous underwater sound reveal potentially recurrent harmful non-auditory effects such as lightheadedness, agitation, and the inability to concentrate, but these effects are difficult to validate.[106] Other human studies show that harmful effects from sonar exposure during dives are unlikely, but also conclude that further studies should be conducted for adequate conclusions.[107,108]

OPEN QUESTIONS

Although comparisons can be made between post-dive echocardiography and the use of both clinical diagnostic imaging with contrast agents and with sonar exposure during dives, there are too many parameter differences for either to provide a true parallel. Current post-dive protocols stipulate that measurements should be conducted for 120 minutes from completion of the decompression period, that an initial measurement should be made within 15 minutes following decompression followed by measurement intervals of no more than 20 minutes, and that sonication intensities and scan durations kept as low as reasonably achievable.[14] It should be noted that protocols such as these have been used for decades and adverse reactions in divers have not been reported. These conclusions are outlined in Figure 5.

The above studies on pulmonary exposure during diagnostic echocardiography on humans or large mammals appear

to indicate that damage is unlikely during typical clinical conditions,[68,70,72] even potentially during extended imaging durations. Post-dive, however, the pulmonary system plays an important role in filtering out circulating VGE. The lungs post-dive could be more sensitive to the effects of ultrasound due to the presence of VGE, which could in turn hinder this filtering capacity.

The results from clinical contrast echocardiography studies are difficult to interpret. Many studies indicate cardiovascular damage is possible at clinical settings with the introduction of microbubble contrast agents,[80–83,85,87] but most of these studies were conducted on small animals with contrast agent concentrations larger than typically used in therapeutic procedures.[80,81,83,86] Even with high VGE concentrations, individual bubbles can normally be detected on echocardiograms, indicating potential VGE concentrations much lower than the bubble concentrations used in ultrasound contrast agent procedures; however, there are varying radii of VGE and small circulating bubbles, or stationary tissue bubbles if present, that may not be picked up by echocardiography, making concentrations unknowable. Additionally, dissolved gas in the plasma from tissue supersaturation is not detectable with echocardiography, further complicating the question of gas concentration within the bloodstream. Ultrasound contrast agents are also confined to the vasculature, whereas VGE probably arise in the microcirculation of supersaturated tissues where extravascular bubble formation is also likely to be occurring. Most studies indicate that higher pressures and lower frequencies (higher MIs) result in more damage,[34,35,81,84,85] which does lead to the question of whether the typical 1–2 MHz and 1.2 MI post-dive echocardiography could result in damage from cavitating decompression bubbles. Human studies resulting in cardiovascular damage or premature ventricular contraction from contrast echocardiography were also conducted on populations more likely to experience cardiovascular difficulties.[82] Finally, contrast echocardiography studies have indicated higher occurrences and greater damage with extended sonication times,[78,79,84,87] indicating that extended post-dive sonication times could potentially result in a greater risk of bioeffects.

Studies on diving humans and animals exposed to sonar may leave the most unanswered questions, although this sonar sonication is very different to diagnostic ultrasound. These studies indicate that decompression bubbles in supersaturated conditions can grow when exposed to ultrasound[100–103] and potentially result in more severe decompression sickness.[104,105] These studies, however, use a much lower sonication frequency, and therefore higher MI, than that used in diagnostic imaging. Whereas the sonar studies focus on ultrasound in the low kHz range, echocardiography uses frequencies on the order of 1 MHz. Although it was previously thought that bubbles would most strongly oscillate when exposed to their resonant frequency, meaning that bubbles with low μm diameters would respond most strongly to MHz ultrasound, new studies have shown that lower frequency ultrasound, such as 250 kHz, causes bubbles to expand to more than 30 times their equilibrium size.[109] This raises the question as to whether sonar might cause bubbles to oscillate more strongly than diagnostic frequencies, meaning that the expansion seen in sonar conditions could potentially be less likely for diagnostic conditions. There are also questions as to whether supersaturated tissues exposed to ultrasound during dive bottom times would be more likely to grow or produce more bubbles than tissues that have decompressed post-dive, or whether the presence of circulating bubbles that result from the decompression could lead to stronger effects from ultrasound. The location of the sonication probe also differs from sonar studies and diagnostic studies; in diagnostic imaging, the probe is placed directly on the skin of the patient, giving them direct ultrasound exposure, whereas when humans and animals are exposed to sonar, the transducer is typically much further away. Lastly, the pulse repetition frequencies differ greatly between sonar exposure and diagnostic imaging. Sonar uses much lower pulse repetition frequencies than diagnostic imaging, meaning that patients under diagnostic imaging are subject to more frequent ultrasound exposure. These numerous considerations make it difficult to assess the potential hazards of continuous post-dive echocardiography.

Conclusion

Ultrasound has the potential to generate bioeffects in divers through sonar and in the pulmonary and cardiovascular systems through diagnostic ultrasound imaging, especially under conditions of high acoustic pressure, low frequency, and long duration sonication. Despite this, no research has been conducted on the safety of echocardiography for the evaluation of VGE load post-dive. Although the above research offers interesting insights into the role of ultrasound in bioeffect production and areas of possible concern, no conclusive statements can be made regarding the safety of continuous post-dive echocardiography. Since little information is known, sonication pressures should ideally be kept as low as reasonably achievable (the ALARA principle) to avoid any potential bioeffects. To avoid cavitation-related effects, sonication frequency should also be kept as high as possible. Further studies should also be conducted investigating the potential for post-dive echocardiography to produce bioeffects in divers.

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Case report

Hyperbaric oxygen treatment in a rare complication of intramuscular injection: four cases of Nicolau syndrome

Sefika Korpinar[1]

[1] *Department of Underwater and Hyperbaric Medicine, Faculty of Medicine, Canakkale Onsekiz Mart University, Canakkale, Turkey*

Corresponding author: *Dr Sefika Korpinar, Department of Underwater and Hyperbaric Medicine, Faculty of Medicine, Canakkale Onsekiz Mart University, Canakkale, Turkey*
sefikkorpinar@yahoo.com

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Abstract

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Intramuscular injections are one of the most common clinical procedures. The objectives of this case series are to analyse the role, timing and efficacy of hyperbaric oxygen treatment (HBOT) in the management of Nicolau syndrome (NS), an extremely rare complication of this common intervention. Clinical, demographic, laboratory and microbiological data extraction were performed through retrospective analysis of the medical records of all patients with NS who were referred for HBOT over a 10-year period with wounds, ischaemia, infection or necrosis at the injection site following drug injection; four patients with NS were included. All injections were made via the intramuscular route; three adult cases followed a non-steroidal anti-inflammatory drug, diclofenac sodium and one in a child followed penicillin injection. The time between diagnosis/injection and HBOT ranged from five to 33 days. NS can develop despite all preventive measures based on injection technique guidelines. HBOT appeared beneficial to healing of NS when administered with other therapeutic approaches. Due to the missing pieces of the puzzle in pathogenesis, NS is rarely completely reversible; keeping the awareness high for undesirable complications stands out as the most effective approach.

Introduction

Although it is not among the ancient symbols of the medical profession, such as the Caduceus or the staff entwined with serpent symbol that is known as the "*Rod of Asclepius*", the syringe is one of the most widely used devices in interventional medicine and everyday practice. At least 16.7 billion injections are estimated to be administered worldwide every year, the vast majority for curative care.[1] Hyperbaric medicine practitioners are unlikely to be involved in the management of complications due to these injections with a rare exception.[2–4] Nicolau syndrome (NS) was described originally as iatrogenic cutaneous necrosis following intramuscular injection of bismuth salts for the treatment of syphilis. This new clinical entity was described first in 1924 as "*embolia cutis medicamentosa*"; and was highlighted as early-stage livedoid dermatitis and subsequent gluteal gangrene a year later.[5,6] The objectives of this case series were to assess the role, timing and apparent efficacy of hyperbaric oxygen treatment (HBOT) in the management of NS, an extremely rare complication of intramuscular injection.

Methods

Approval was obtained from the Clinical Research Ethics Committee of Canakkale Onsekiz Mart University (2021/03, 03.03.2021) for a retrospective analysis of the medical records of all patients with NS who were referred to the Med-Ok Hyperbaric Oxygen Therapy Centre for HBOT between 1 January 2006 and 30 June 2016 with wounds, ischaemia, infection or necrosis at a drug injection site. The clinical data were reviewed for patient demographic characteristics (age, sex and comorbidities), body mass index (BMI), administered pharmacological agent, administration route, period and frequency, microbiologic evaluation, medical treatment received before HBOT (nature, duration), surgical intervention, HBOT received (number and duration of sessions), interval between onset of symptoms and HBOT and final clinical outcome based on laboratory, radiologic and/or clinical evaluations performed by the referring department.

Prior to HBOT, all patients were evaluated for contraindications such as the presence of untreated pneumothorax, radiologically indicated lung bullae or blebs,

pregnancy, severe emphysema and chronic obstructive pulmonary disease (COPD) assessed by pulmonary function tests, uncontrolled seizure disorders and cardiovascular instability. HBOT was administered in a multiplace hyperbaric chamber once or twice daily, five or six times per week, depending on the severity of the clinical findings. The treatment pressure was 253 kPa and each session consisted of three 25-minute oxygen periods with five-minute air-breaks to reduce the risk of oxygen toxicity. The decision when to terminate HBOT was made by the referring department.

Results

Over a 10-year period, four patients (one male, three female – one a child) were referred (Table 1). The injection site was dorsogluteal in two cases, ventrogluteal and vastus lateralis in one case each. All three adult cases occurred following administration of diclofenac sodium. Low back pain secondary to lumbar discopathy-spondylolisthesis and postoperative shoulder pain were the indications for intramuscular diclofenac administration in two and one patients, respectively. Benzathine penicillin was given intramuscularly for an upper respiratory tract infection in the child. None of the adult patients had a history of smoking. Non-insulin dependent diabetes mellitus was among the comorbidities in one patient, while arterial hypertension was present in two. All four patients had surgical interventions prior to HBOT referral; debridement in the three adults and thigh, leg and foot fasciotomies with dual incisions in the child. The time between diagnosis/injection and HBOT ranged from five to 33 days (Table 1).

In microbiological analyses of deep tissue samples taken from the wounds, methicillin-sensitive *Staphylococcus aureus*, *Escherichia coli* and *Pseudomonas spp* respectively were detected in the three adult cases, whilst there was no growth from the child's wounds. An appropriate antimicrobial regimen was chosen in all patients based on microbial sensitivity results and the recommendations of infectious disease consultants. Although the tissue samples were culture-negative, the child received empiric broad-spectrum antibiotic treatment.

The patients had been referred for HBOT for the presence of necrotising soft tissue infection with deterioration or delay in wound healing despite proper wound care, or after refusal

Table 1
The clinical characteristics and course of four patients with Nicolau syndrome who received hyperbaric oxygen treatment (HBOT); F – female; M – male

Patient	1	2	3	4
Gender/age (years)	M/66	F/48	F/76	F/3
Body mass index ($kg \cdot m^{-2}$)	26.1	34.6	30.5	16.0
Injection side/site	Right/dorsogluteal	Left/ventrogluteal	Right/dorsogluteal	Left/vastus lateralis
Drug administered	Diclofenac Na	Diclofenac Na	Diclofenac Na	Benzathine penicillin
Number of injections	1	Multiple	1	1
Comorbidities	Diabetes mellitus	Hypertension	Hypertension	No comorbidities
Microbiology	*Staph. aureus methicillin-sensitive*	*Escherichia coli*	*Pseudomonas spp*	No growth
Treatments prior to HBOT	Debridement	Debridement	Debridement	Heparin, pentoxifylline, fasciotomy
Time from injection/ diagnosis to HBOT (days)	7	32	33	5
Number of HBOT	25	40	28	13
Final outcome	Complete wound healing (before planned HBOT sessions completed)	Complete wound healing (before planned HBOT sessions completed)	Complete wound healing (+ graft reconstruction before planned HBOT sessions completed)	Complete wound healing (+ graft reconstruction in fasciotomy areas without limb loss

Figure 1
Images of the injection site in the left gluteal region of a patient with Nicolau syndrome and evolution of the lesion over three months; A) appearance of the 8 x 5 x 2 cm wound prior to HBOT; B) marked granulation tissue formation in fifth week; C) eighth week; D) third month follow-up

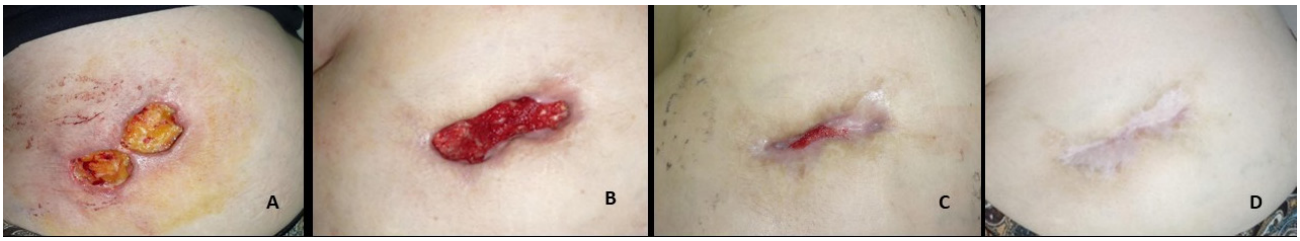
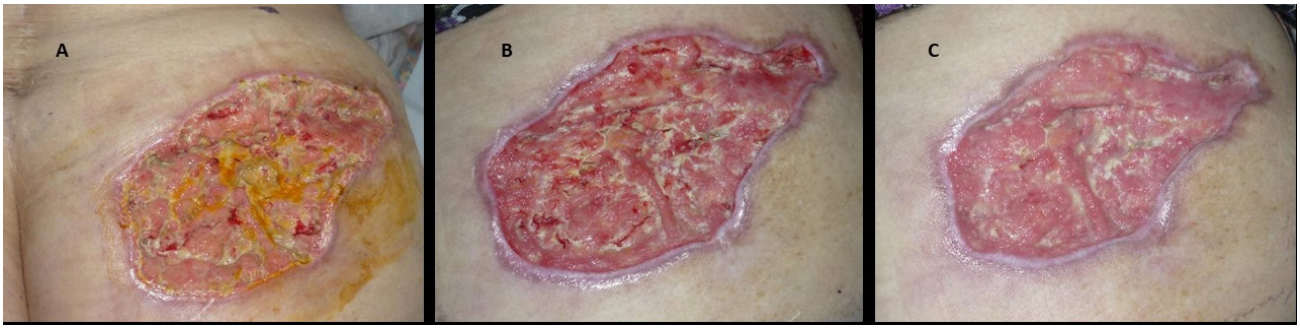


Figure 2
A) Appearance of the injection site of a patient with Nicolau syndrome on presentation to hospital; B) progression of wound (17 x 12 x 1.5 cm) during HBOT at second week; and C) fourth week of treatment; the wound demonstrates good granulation tissue without signs of infection



of further surgical intervention in order to increase oxygen concentration in the affected tissue as an adjuvant to heparin, pentoxifylline and fasciotomy respectively.

The average number of HBOT sessions was 26 (range 5–40), the treatments being well tolerated by all four patients. The three adults received daily wound dressings along with HBOT and all were followed up by the cardiovascular and plastic-aesthetic surgery departments. In two adult patients, complete wound healing without functional impairment was achieved before the planned HBOT sessions were completed (Figure 1), whilst the other two patients underwent skin graft reconstructions (Figure 2). None of the patients experienced limb loss. Post-HBOT physiotherapy rehabilitation was required in one patient who developed compartment syndrome.

Discussion

NS is an adverse dermatological reaction to the injection of a variety of drugs neither limited to bismuth suspensions nor to the intramuscular route.[2–15] Clinically, this rare syndrome is characterised by severe pain at the injection site with the immediate development of pallor and oedema. This is followed by erythematous maculae evolving within hours into livedoid reticular patches and plaques with dendritic extensions which culminate in cutaneous, subcutaneous, sometimes adipose and deep intramuscular necrosis. The necrotising lesion eventually sloughs, and the underlying ulcer evolves towards an atrophic pink scar devoid of adnexa over a few months.[7–14] However, not all cases progress in this predictable manner. NS has also been associated with fatal morbid complications such as widespread cutaneous necrosis, transient or permanent ischaemia of the ipsilateral limb, various neurological disorders, secondary infections, rhabdomyolysis, compartment syndrome and severe renal failure; it may result in medical malpractice claims.[8,9,11,13–15]

Local arterial vasospasm secondary to sympathetic stimulation, arterial embolism caused by the intra-arterial injection of microcrystals and ischaemia caused by compression following vascular or perivascular injection have all been suggested in its pathogenesis. Cytotoxic effects are also highlighted, depending on the composition of the drug, the injection site and individual skin sensitivity.[10–12] Diclofenac sodium may create vasospasm following inhibition of prostaglandin synthesis and cyclooxygenase inhibition.[10,16]

There is no standard treatment regimen. Positive results have been obtained with the use of sympathetic nerve block, heparinisation, arteriotomy and extraction of clot, calcium channel blockers, dipyridamole, trinitrine, pentoxifylline, corticosteroids and HBOT, suggesting that a vascular origin is the most realistic theory.[2–4,8,11] On the other hand, after this acute period and/or the limitation of

necrosis, various treatment regimens have been proposed recently for infection, tissue healing and reconstruction.[17] These algorithms do not include HBOT. In this context, the main inference of this small case series is that HBOT appears to have beneficial effects when combined with other treatments such as antibiotics and appropriate wound care after debridement of necrotic areas that are not progressing satisfactorily.

The rationale for HBOT in the acute phase is based on anti-hypoxic, anti-oedema effects and the mitigation of reperfusion injury. Therefore, it should be initiated as early as possible and administrated more frequently.[2–4] Following a longer interval between the incident injection and referral, HBOT may be of benefit through antibiotic and wound healing-accelerating effects, particularly in complicated cases with secondary infection as in three of the present cases. In the post-acute, early regenerative phase, granulation tissue fills the void caused by the necrosis (Figure 2) after debridement and drainage of abscesses, if any. Resolution of infection and granulation tissue formation may be impeded in the presence of comorbidities such as diabetes mellitus, peripheral vascular disease and obesity.

HBOT helps provide adequate oxygen for fibroblastic activity, leukocyte function, angiogenesis and wound healing in hypoperfused, hypoxic and infected tissues.[18] These benefits are particularly important when primary closure is not appropriate and/or the planned reconstructive surgery is declined by the patient. The long, variable referral interval (up to 33 days in this series) also suggests that hyperbaric physicians may not encounter the early clinical characteristics. Thus, one should be familiar with the course of the syndrome and its unpredictable progression. Moreover, in such presentations, a standard algorithm should not be expected, since therapeutic measures should be based on the clinical status of the individual patient.

Thicker subcutaneous adipose tissue makes it more difficult to reach the target muscular tissue.[19] High BMI, female gender, the use of the dorsogluteal site and diclofenac sodium predominance were consistent with the literature in this series.

Prevention should be the cornerstone of care. Choosing the appropriate needle according to the patient's weight to avoid the risks of subcutaneous injection, preferring different anatomical sites for repeated injections, use of the Z-track method of injection and reassessing the site for any signs of complication after injection are well known and widely practiced measures to avoid this iatrogenic complication.[7,8,10,11,19,20] However, it is unclear whether or not they prevent NS. Particularly in the outpatient setting, where intramuscular administrations are more frequently preferred, healthcare personnel as well as the patient or accompanying adult should be warned about reporting complications without delay and advised how to assess the site.

Conclusions

NS can develop despite adherence to all preventive measures based on injection technique guidelines. As seen in these four patients, HBOT may have beneficial effects in minimising damage when administered with other therapeutic approaches, not only in the acute phase but also later, particularly in cases with compartment syndrome, secondary infection, surgical intervention refusal and/or impaired wound healing. However, due to the missing pieces of the puzzle in the pathogenesis of NS it is rarely completely reversible. It remains unclear how the various approaches to treatment affect the natural course of NS. Maintaining a high awareness for undesirable complications is the most effective approach until the missing pieces are in place.

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Conflicts of interest and funding: nil

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Obituary

Professor Alf O Brubakk MD, PhD

Alf Brubakk was born in 1941 in Bergen, Norway. His doctorate degree was awarded by Justus Liebig University Giessen in Hessen, Germany, followed by his obligatory internship on a small island in West Norway. Divers are exposed to intermittent hyperoxia and pressure reductions, which evoke the production of radical oxygen species and microparticles that are central to many mechanisms involved in several severe human diseases. Alf believed that diving could serve as an important model of disease and allow the study of these effects on healthy individuals.

With only two Norwegian medical faculties in Bergen and Oslo, Alf was asked to establish one in Trondheim, in cooperation with the Norwegian Technical High School. In 1970, in collaboration with Rune Aaslid, a mathematical model of the cardiovascular system was constructed that could be used clinically along with a pulsed echo Doppler flowmeter to record blood flow velocity in the aorta and heart. Jarle Holen's work led to ultrasound measurements being possible to obtain intracardiac pressure non-invasively, thus avoiding heart catheterization. By 1978, Alf had submitted to NTNU Trondheim his doctoral thesis "*Methods for studying flow dynamics in the left ventricle and the aorta in man; use of a simulation model and ultrasound.*" At the beginning of offshore oil exploration in the North Sea, Bård Holand, an experienced commercial diving friend, suggested ultrasound's usefulness in studying decompression in diving which led to several ultrasound studies of experimental dives to 500 metres of seawater at the Norwegian Underwater Institute in Bergen.

Alf and colleagues were the first to show that physical exercise could significantly reduce bubble formation and hence reduce the risk of injury. Over his career he published 153 scientific papers, co-edited Bennett and Elliott's 5th edition of The Physiology and Medicine of Diving, and in the last 20 years alone supervised 15 Masters and 10 PhD students. His two major influencers were Professor Jens Glad Balchen, who believed in the importance of having a basic idea to follow through to the end, regardless of opposition, and John Scott Haldane, the first environmental physiologist who showed the value of using basic physiology to understand man's response to his environment.

With Bård Holand Alf conducted extreme environment survival courses in Svalbard over a 20-year period. He served in various capacities on the Diving Medical Advisory Committee, European Underwater Baromedical Society, European Diving Technology Committee, and received the Undersea and Hyperbaric Medical Society's Behnke award twice.

Stephen Thom wrote "*I first knew Alf from his scientific presentations as a disciplined and sometimes stern Norwegian but really got to know him as a fun-loving person, if with a dry sense of humor. It has been a great privilege to spend time with Alf and our last collaboration on a Comprehensive Physiology review of saturation diving*." Michael Gernhardt mentioned "*Alf was a smart researcher with whom I enjoyed a productive collaboration on biochemical countermeasures for the reduction of DCS risk on spacewalks from the International Space Station*." Hans Örnhagen relayed "*I have known Alf for a long time. He participated with his special knowledge of bubbles in our Swedish hydrogen experiments. Alf has helped make the world wiser in terms of diving medicine*."

Alf's favorite pastimes were skiing, scuba diving, running/cardiac exercise and the occasional beer with his friends. Our adventures included dive sites on the Great Barrier Reef, Corsica, San Clemente Island, Stokkøya, Svalbard, and the Red Sea. Alf passed away on 5th April 2022.

Alf was survived by his wife Greta Bolstad (since 1980) who also passed peacefully on 18th May 2022, sister Ann Mari, children Kirsten, Berit, Katrin and Axel, and seven grandchildren. On behalf of the Brubakk family, Katrin shared "*Our father was an engaged and funny man, dedicated and creative, always thinking out of the box. We will miss him*."

Dr Michael A Lang
UC San Diego – Emergency Medicine
Center of Excellence in Diving

Acknowledgement

The editor gratefully acknowledges permission to reproduce this obituary from Undersea and Hyperbaric Medicine Journal.

Notices and news

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

SPUMS President's message

Neil Banham

The past year has seen many changes, with a gradual return towards 'normality' despite high numbers of COVID-19 cases, which fortunately has become less virulent with a high percentage of the Australasian population being fully vaccinated.

Despite this, because of the ongoing uncertainty regarding international travel some months ago when a decision had to be made, it was decided that the safest and lowest risk to SPUMS financially was to hold our Annual Scientific Meeting (ASM) virtually.

The theme was "*Take a deep breath – diving and hyperbaric respiratory physiology in 2022*". The ASM was convened from New Zealand by Greg van der Hulst and his team, to whom we are very grateful for organising. The ASM was successful, with 72 registrants and a varied and interesting range of presentations. Thanks to all presenters and registrants for making this ASM a success during difficult times.

In 2023 we are in the planning stages of holding an in-person ASM in Cairns, allowing participants to dive the Great Barrier Reef, in keeping with the traditional SPUMS conference format. The planned dates are 04–11 June. 2024 will hopefully see a return to an overseas venue, with Komodo, Indonesia being amongst the destinations being considered. If you are interested in convening the 2024 ASM or have another SPUMS suitable venue in mind, then please contact me.

An in-person diving and hyperbaric medicine conference – the HTNA 30th ASM, will be occurring in Hobart from 07–09 September 2022. This conference is supported by SPUMS and is usually of high quality, both academically and socially, this year should be no exception. SPUMS members are welcome to register, but it may be somewhat cold in Tasmania in September to consider diving. SPUMS member, Dr Richard Harris SC, OAM ("*Harry*") of the Thai Cave Rescue fame and Mr Chris Lemons whose story of survival was immortalised in the documentary "*Last Breath*" are the invited speakers. Tom Workman, the author of Hyperbaric Facility Safety – a practical guide, will speak on Ziplock chambers. The ANZHMG will also meet immediately prior to the conference, and any SPUMS member working in the field of diving and hyperbaric medicine is welcome to attend. As there are currently limits to registrant numbers due to COVID-19 restrictions, I suggest that you do not delay registering if you are keen to attend.

Our journal *Diving and Hyperbaric Medicine* (DHM) continues to publish high quality material, maintaining its position as the pre-eminent journal in the field of diving and hyperbaric medicine. Our Editor Professor Simon Mitchell reports a return to a normal volume of submissions during the last year, although the hard work of producing a high quality journal remains. Many thanks to Simon and his Editorial Assistant Nicky Telles for their efforts, as well as to the many reviewers and to the SPUMS members who have submitted to DHM. All previous issues of our journal (back to the first Newsletter in 1971) are now available via our website, thanks to the generous funding of the Australasian Diving Safety Foundation and the hard work of our Web Assistant Nicky Telles. Only SPUMS members however, can access issues from the last 12 months.

Our Webmaster Xavier Vrijdag and Nicky have been working hard on the development of a new SPUMS website which will hopefully be operational by the end of the year. The website will be on a new platform with many more features, including the ability to pay your subscription on a recurring basis automatically. There will even be a new logo which is in the final stages of refinement.

The ANZHMG Introductory Course in Diving and Hyperbaric Medicine will be held in 2022 in Fremantle (13–24 June) following a COVID-19 induced postponement in February/March. Dr Ian Gawthrope, Course Convenor, has successfully had this course accredited by ANZCA towards the ANZCA Diploma of Advanced Diving and Hyperbaric Medicine. The planned course dates for 2023 is tentatively 20 February–03 March.

The Royal Australian Navy Medical Officers' Underwater Medicine (MOUM) Course was successfully held in March 2022 and will be held again from 17–28 October 2022 and 13–24 March 2023 at HMAS Penguin, Sydney. Details of both courses are available on the SPUMS web site: https://www.spums.org.au/content/approved-courses-doctors

Finally, I would like to thank all my ExCom team for their hard work and ongoing support in these difficult times, and to members for staying engaged with SPUMS. We

need you all to encourage others to join or maintain their SPUMS membership, such that our society can continue our stated purpose: "*To 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 the Society annually at a scientific conference*".

Neil Banham
SPUMS President

The Australian and New Zealand Hyperbaric Medicine Group 2022

Introductory Course in Diving and Hyperbaric Medicine

Dates: 13–24 June 2022
Venue: Hougoumont Hotel, Fremantle, Western Australia
Cost: AUD$2,700.00 (inclusive of GST) for two weeks

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

The SPUMS website is at

https://spums.org.au/

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

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

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

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

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

Examination dates for 2022
Written section:

Short answer questions	10 August 2022
Viva examination	14 September 2022
Withdrawal date	26 July 2022

Accreditation

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

Transition to new qualification

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

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

SPUMS Facebook page

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

Government of **Western Australia**
South Metropolitan Health Service
Fiona Stanley Fremantle Hospitals Group

The Australian and New Zealand Hyperbaric Medicine Group

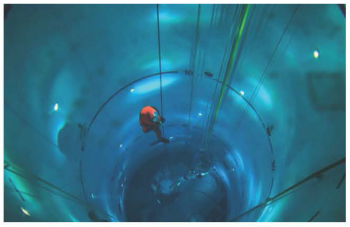
Introductory Course in Diving and Hyperbaric Medicine

Dates: 20 Feb – 03 Mar 2023
Venue: Hougoumont Hotel, Fremantle, Western Australia
Cost: AUD 2,700 for 2 weeks

The course is for medical graduates with an interest in diving and hyperbaric medicine. It is designed both for those wishing to pursue a career in this specialised field and those whose primary interest lies in related areas. The course will be held in Fremantle with excursions to the Fiona Stanley Hyperbaric Medicine Unit, HMAS Stirling and the local Royal Flying Doctor base. The course is accredited with the South Pacific Underwater Medicine Society and ANZCA for the Diploma of Diving and Hyperbaric Medicine.

The Course content includes:

- History of diving medicine and hyperbaric oxygen
- 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 Ovens, **Course Administrator**
Phone: +61-(0)8-6152-5222
Fax: +61-(0)8-6152-4943
E-mail: fsh.hyperbaric@health.wa.gov.au
Accommodation information can be provided on request

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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 President's message

Jean-Eric Blatteau

On the French Mediterranean coast, every year from May and June onwards, hyperbaric centres see their activity surging with the treatment of many diving accidents.

This year is no exception, with the occurrence of a large number of cases of neurological spinal cord decompression sickness. These divers, very often, have not made any procedural errors and do not understand what is happening to them. Despite hyperbaric treatment, it turns out that about 30% of these accidents will have sequelae of varying severity after hyperbaric treatment. The most surprising thing is that a certain number of these accidents continue to worsen for 12 to 24 hours even if treated with hyperbaric oxygen in a timely manner, which testifies to the activation of biological cascades which are not stopped despite the elimination of bubbles by recompression.

For more than 20 years, different management modalities with initial recompression tables at 4 or 2.8 ATA, with the use of different heliox or oxygen mixtures and different drug treatments, have been tried. The means of evacuation were also specifically regulated to reduce recompression times. The continued reporting of these refractory or worsening cases is a tell-tale sign that there is still room for scientific advance and improvement of the treatment approach. The level of evidence in this field can and must still be improved, and it appears that the analysis of retrospective data is an important source of information – as well as exchanges and collaborations between different centres.

The next EUBS Congress in Prague from 31 August to 3 September will be an excellent opportunity to present your experience, explore possible significant advances in this field and to benefit from the experience of all hyperbaric centres involved in the management of diving accidents.

Of course, many other topics will also be discussed, and we are very happy to finally be able to meet and exchange ideas face to face, if the pandemic and geopolitical situation allows it.

It will also be an opportunity to pay tribute to all our colleagues and friends who have recently passed away and who have contributed greatly to our field. See you all in Prague!

Jean-Eric Blatteau
EUBS President

EUBS Notices and news

FINALLY HAPPENING: EUBS2022 Scientific Meeting on Diving and Hyperbaric Medicine

After two years of postponement, our 46th Annual Scientific Meeting (originally scheduled for 2020) will held as a 'in person' event, and we welcome you to our meeting in Prague, Czech Republic, from 31 August to 3 September.

All details for the meeting are available on the website www.eubs2020.com – yes, the name of the website has not changed, paying tribute to the patience we've all had. We hope you are as excited as we are and will be able to join us for the first 'post-COVID' scientific meeting.

Even though the 'early bird' registration rates are no longer available, there is still time for you to register for EUBS2022, so go ahead, book your flight/train/hotel and register now. Your friends will be there too.

Ukraine war position statement on EUBS website

While science should be and remain apolitical and non-judgmental of other peoples' convictions and beliefs, we cannot stand by idly in the face of inhuman (as in: non-respectful of human life, dignity and right to autonomy) events happening in the world. There has and has always been war, conflict, terror, famine, injustice in many places around us. All of these are worthy of consideration and protest. However, we chose, like many organisations devoted to science and medicine, to specifically let our voice be heard in the case of the Russian-Ukrainian conflict. On our website, we have placed the Ukrainian flag and have coloured our header in blue and yellow, and have placed the text:
"*As scientists, devoted to human wellbeing, we abhor the use of military violence to resolve any conflict, be it political or economic, between free independent states. The Russian attack on Ukraine is a flagrant and utterly unacceptable act*

of violence and we urge all political and economical leaders worldwide to react strongly with any diplomatic or economic measures necessary to stop this senseless invasion. We are calling upon the Russian Federation and its leaders to end this aggression immediately, and express our undivided solidarity with the people of Ukraine."

While we hope that by the time of publishing of this issue of Diving and Hyperbaric Medicine (DHM), state leaders from the Russian Federation have come to their senses and have stopped military actions in order to pursue a diplomatic – humane – solution to the issues perceived, we will continue to have this statement on our website until this happens. At the time of writing, the events show (once again) that resorting to the use of military force leads to deviation from moral behaviour and causes horrific suffering and wounds, both physical and psychological that will never heal.

EUBS elections – Member-at-Large

Around the time of publication of this issue of DHM, the election process for the 2022 ExCom Member-at-Large of EUBS will have been started.

We will be saying goodbye to Dr Gerardo 'Dino' Bosco (Padua, Italy) as Member-at-Large 2019. The ExCom extends their thanks to Dino for his work, and hopes to be able to continue counting on his support and help, despite his workload as President of SIMSI.

Candidates for the position of Member-at-Large 2022 will present themselves on the EUBS website with a picture and short CV, and by the time this journal issue is published you will have received an internet ballot by email allowing you to cast your vote.

If you have not received the email yet by the end of June, please notify us at secretary@eubs.org. As the system works via email, it is possible the message ended up in your spam folder. There may be other reasons but usually, we are able to solve them.

Losing friends is never easy

In the past quarter, we unfortunately had to say goodbye and farewell to two friends and colleagues.

On 05 April 2022, Professor Alf Ottar Brubakk (24 January 1941) passed away peacefully.

A long-time member and former President of EUBS (2006–2009), Alf has marked many of our lives with his intellect, wit, and honesty – always speaking his mind, inspiring many of us to try and do as well as him in science, and having great fun together 'after work'. His contributions to the knowledge of diving decompression physiology place him in the gallery of the Greats. He leaves a great legacy as well as a void in our hearts.

On 21 May 2022, Dr Cecilia J Roberts, former President of SAUHMA, co-organiser of TRICON2018 and a good friend to many of us here at EUBS, tragically died in a car accident in South Africa at the age of 43. Cecilia will be remembered as a loveable, intelligent, funny companion, deeply religious and living up to her faith. She will be sorely missed.

EUBS has extended formal condolences to her family, and friends at SAUHMA, DAN South Africa.

Website and social media

As always, please visit the EUBS Website (www.eubs.org) for the latest news and updates.

On the 'Research Page' (http://www.eubs.org/?page_id=284) you will be able to find information on planned and recruiting clinical trials, including one on the use of HBOT for COVID-19.

While we value the membership contributions of all our members (after all, members are what constitutes our Society), EUBS ExCom would specifically like to thank our Corporate Members for their support to the Society. You can find their names, logos, and contact information on the Corporate Members page under menu item "*The Society*". Please follow our Facebook, Twitter and Instagram account! While we will continue to use our "*EUBS Website News*" email messages as a way to communicate important information directly to our EUBS members, Twitter and Instagram will be used to keep both members and non-members updated and interested in our Society.

Here are the links to bookmark and follow:
Facebook: https://www.facebook.com/European-Underwater-and-Baromedical-Society-283981285037017/
Twitter: @eubsofficial
Instagram: @eubsofficial

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

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

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

SIMSI Società Italiana di Medicina Subacquea ed Iperbarica

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

Date: 02–04 December 2022, Padua

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

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

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

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

Gerardo Bosco and Vincenzo Zanon

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

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 29 years. SHF is targeting on an international audience with courses worldwide.

We are happy that the world has reopened after the COVID-19 pandemic and we can announce courses around the world again. Below the schedule of upcoming SHF-courses in 2022.

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

2022

06–07 October	In-depth course Psyche under pressure (level 2d) Loosdrecht (NL)
05–12 November	In-depth course Nightmares for the diving doc (level 2d) Bali, Indonesia
12–19 November	In-depth course Nightmares for the diving doc (level 2d) Bali, Indonesia
19–26 November	In-depth course Diving medicine (level 2d) Bali, Indonesia
In planning	Decompression,recompression and HBOT (level 2d), tbd In-depth course Diving after (long) Covid (level 2d), tbd
On request	Internship HBOt (level 2d certification), NL/Belgium

The course calendar will be supplemented regularly. For the latest information see: https://www.scotthaldane.nl/en/.

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/

Diving and Hyperbaric Medicine: Instructions for authors (summary)

(updated August 2021)

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

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

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

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

Types of articles

DHM welcomes contributions of the following types:

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

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

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

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

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

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

Formatting of manuscripts

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

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

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

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

DIVER EMERGENCY SERVICES PHONE NUMBERS

AUSTRALIA – DAN
1800-088200 (in Australia toll free)
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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.
