Diving and Hyperbaric Medicine

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SPUMS

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EUBS

Anaesthesia in the Thailand cave rescue

Diving injuries in Japan

Medical conditions in Australian scuba fatalities

Diver characteristics in Australian scuba fatalities

Pupillometry to measure gas narcosis?

Anxiety trait effect on scuba skill performance

Diving with diabetes

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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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Editorial

Diving and hyperbaric medicine in the SARS-CoV-2 pandemic

The world is living through a tragic and historic event. It is difficult to overstate (or even appreciate) the scale of medical, social and economic upheaval wrought by the SARS-CoV-2 (Covid-19) pandemic, and few would expect the fabric of life to return to normal any time soon.

The fields of diving medicine and hyperbaric medicine stand to be impacted in multiple ways, not least because hyperbaric oxygen treatment (HBOT) appeals as an intuitively obvious means of improving oxygenation in a disease process where hypoxia is a prominent and sometimes fatal feature. There would be few HBOT providers who have not fielded questions about providing treatment for Covid-19 patients.

There is little doubt that hypoxia in a critically ill Covid-19 patient could be improved during HBOT. However, this would only last for the duration of the treatment, and it is unknown whether any other benefit would accrue. HBOT is not known to have specific antiviral effects. Nevertheless, as practitioners in this field are aware, there are potentially beneficial immune-modulatory and anti-inflammatory actions elucidated and reported in other indications.[1] Their net effect on the natural history of Covid-19 is unknown.

There are predictable logistic difficulties in providing HBOT to Covid-19 patients, including patient transfer and access, staff protection, infection control, and (depending on patient selection) the challenges of caring for very sick patients in a hyperbaric environment. There are also potential risks. Any hyperbaric dose of oxygen will promote pulmonary oxygen toxicity. Some patients may have existing pulmonary oxygen toxicity due to prolonged high-fraction normobaric oxygen administration, and their vulnerability to exacerbation by HBOT is unknown. There is also the likelihood that Covid-19 may enhance risk of pulmonary barotrauma. In a series of 202 Covid-19 patients intubated for ventilatory support 5.9% developed pneumothorax;[2] an unusually high number (even in pulmonary pathology) suggesting that the disease promotes gas trapping (substantially confirmed by computed tomography scans)[3] or structural lung damage or both. There is also the distressing potential for patients to become 'oxygen-trapped' toward the end of HBOT sessions if developing worse oxygenation than pre-treatment levels during decompression to surface pressure.

The crucial balance between these potential benefits and risks is simply not informed by adequate evidence at this point. The Undersea and Hyperbaric Medical Society is taking an appropriately cautious position on this matter. They state that "*there is insufficient evidence to endorse the use of routine adjunctive HBO_2 for COVID-19 patients outside the context of an IRB-approved clinical trial*".[4] It is gratifying that multiple groups have taken up the implied challenge of answering the relevant questions with controlled studies.[5–8] Study primary end points include: incidence of intubation,[5] mortality,[6] effect on oxygen requirement,[7] and PO_2/FiO_2 ratios and immunological responses.[8]

One consequence of the pandemic for all clinical hyperbaric units is the challenge of maintaining a service for patients with the usual indications for HBOT amidst lock-downs, patient reluctance to interface with medical services for fear of infection, and inevitable uncertainties around patient Covid-19 status even in the absence of symptoms. Many patients at high risk of poor outcomes if infected (elderly or co-morbid patients) are treated at hyperbaric units, and it follows that high levels of attention to social distancing, staff and patient personal protective equipment, and equipment and environmental hygiene must be maintained. These matters can be particularly challenging in practices utilising multi-place chambers and an attempt to provide relevant guidance has been promulgated by the European Committee for Hyperbaric Medicine.[9]

For those more focused on the diving medicine side of practice the effects of this pandemic may reverberate for longer. In particular, there are obvious but (at this point) poorly understood implications for future fitness for diving after Covid-19 infection.

Experience with persisting lung changes following the original SARS-CoV-1 epidemic in 2003 have raised fears that the risk of pulmonary barotrauma may be heightened in Covid-19 survivors. The mid to long-term natural history of lung changes caused by Covid-19 are not yet characterised, and this uncertainty has encouraged conservatism, at least for the time being. For example, a guideline on diving after Covid-19 pulmonary infection released by the Belgian Society for Diving and Hyperbaric Medicine states that "*a diver who has been hospitalised with or because of pulmonary symptoms in relation to COVID-19, should, after a three-month waiting period (with no diving), undergo complete pulmonary function testing as well as a high resolution CT scan of the lungs*".[10] It further states that the "*CT scan should show a return to normal before resuming diving*". Another thoughtful and highly structured guideline has been promulgated by the University of California San Diego group.[11]

Advocacy for considering detailed radiological investigation of Covid-19 affected divers or diving candidates before diving seems reasonable in the present uncertain circumstances, but in the likely absence of a baseline CT scan, interpreting "*normal*" may be problematic. For example, Covid-19 infection seems to promote gas trapping detectable by CT,[3] but gas trapping can also be seen on CT in subjects who are in perfect health with normal lung function.[12] This is likely to become a challenging issue for our field.

The prospect of mid to long-term pulmonary effects also signals other possible problems for diving fitness. Damaged lungs may become less efficient at filtering the venous gas emboli (VGE) that are commonly formed after surfacing from compressed gas dives, raising concerns about an increased risk of those forms of decompression sickness associated with arterialisation of VGE. There is also concern that Covid-19 (like SARS-CoV-1 in 2003) may leave survivors with significantly reduced exercise capacity. This was seen in patients whose lungs appeared largely recovered, and may be multifactorial in origin.[13]

These considerations, along with other potential complications, suggest that it would be wise for any diver or diving candidate who has suffered Covid-19, but particularly cases with obvious pulmonary, cardiac or neurological symptoms to be reviewed by a diving physician, investigated appropriately and counselled about risk prior to diving, or advised against diving if risk is considered excessive. With time, experience and more research, consensus on an evidence-informed pathway for pragmatic management of these consultations will emerge.

These are challenging times for our discipline. Many hyperbaric practitioners in large centres are also qualified in front-line hospital-based disciplines like anaesthesiology or emergency medicine where they will have been distracted from hyperbaric practice, but will have made significant contributions to caring for patients under trying and hazardous conditions. Indeed, thanks and respect are due to front-line medical staff world-wide for a response that has engendered a profound appreciation of the medical profession. Back in the world of hyperbaric medicine the quiet, professional and ethical initiatives currently underway to scientifically define the role (if any) for HBOT in treating Covid-19 are also deeply appreciated. An answer may not come until the current pandemic is in decline, but there will still be patients to treat, and we can be sure that it will happen again.

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Covid-19; Hyperbaric oxygen treatment; Fitness for diving; Pulmonary barotrauma; Decompression sickness

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Front cover: Dr Craig Challen (left) and Dr Richard Harris (right) after emerging from the cave on one of the Thailand cave rescue days. Image taken by Australian Federal Police.

Original articles

Recreational diving-related injury insurance claims among Divers Alert Network Japan members: Retrospective analysis of 321 cases from 2010 to 2014

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Key words
Decompression illness; Decompression sickness; Diving incidents; Epidemiology; Injuries; Risk; Trauma

Abstract
(Kojima Y, Kojima A, Niizeki Y, Yagishita K. Recreational diving-related injury insurance claims among Divers Alert Network Japan members: Retrospective analysis of 321 cases from 2010 to 2014. Diving and Hyperbaric Medicine. 2020 June 30;50(2):92–97. doi: 10.28920/dhm50.2.92-97. PMID: 32557409.)
Introduction: Monitoring trends in diving-related injuries enables implementation of effective safety measures. Divers Alert Network Japan (DAN Japan) membership includes insurance covering recreational diving-related injuries and fatalities. Use of claim data provides both a known denominator and demographic data about injured members.
Methods: The study analysed 325 insurance claims reported to DAN Japan from 2010 to 2014. Four fatalities were excluded, leaving 321 claims for analysis. Claimants were divided into three age groups: young adults (< 40 years); middle-aged (40–59 years) and older adults (≥ 60 years). The total injury claims rate (ICR), decompression illness (DCI) rate (DCR) and trauma rate (TCR) were calculated. Differences between the sexes within each age group were analysed.
Results: The total number of DAN Japan member-years in the period was 80,617, with a mean age of 45 years. Claims were made by 153 males and 168 females with a mean and median age of 46 years. Trauma was the most frequent reason for a claim (113 cases, 35%), followed by DCI (109 cases, 34%). The ICR (per 10^4 member-years) was 39.8 (95% confidence interval 35.5 to 44.2) and the TCR was 14.0 (11.4 to 16.6). For every age group, the ICR and TCR were significantly higher for females than males. The DCR was 13.5 (11.0 to 16.1) and did not significantly differ between the sexes.
Conclusions: The incidence of trauma-related diving injuries exceeds that of claims related to DCI. Females appear to have a higher risk of injury than the general diving population.

Introduction

Recreational diving is popular in Japan, with more than 40,000 new divers certified each year.[1] The mean age of Japanese divers is unknown; however, the mean age of Divers Alert Network Japan (DAN Japan) members has increased from 38 years in 2004 to 47 years in 2014.[2] This increase in the mean age of DAN Japan members may bring an increase in diving-related injuries. It is essential to monitor the trends in diving-related injuries to plan effective interventions for optimising diving safety. Diving-related fatalities have been investigated in various areas,[3–6] whilst the frequency of decompression illness (DCI) (a dive-related injury) has been estimated by many researchers.[6–9] A recent study evaluated both diving-related and non-diving-related injuries recorded by a recreational diving business;[9] however, there are limited data available on diving-related injuries other than DCI.

As diving-related injuries range from mild to severe, it is difficult to accurately determine the incidence. Membership with DAN Japan automatically includes basic insurance, covering losses from recreational diving-related injuries and fatalities. Questionnaire surveys generally tend to identify cases involving milder injuries rather than the injuries involved in treated cases and insurance claims. The present study focused on the relatively severe diving-related injuries reported in DAN Japan insurance claims, as it is important to understand and to prevent such injuries. Use of claims data also provides a known denominator, which enables better statistical analysis.

Methods

The study protocol was approved by the Institutional Review Board of DAN Japan and the Japan Marine Recreation Association (approval number: R01363). The study was conducted in accordance with the principles of the Declaration of Helsinki. The present study retrospectively reviewed the insurance claims submitted to DAN Japan from 01 January 2010 to 31 December 2014. The claims data were provided by DAN Japan in a single Excel™ database, under the condition that each claim report was de-identified and given a case identification number. The only demographic data were the sex and age of each claimant. The collected data included the injury date, diving location, diagnosis and course of treatment. Some claims reported the occurrence of injuries at two or more body locations at the one time. However, not all claims accurately reported every diagnosis; for example, 'contusions of the upper body' indicates that injuries occurred at more than one site, but does not identify the actual injury sites. In the present study, one main diagnosis was considered for each claim. For example, for a claim in which the diver incurred a dental injury after hitting their head on the gas cylinder during boat-diving entry, the case was counted as a dental injury rather than a head contusion.

Each case was classified as either a diving disorder or a non-diving disorder. Diving disorders were defined as DCI, barotrauma, and hazardous marine life injury. Non-diving disorders included other injuries such as fractures and sprains that occurred at any time during the diving trip, including on the diving boat; non-diving disorders were further classified into two groups: trauma and others. Cases that could not be categorised into a specific group due to incomplete diagnostic information were classified as unknown.

The total number of DAN Japan member-years during the study period was calculated as the sum of the total number of members at the end of each of the five years. The total injury claims rate (ICR), DCI claims rate (DCR), and trauma claims rate (TCR) were calculated. Prevalence was calculated as the number of claims divided by the number of member-years and was converted to the number of injuries per 10^4 member-years. This means that, if one diver was continuously a DAN Japan member from 2010 to 2014 and was injured three times but only made two insurance claims, she/he would be counted as having two injury cases and contribute to five member-years in the dataset. The renewal rate of DAN Japan members during the assessed period was around 90%. However, as all personal information other than sex and age had been deleted from the database, it was not possible to identify whether multiple claims had been made by the same member. For the DCI claims, the time between the onset of symptoms and initiation of recompression therapy was recorded.

STATISTICAL ANALYSIS

Divers were clustered into three age groups: young adults (< 40 years), middle-aged (40–59 years), and older adults (≥ 60 years). The ICR and 95% confidence interval (CI) were calculated for each age group and compared with the middle-aged group, which comprised 57.6% of the DAN Japan member-years during the study period. The rate ratio (RR) of the ICR was calculated as the ratio of the ICR of each age group with respect to the middle-aged group. The rate difference (RD) of the ICR was calculated as the difference between the ICR for each age group and the middle-aged group. The same RR and RD calculations were made for the DCR and TCR. In addition, a cross-tabulation table of sex with respect to the total number of injuries, DCI, and trauma was created. The chi-squared test was used to assess the differences between males and females within each age group. The significance level for statistical analysis was set at $P < 0.05$. Data analyses were performed using IBM SPSS Statistics ver.23.0 (IBM Japan, Ltd.).

Results

The total number of DAN Japan member-years during the study period was 80,617. Males accounted for 59% of DAN Japan members, and the mean age was 45 years. There were 325 recreational diving-related injuries and fatalities reported to DAN Japan for insurance claims. After excluding four fatalities, 321 diving-related injuries were analysed. The diving-related injuries were incurred by 153 males and 168 females (mean and median age 46 years, range 20–77 years).

The diving location was in Japan in 236 cases and overseas in 85 cases. Many of the Japan-based injuries occurred in summer and autumn. In contrast, overseas injuries occurred evenly throughout the year (Figure 1). Table 1 shows the distribution of diving-related injuries. Diving disorders accounted for 168 cases (52%) of injuries, while non-diving disorders accounted for 132 cases (41%). Within the diving disorder category, DCI was the most common type of injury (109 cases, 34%). Among non-diving disorder categories, trauma was the most common type of injury (113 cases, 35%) and also the most common type of injury overall, followed by DCI.

Fracture and dislocation accounted for 43 cases of trauma (38%). Other common injuries reported in trauma cases were sprain, contusion, wound, ligamentous injury and dental injury (Table 2). The location of fractures and dislocations was the lower limb in 20 cases, the upper limb in 12, ribs in seven, spine in three, and unknown in two; one case involved two fractures that occurred at the same time and were dealt with as a single claim. The causes of fractures and dislocations varied; six cases were related to ladders, while eight were related to issues with gas cylinders.

Figure 1
Distribution of diving-related injuries by month and location

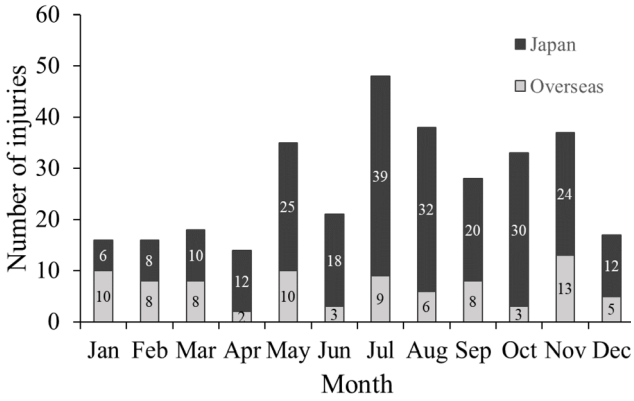


Figure 2
Distribution of the total injury claims rate by three age groups (young adults, middle-aged and older adults) and sex; *$P < 0.05$, females versus males

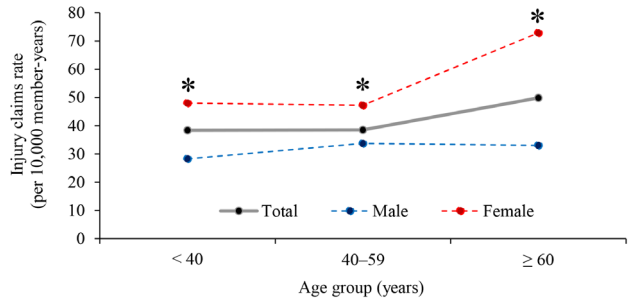


Table 1
Distribution of diving-related injuries by type of injury

Injury type	*n* (%)
All types	321 (100)
Diving disorders	168 (52)
Decompression illness	109 (34)
Barotrauma	39 (12)
Marine animal injuries	20 (6)
Non-diving disorders	132 (41)
Trauma	113 (35)
Other	19 (6)
Unknown	21 (7)

Table 2
Distribution of diving-related traumatic injuries by injury type

Injury type	*n* (%)
Fracture / dislocation	43 (38)
Sprain	14 (12)
Contusion	12 (11)
Open wound	11 (10)
Ligament injury	4 (4)
Dental injury	3 (3)
Head injury	2 (2)
Muscle injury	2 (2)
Other	6 (5)
Unknown	16 (14)

The ICR was 39.8 per 10^4 member-years (95% CI 35.5 to 44.2). The DCR was 13.5 per 10^4 member-years (95% CI 11.0 to 16.1). The TCR was 14.0 per 10^4 member-years (95% CI 11.4 to 16.6.). Table 3 shows the ICR, DCR, and TCR estimates for each age group and the corresponding RR and RD in comparison with the middle-aged group. Although the ICR did not significantly differ between age groups, the highest ICR of 49.9 per 10^4 member-years (95% CI 35.6 to 64.1) was seen in the older adults group, with a RR of 1.3 (95% CI 0.9 to 1.8) and a RD of 11.3 per 10^4 member-years (95% CI -4.0 to 26.7). The lowest TCR of 9.3 per 10^4 member-years (95% CI 5.5 to 13.1) was seen in the young adults group, with a RR of 0.6 (95% CI 0.4 to 1.0) and a RD of -6.2 per 10^4 member-years (95% CI -11.4 to -1.0).

Figure 2 shows the total ICR by sex and age group. For every age group, the ICR was significantly higher for females than males. The DCR did not significantly differ between males and females in each age group (Figure 3). Figure 4 shows that for every age group, the TCR was significantly higher for females than males.

Only 20 (18%) of the 109 reported DCI cases underwent recompression within 24 hours. Recompression was performed within one week in 71 cases (65%), within two weeks in 89 cases (82%), and within one month in 98 cases (90%). In one case, recompression was performed on the 48th day after the onset of symptoms. In two cases, no recompression was performed. There were three cases where it was unknown when recompression was performed, whilst in five cases it was unknown whether or not recompression was performed. The DAN Japan hotline service was used in only 16 DCI cases (15%). Among the DCI claims, payment for residual disability was confirmed in only one case.

Discussion

In the present study, the DCR was 13.5 per 10^4 member-years, which is similar to the previously reported DCI rate among insured DAN USA members of 20.5 per 10^4 member-years.[10] The importance of claims data is that we had a known denominator, which allows for better cross-regional comparisons of diving injury incidence. However, insurance is not automatically included in DAN USA membership, and so the denominators differed and the number of dives per year also differed. The DCI rate of 20.5 per 10^4 member-years was considered roughly equivalent to that of the British Sub-Aqua Club members (32.59 per 10^4 member-years) when the annual number of dives is taken into account (DAN USA 0.82 per 10^4 dives; British Sub-Aqua Club 0.88 per 10^4 dives).[10]

Table 3

Claims rate (per 10^4 member-years) for injury, decompression illness (DCI) and trauma, rate ratio, and rate difference in young (< 40 years) and older (≥ 60 years) adults groups in comparison to the middle-aged (40–59 years) group. CI = confidence interval; Rate diff. = rate difference

	Age group	Member-years	No. claims	Claims rate	95% CI	Rate ratio	95% CI	Rate diff.	95% CI
Injury	Young adults	24737	95	38.4	30.7 to 46.1	1.0	0.8 to 1.3	-0.1	-9.7 to 9.4
	Middle-aged	46452	179	38.5	32.9 to 44.2				
	Older adults	9428	47	49.9	35.6 to 64.1	1.3	0.9 to 1.8	11.3	-4.0 to 26.7
DCI	Young adults	24737	40	16.2	11.2 to 21.2	1.3	0.9 to 2.0	4.1	-1.8 to 10.0
	Middle-aged	46452	56	12.1	8.9 to 15.2				
	Older adults	9428	13	13.8	6.3 to 21.3	1.1	0.6 to 2.1	1.7	-6.4 to 9.9
Trauma	Young adults	24737	23	9.3	5.5 to 13.1	0.6	0.4 to 1.0	-6.2	-11.4 to -1.0
	Middle-aged	46452	72	15.5	11.9 to 19.1				
	Older adults	9428	18	19.1	10.3 to 27.9	1.2	0.7 to 2.1	3.6	-5.9 to 13.1

Figure 3

Distribution of the decompression illness claims rate by three age groups (young adults, middle-aged and older adults) and sex (no statistically significant differences)

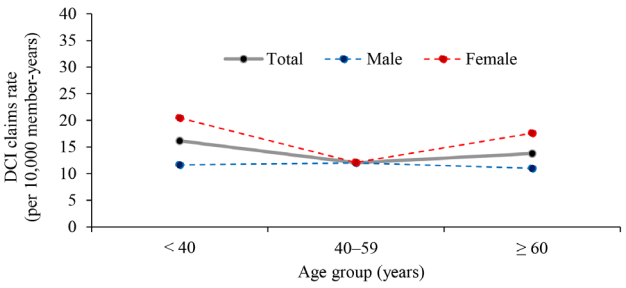
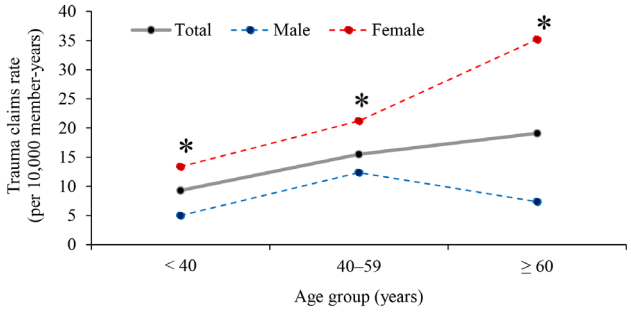


Figure 4

Distribution of the trauma claims rate by three age groups (young adults, middle-aged and older adults) and sex; *$P < 0.05$, females versus males



Japanese divers reportedly dive 60 times per year.[11] However, this figure includes both recreational divers and instructors. Instructors tend to dive more frequently than recreational divers, and many DAN Japan members are recreational divers, not instructors. Furthermore, even if a member is an instructor, an injury incurred while diving recreationally during a recreational dive trip is covered by the insurance. Recreational divers in Japan reportedly dive 34 times per year.[12] Using the figure of 34 dives per year, the DCR calculated in the present study of 13.5 per 10^4 divers is adjusted to 0.4 per 10^4 dives, which is half of the DCI rates reported in the above-mentioned studies. However, other studies have reported DCI rates of 0.53 per 10^4 dives[11] and 0.57 per 10^4 dives,[13] which are similar to the estimate for the present study. Notably, the prevalence of DCI in three previous studies[10,11,13] and the present study was based on retrospective self-reported data. However, the prevalence of DCI reported in a New Zealand study[9] was based on prospective field data, which makes this estimate more reliable. That study reported a DCR of 0.41 per 10^4 dives,[9] which is similar to the estimate for the present study.

The ICR in the present study was 39.8 per 10^4 member-years. This can be converted to 1.17 injuries per 10^4 dives using the same adjustment method as mentioned above. A previous study reported an ICR of 302 per 10^4 dives, which was based on self-reporting rather than medical records.[13] Another study using self-reported data found that DCI-like symptoms represent a small fraction of total diving-related injuries.[14] These findings indicate that many injuries do not require medical treatment. Questionnaire surveys generally tend to identify more cases of mild injury than studies reviewing treated cases and insurance claims. The New Zealand study, based on prospective field data, reported an ICR of 5.7 per

10^4 dives,[9] which is five times higher than that calculated in the present study. However, many reported cases were minor injuries, suggesting that an insurance claim was not submitted in most cases.[9] In addition, a study evaluating treated cases from a Scottish cold-water environment (Scapa Flow) also reported higher ICRs (4.4 and 7.6 per 10^4 dives), mostly for DCI, than that calculated in the present study,[15] suggesting that the ICR may vary in different diving locations. Variations in the diving location or type of diving can make comparative analysis problematic. This is important as the diving in Scapa Flow is deep, technical, drysuit diving. Therefore, comparisons of diving injury rates between specific cohorts must be made cautiously, as these rates are affected by multiple environmental variables as well as the type of diving.

In the present study, the DCR was 13.5 per 10^4 member-years, and the TCR was almost the same as the DCR. Fractures and dislocations accounted for most of the TCR. A recent study that evaluated diving-related injuries based on prospective field data also reported that trauma (35 cases) was more common than DCI (four cases),[9] with a higher TCR (3.6 per 10^4 dives) than the 0.4 per 10^4 dives reported in the present study. However, among the 35 traumatic injuries reported in this previous study, only seven cases needed emergency care and 10 cases required consultation with a general practitioner,[9] suggesting that this previous study included milder injuries than the present study. Thus, although DCI tends to be the focus of discussions about diving-related injuries, the findings from this and the present study indicate that it is also important to act to prevent other types of injuries.

Older age and the male sex are considered to be risk factors for diving-related fatalities.[4] Our findings suggest that females may be at greater risk of injury than the general diving population. However, this cannot be definitively concluded from the present data, as this difference in the number of injury claims might simply reflect the differences between males and females in the number of dives and diving style. A similar trend was seen in the trauma category, and some fractures (such as distal radial fractures) are more common in middle-aged females than in males, possibly due to osteoporosis.[16] Further research is needed to determine whether females are at greater risk of diving-related injuries than the general diving population.

In the present study, only 20 DCI cases (18%) underwent recompression within 24 hours, although at least 102 (94%) were eventually treated. A previous study reported that the number of cases with self-reported DCI symptoms is more than 25 times the number of treated DCI cases.[13] This suggests that mild DCI symptoms may resolve spontaneously. In the present study, only one DCI case received payment for residual disability. Our findings suggest that even among cases of treated DCI, many cases were mild and/or responded effectively to delayed treatment.

The present study has the following limitations. Each insurance claim was made by DAN Japan members or their families, not by physicians. Thus, the accuracy of the diagnosis written in the document is unconfirmed. Furthermore, there might have been other diving-related injuries that were not reported to DAN Japan, and members may not be representative of all recreational divers in Japan. Finally, it is impossible to accurately determine the number of times that each diver dived, which would provide the injury rate per number of dives.

Conclusions

The ICR was 39.8 per 10^4 member-years. DCI accounted for one-third of the total number of injuries, whilst trauma was the most common injury for which a DAN insurance claim was made. Compared with the general diving population, females may be at increased risk of diving-related injury, particularly injury due to trauma. It is important to take action to prevent injuries other than DCI. DAN Japan is considering what preventative interventions to implement.

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Medical conditions in scuba diving fatality victims in Australia, 2001 to 2013

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Abstract
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Introduction: This study identified pre-existing medical conditions among scuba diving fatalities in Australia from 2001 to 2013, inclusive, and assessed whether these conditions likely contributed to the deaths.
Methods: The National Coronial Information System (NCIS) was searched for scuba diving-related cases during 2001–2013, inclusive. Coronial findings, witness and police reports, medical histories, and autopsy and toxicology reports were scrutinised for pre-existing medical conditions and autopsy findings. Predisposing factors, triggers, disabling agents, disabling injuries and causes of death were analysed using a validated template.
Results: There were 126 scuba diving-related fatalities identified during the study period. Forty-six (37%) divers were identified as having a significant medical condition which may have contributed to their incident. The most common condition was ischaemic heart disease (IHD) which had been diagnosed in 15 of the divers. Thirty-two (25%) deaths were attributed to cardiac disabling injuries (DI) such as ischaemic heart disease and arrhythmias, although a cardiac DI was thought likely in another six. Respiratory conditions were implicated in eight (6%) deaths, at least four associated with cerebral arterial gas embolism. At least 14 (11%) divers who had contributory pre-existing medical conditions had been cleared to dive by a medical practitioner within the year prior.
Conclusions: Chronic health-related factors played a major role in almost half of these deaths; primarily cardiac conditions such as IHD and cardiac arrhythmias. Although fitness-to-dive (FTD) assessments have limitations, the high incidence of cardiac-related deaths indicates a need for 'older' divers to be medically assessed for FTD.

Introduction

Scuba diving is conducted in an inherently hostile environment and sometimes under conditions which can be challenging to even the most healthy and fit individuals. It is generally accepted that divers should have an appropriate level of medical and physical fitness and psychological health in order to facilitate safe diving.[1] However, some of the medical conditions believed to constitute an unacceptable risk have been subject to debate over the years.

Historically, most cardiac conditions, asthma and diabetes were considered absolute contraindications to scuba diving. However, over time, various diving medical organisations have modified their positions and a substantial number of divers with these and other comorbidities are diving, largely without incident.[2–4]

Coupled with this, the sport is attracting and accepting a more widely-aged cohort of the population and long-time divers are ageing.[5,6] Associated with increasing age is a higher prevalence of co-existing disease.[7] Such conditions may have been diagnosed and medically managed. However, they may often be undiagnosed as the diver may have been asymptomatic, or symptoms may not have been reported and appropriately investigated and managed.

Diving fatality reports reflect a substantial increase in the prevalence of deaths in older divers.[8,9] This is likely the result of the combination of increased participation and increased risk imposed by age-related co-morbidities. It is, therefore, important to better understand the impact that pre-existing medical conditions may have had on diving fatalities to better inform physicians and divers about the potential risks. Given this, we aimed to identify pre-existing medical conditions in the victims of scuba diving fatalities in order to assess whether these conditions likely contributed to the deaths.

Methods

We undertook a retrospective review of scuba diving-related deaths reported to various Australian State Coronial Services

during 2001–2013, inclusive. Approval for the study was received from the Human Research Ethics Committees of the Victorian Department of Justice, the Royal Prince Alfred Hospital and the Coroner's Court of Western Australia, the Queensland Office of the State Coroner and Deakin University.

The methodology for identifying relevant cases is described in detail elsewhere.[8] In brief, it included a comprehensive key word search of the National Coronial Information System (NCIS).[10] Relevant coronial findings, witness statements, police, autopsy, toxicology and equipment reports, as well as medical histories were critically reviewed by a team which included an experienced forensic pathologist, at least two diving physicians and an experienced diving fatality researcher. Possible contributing factors to the deaths, including the impact of identified medical conditions, were discussed until consensus was reached. Annual case series were prepared and published.[11] These were used in conjunction with a validated template to identify predisposing factors to each of the deaths.[12]

Results

One hundred and twenty-six scuba diving-related fatalities were identified during the study period. Forty-six (37%) divers were identified as having a total of 62 significant medical conditions which may have contributed to their incident (Table 1).

DIAGNOSED OR UNDIAGNOSED BUT REPORTEDLY SYMPTOMATIC CARDIAC CONDITIONS

The most common condition was ischaemic heart disease (IHD) which had been diagnosed in 15 (12%) of the divers. However, another eight had not been diagnosed with cardiac disease but had experienced previous dyspnoea and/or chest pain prior to the incident. These divers had significant autopsy evidence of IHD. The disabling injury (DI) in 19 of these 23 divers appears to have been cardiac-related.

Pre-existing cardiac arrhythmic conditions were assessed as likely to have contributed to the deaths of two divers. One of these had known atrial fibrillation. The other was a 17-year-old with undiagnosed prolonged QT syndrome. Cardiac dysfunction associated with viral myocarditis was believed likely to have contributed to the rapid ascent and subsequent cerebral arterial gas embolism (CAGE) in one diver. One other diver is believed to have been disabled by an arrhythmia precipitated, in part, by mitral valve incompetence.

CARDIAC FINDINGS AT AUTOPSY

Internal post-mortem examinations were available for 123 (98%) of the divers. Overall, it was determined that 32 (25%) deaths resulted from a cardiac-related DI. Some cardiac pathology was found in 25 (20%) other cases although this was not believed to have been contributory in most of these as other more likely DI's were identified. However, in six cases, a cardiac event was believed to have been reasonably possible, although the overall evidence was not sufficiently strong to make a confident determination.

Details of cardiac findings believed to have been contributory are shown in Table 2. The data in the table relate to the 38 cases (i.e., the 32 attributed to cardiac causes and the other six thought likely).

Autopsy evidence of left ventricular hypertrophy (LVH) was found in 24 (20%) of all divers (i.e., those with either cardiac or non-cardiac DIs) for whom internal post-mortem examinations were conducted and pertinent details reported (Table 3).

RESPIRATORY CONDITIONS

Pre-existing respiratory conditions likely contributed to the deaths of eight (6%) divers. These included asthma (three divers), chronic obstructive pulmonary disease (COPD, two), pulmonary cyst (one) and pleural adhesions in two divers, one of whom had a prior pneumothorax. Five of these divers were disabled by pulmonary barotrauma (PBT) and/or CAGE.

OTHER CONDITIONS

Twelve (10%) divers were being treated for hypertension, two for non-insulin dependent diabetes mellitus (NIDDM) and two had suffered a previous stroke. At least 11 of these divers appear to have had a cardiac event while diving. Three divers had a history of seizures or epilepsy. The asphyxia-related deaths of two of these three divers were unwitnessed, and it is possible, albeit unproven, that an underwater seizure occurred.

MEDICAL ASSESSMENT

There was some information on prior medical examinations in only 38 (30%) of the cases. In these, the assessments ranged from several days to 17 months prior to the incidents. At least 14 divers who had pre-existing medical conditions that contributed to their deaths had been cleared as fit-to-dive by a medical practitioner within the year prior to their incident. Although there might have been more, only three of these 14 reports specifically indicated that the examining doctor had training in the assessment of fitness to dive (FTD) and it is likely that most of the doctors had no specific FTD training. Three of the victims had pulmonary disease (emphysema and pleural adhesions) and the remainder had cardiac conditions. One of these had undergone a cardiac stress test five months prior, with a negative result.

MEDICATIONS

The medications taken by 38 of the divers were known,

Table 1

Sixty-two pre-existing medical conditions found in 46 scuba divers and the associated disabling injuries. CAGE = cerebral arterial gas embolism; COPD = chronic obstructive pulmonary disease; CVA = cerebrovascular accident; PBT = pulmonary barotrauma; IHD = ischaemic heart disease; IPO = immersion pulmonary oedema; MV = mitral valve; NIDDM = non-insulin dependent diabetes mellitus

Condition	Cases	Disabling injuries likely associated with the condition and number of injuries
Respiratory		
Asthma	3	PBT 1, asphyxia 1, unknown (cardiac? CAGE?) 1
Pleural adhesion	2	CAGE 2
Lung cyst	1	CAGE
Emphysema	1	cardiac (also had IHD)
COPD	1	CAGE
Cardiac		
IHD (diagnosed)	15	Cardiac 12, CAGE 1, unknown (cardiac? CAGE?) 2
IHD (undiagnosed but symptomatic)	8	cardiac 7, IPO 1
Myocarditis	1	CAGE
MV incompetence	1	cardiac
Arrhythmia	2	cardiac 2
Other		
Hypertension	13	cardiac 7, asphyxia 3, CAGE 1, IPO 1, unknown (CAGE?) 1
Diabetes (NIDDM)	2	cardiac 2
IPO	1	IPO
CVA	2	cardiac 2
Aneurysm	1	asphyxia
Epilepsy/seizures	3	CAGE 1, asphyxia 2
Bipolar	1	CAGE
Ankylosing spondylitis	1	cardiac
Alcohol/drug abuse	2	cardiac 2
IgA nephritis	1	cardiac

although seven others were reported to have been taking medications, the nature of which were unknown to investigators. It is possible that some others were also taking medications, but this information was not gathered by local investigators. Non-steroidal anti-inflammatory agents ($n = 9$), ACE inhibitors ($n = 7$), SSRI agents ($n = 7$) and antiplatelet agents ($n = 6$) were the most common medications taken by the scuba victims. The specific medications and potential impact of these will be discussed in a future report.

Discussion

Pre-existing health-related factors were determined to have played a major role in many of these deaths, the main ones being cardiac-related conditions such as IHD. Many incidents were believed to have resulted from cardiac arrhythmias occurring during the dive, generally based on existing cardiac pathology and its interaction with various diving-related precipitants.

CARDIAC ARRHYTHMIAS

Arrhythmias can be precipitated by immersion *per se,* especially in cold water.[13–16] Immersion counters the effect of gravity and encourages redistribution of venous blood from the limbs into the thorax. As a result, there is a substantial increase in the cardiac pre-load and work.[16–19] In addition, myocardial work can be further increased during diving by exercise, anxiety, cold-induced vasoconstriction, respiratory resistance and increased heart rate. The potential for an arrhythmia may also be increased by hyperoxia associated with diving.[20] Together these factors increase the likelihood of a cardiac-related event in a predisposed diver.[21–23]

As there is no definitive post-mortem test to confirm whether an arrhythmia has occurred, such a determination must be based on witness reports, medical history, and surrogate markers such as evidence of cardiac disease or abnormality at autopsy. Attributing a death to an arrhythmia is, therefore,

Table 2

Cardiac findings at autopsy of 38 cardiac-related scuba deaths. Multiple abnormalities (e.g., atheroma and ischaemia) were found with many. †Significant atheroma usually taken as > 75% vessel occlusion. *includes two divers with reported myocardial infarction (MI) and two with a previously diagnosed MI. #includes eight divers with reported MI and five with a previously diagnosed MI

Condition	Divers *n* (%)
Atheroma†	26 (68)
Ischaemia*	23 (61)
Cardiomegaly#	18 (47)
Left ventricular hypertrophy	14 (37)
Undiagnosed cardiac symptoms	6 (16)
Aortic stenosis	1 (3)
Left ventricular bridging	1 (3)
Cardiomyopathy	1 (3)
Tunnelling coronary arteries	1 (3)

somewhat speculative and must be carefully assessed on the basis of the available evidence. Arrhythmias are commonly associated with coronary atherosclerosis.[24] A critical stenosis believed likely to result in ischaemia is often regarded as being greater than 75% narrowing of the lumen. However, a substantially smaller stenosis may be significant when associated with other potential risk factors.[25]

Left ventricular hypertrophy, generally consequent to hypertension, is a known risk factor for sudden cardiac death and an increased incidence of serious arrhythmias.[26–28] A comparative study of matched scuba diving and traffic accident victims reported that both heart mass and left ventricular wall thickness were greater in the scuba victims.[29] In this current Australian series, although the numbers are small and should be interpreted cautiously, 37% of the 38 divers who were believed likely or possibly to have died as a result of a cardiac-related disabling event were found to have LVH of varying degrees at autopsy. This is an area that merits further investigation with larger groups of divers matched with appropriate controls.

The 25% incidence of cardiac conditions as the disabling injury in this series is consistent with the 26% incidence reported for divers in the USA.[30] That at least one quarter of scuba deaths in this era are cardiac-related is a reflection of the increased participation of older divers, as reported elsewhere[31–33] and confirmed in this Australian series.

Although potentially limited by low response rates, some Australian diver surveys have indicated a prevalence of cardiovascular disease and/or abnormality in the diver respondents ranging from 9 to 16%.[5] These reports are considerably higher than the 6% prevalence reported for the general Australian community,[34] and are consistent with the higher age of the divers. This may help explain the incidence of cardiac-related deaths in divers.

Table 3

Autopsy findings of left ventricular hypertrophy (LVH) in scuba divers with either cardiac or non-cardiac disabling injuries. CDI = cardiac event as the disabling injury

	LVH CDI *n* = 38	LVH non-CDI *n* = 81	LVH combined *n* = 119
n (%)	14 (37)	10 (12)	24 (20)
Age (years) Mean (SD)	53 (11)	41 (12)	45 (13)

As reported elsewhere,[8] the prevalence of obesity in victims in this series is substantially higher than in the general community and suggests that being overweight or obese may be a risk factor for a scuba diving fatality, which is often cardiac-related. The body mass index (BMI) of scuba fatality victims in the USA has increased over the last decades.[9]

Our findings suggest the need for 'older' divers to be medically assessed for fitness-to-dive in order to identify or risk manage medical conditions associated with diving fatalities. Current diving medical screening guidelines recommend a cardiovascular review for all males at age 45 and females at age 55 years.[35] However, Divers Alert Network Asia-Pacific insurance records indicate that these guidelines are often not followed.[36]

RESPIRATORY CONDITIONS

Pre-existing respiratory conditions including pulmonary adhesions or cyst, previous pneumothorax, emphysema and COPD were, unsurprisingly, mainly associated with CAGE and/or PBT. These conditions, where known, would have rendered the individuals unfit to dive.

There is a considerable number of individuals with asthma who are diving in Australia,[2–5] and elsewhere.[37–39] Although asthma was a contributing factor in several deaths, asthmatics are not over-represented in this series. Some individuals with well-controlled asthma appear to be able to dive relatively safely, especially under appropriate diving medical guidance.[2]

IMMERSION PULMONARY OEDEMA

Three of the deaths in this series were attributed to immersion pulmonary oedema (IPO). However, it might have been a contributing factor or the disabling injury in another five incidents. There have been an increasing number of reported cases in divers, both fatal and otherwise but it is probable that IPO is substantially under-reported.[40–43]

Most of the victims with pre-existing medical conditions were aware of these conditions, although, in others, it was occult. Some divers, both with diagnosed or occult conditions, had prior symptoms which were unrecognised or ignored, sometimes shortly before the fatal dive. This

can be particularly problematic in the diving environment with its physical and psychological challenges and where immediate first aid and rapid medical intervention is rarely readily available.

Some divers with potentially contraindicated conditions have dived with relatively few adverse incidents, often doing many dives over many years.[2] However, this survivor cohort of divers may not have been representative of the victim cohort as they were typically managed medically and, given that they had taken out diving insurance, might be generally more aware of their health limitations.

FITNESS TO DIVE ASSESSMENT

Until relatively recently in Australia, diver certification agencies required diver trainees to undergo a FTD assessment prior to certification. However, this has been progressively abandoned since about 2013 and a FTD assessment is now only required in limited circumstances based on responses to a self-reported medical questionnaire.

Although widely-used and supported by some data,[44] this self-reporting system has limitations.[45] It relies on the divers or prospective divers, as well as the dive professionals overseeing them, to fully understand the questions and the potential implications of failing to declare a relevant health condition. In any event, even if a FTD dive medical assessment was required initially, there is generally no requirement for further assessments, except for some higher-level training. This means that much of the diving population have aged without having undergone a subsequent diving medical check.

There are several on-going issues with FTD assessments. Many are conducted by doctors who have no specific training or knowledge of diving medicine and, therefore, have little or no appreciation of the physiological effects and demands of diving. This can lead to a poor assessment of a candidate's suitability.[46] Only one half of a survey cohort of DAN AP members with known medical conditions had discussed their condition with a diving doctor.[2] All divers with potentially contraindicated conditions should be encouraged to consult with a diving medical specialist in conjunction with their treating doctor.

Some individuals intentionally withhold information for fear that they will be prevented from participating.[47] An honest and complete medical history needs to be provided in order to facilitate a more accurate assessment.

Even when conducted by appropriately trained doctors, FTD assessments are fallible and have some inherent limitations.[48] Relatively few tests are routinely performed and appropriate tests will not always reveal underlying problems. Fitness-to-dive examiners are faced with the difficulty of selecting which diver candidates to investigate further, and what are the most appropriate tests to conduct. Medicals for older divers should be viewed somewhat differently to those of younger prospective divers due to the increased likelihood of cardiovascular disease with age.[34] Cardiac investigations may often be considered based on medical history, apparent fitness and/or family history. Such investigations have a cost, an associated risk, and are prone to both false positive and false negative results. However, if concerned about the cost of such investigations, the diver or prospective diver should balance these against the cost of equipment, dive travel and the potential implications of failing to detect a serious condition.

As mentioned, despite having been assessed as FTD within the previous year, at least 14 divers in this series died as a consequence of their pre-existing health conditions. Well-considered guidance is available to FTD examiners and much of the experienced diving medical community readily avail themselves to medical colleagues to discuss and advise on diving medical issues[1,49]

Although most divers who undergo diving medicals are assessed to be 'low risk', a dive medical, especially one performed by a doctor with appropriate training, will sometimes determine that an individual has an unacceptable risk of a diving accident. This risk assessment is usually carefully explained to, and discussed with, the individual. However, there is anecdotal evidence that some undeterred and determined individuals resort to 'doctor shopping' to obtain a diving medical clearance. In fact, in this series, a diver with a history of significant emphysema sent a friend as a substitute for another assessment. His subsequent death was a result of a pulmonary barotrauma and CAGE while diving.

LIMITATIONS

As with any uncontrolled case series, the collection and analysis of the fatality data are subject to inevitable limitations and uncertainties associated with the investigations. Witness reports varied in their likely reliability. Police and medical reports varied in their content. As many incidents were unwitnessed, some of the assertions in the reports are speculative. The diagnoses of cardiac disabling injuries were made based on available evidence, including autopsy reports, medical histories and witness reports and determined by consensus of an expert panel which included a forensic pathologist. However, in some cases, the final diagnoses may not be correct.

Many data items were not available which rendered the study data incomplete, thus limiting the conclusions that can be drawn.

Conclusions

Chronic health-related factors were determined to have played a major role in more than one third of the deaths in this series. The main factors identified were cardiac-related

conditions such as IHD and cardiac arrhythmias. At least one quarter of the deaths likely resulted from a cardiac disabling injury.

The comparatively high prevalence of obesity in the victims, as reported elsewhere, suggests that obesity may be a risk factor for a cardiac-related diving fatality, as may left ventricular hypertrophy. A variety of other conditions such as respiratory disease, diabetes, hypertension and possibly epilepsy were likely or possible contributors to these fatal incidents.

Fitness-to-dive assessments have limitations and relatively recent FTD assessments had failed to prevent some health-related deaths in this series. The high incidence of cardiac-related deaths indicates a need for 'older' divers to be medically assessed for FTD.

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Scuba diving fatalities in Australia, 2001 to 2013: Diver demographics and characteristics

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Key words
DAN – Divers Alert Network; Diving deaths; Diving incidents; Obesity; Research; Solo diving

Abstract
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Introduction: This study identified characteristics of victims of fatal scuba diving incidents to determine contributing factors and inform appropriate countermeasures.

Methods: The National Coronial Information System (NCIS) was searched to identify scuba diving deaths for 2001–2013, inclusive. Data were extracted from witness and police reports, medical histories and autopsies. Descriptive statistics were used to analyse these data.

Results: There were 126 scuba diving-related fatalities identified during the study period. The mean age was 44 years, 99 (79%) victims were male and 83 (77%) were either overweight or obese. Most deaths occurred in New South Wales and Queensland, often in a commercial setting. Twenty-three (79%) Queensland victims were overseas tourists. At least 52 (41%) were novices and 17 (13%) died during training or an introductory scuba experience. Only 35 (28%) were with a buddy when the incident occurred and at least 81 (64%) were still wearing weights when recovered.

Conclusions: The age of these victims may reflect an older cohort of participants and the associated higher prevalence of chronic medical conditions. The high prevalence of obesity suggests that this may be a risk factor. The high proportion of deaths in overseas tourists highlights an on-going need for appropriate screening and monitoring in what may be a higher risk cohort. The number of deaths that occurred under instruction highlights the importance of careful assessment of the site, prevailing conditions, an appropriate instructor-student ratio and close supervision.

Introduction

With more than 25,000 km of coastline and a mix of temperate and tropical waters including the Great Barrier Reef, Australia offers a variety of scuba diving opportunities for both locals and tourists. Like many adventure activities, scuba diving is conducted in a potentially hostile environment and involves certain risks. Such risks may arise from the diving environment itself or may be related to various other factors including: the participant's physical, medical and psychological health; training or experience; equipment failure unfamiliarity or misuse; poor decision-making; and attitudinal factors. As a result, there is inevitably an associated morbidity and mortality,[1–7] which has been estimated to be 0.48 deaths per 100,000 dives for Australian residents.[8]

Earlier reports have suggested that the age of fatality victims has increased over time, raising concerns about the increased potential for co-existing medical conditions with age and their impact within the diving environment.[1,2,6,9] Some US data revealed an increase in the BMI of diving victims over recent decades.[1] Other factors including training, experience, supervision and equipment issues can affect diving safety and it is valuable to assess the influence of these over more recent times.[2,4,6]

In order to better understand the causation of Australian scuba diving fatalities and so enable the development of appropriate prevention strategies, it is important to know the characteristics and activities of Australian divers and to comprehensively document and carefully analyse diving fatalities. This has been achieved, in part, through the publication of annual Australian fatality reports.[10–21] However, it is valuable to examine the combined data to identify any trends that may impact safety.

The aim of this study was to identify the demographics and diving characteristics of victims of scuba diving fatalities in Australia from 2001 to 2013. Further reports will examine

the chain of events in these fatalities including associated medical issues and, together, will better inform appropriate countermeasures.

Methods

This was a case series of scuba diving-related fatalities that occurred in Australian waters from 2001 to 2013, inclusive.

ETHICS APPROVAL

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

SEARCH

A comprehensive key word search was made of National Coronial Information System (NCIS)[22] to identify scuba diving-related deaths that were reported to various state coronial services for the years 2001–2013, inclusive. Key words included scuba, compressed air, compressed gas and underwater. As it can take many years for some coronial cases to be closed and the relevant data to become available, the particular period was selected as the principal investigator had access to more complete data for this period. Cases identified were matched with cases collected by the Divers Alert Network Asia-Pacific (DAN AP) via the media or the diving community to minimise the risk of over- or under-reporting.

REVIEW PROCEDURE

For cases in 2001 and 2002, the principal investigator reviewed the coronial reports in conjunction with Walker's relevant Project Stickybeak reports published in the diving medical literature.[20,21] Project Stickybeak collected and reported Australian diving fatality data from 1972 and 2003, inclusive. The 2003 cases were reviewed by both Walker and the principal investigator, consensus reached and a report published.[19]

The procedure followed for cases between 2004 and 2012 was as follows:

1. Both the principal investigator and another investigator reviewed the police reports, witness statements and coronial reports and independently prepared a summary of each incident.

2. The principal investigator reviewed both datasets, investigated any discrepancies and prepared edited incident summaries.

3. The incident summaries, coronial and autopsy reports were independently reviewed by a research team comprising diving physicians, a forensic pathologist with extensive experience in diving autopsies, a retired judge and researchers with substantial experience in diver training, data collection and accident management.

4. Annual series reports were written and published.[10–18]

The principal investigator solely reviewed the coronial data for cases in 2013 and extracted relevant data.

Some historical fatality data from Project Stickybeak was combined with similar data from the current study period in order to investigate any trends over the extended period from 1965 to 2013, inclusive. Both sets of data were based on the available coronial files.

OUTCOME MEASURES

A range of outcome measures were extracted. These included diver demographics, origin, certifications and experience; dive location and setting; buddy circumstances, dive purpose and depth and equipment (weights, buoyancy compensation, breathing gas supply, equipment failure).

It is difficult to satisfactorily define diving experience as it is not only dependent on the number and recency of dives, also on the nature of these dives. Relevant background information was missing in many of the cases, so relatively simple and somewhat arbitrary definitions of experience were used, despite their shortcomings. A 'novice' was defined here as having done 30 dives or less, an 'experienced diver' had performed more than 30 dives, and a 'very experienced' diver had done at least 200 dives.

STATISTICAL ANALYSIS

Trends in numbers of deaths were modelled using log linear Poisson regression, which is the most appropriate analysis technique for trends in count data. This was modified for trends in female deaths by the addition of total deaths as a denominator allowing for analysis of trends in the proportion of deaths among women. Age at death is a continuous variable, so trends in mean age at death were modelled using weighted least squares regression. Trend analyses were done using the Stata 15 software.[23] The exception was where a trend appeared to change across the study period. In this case, the Poisson model was extended to allow the fitting of two simultaneous models using the 'Joinpoint Method'. This technique fits two Poisson models joined at a 'joinpoint', with both the optimal position of the join (if any) and the model parameters estimated simultaneously using a sequential permutation test procedure.[24] This was done using the National Cancer Institute Joinpoint software.[25] Descriptive analyses based on means and standard deviations or medians and ranges as appropriate was conducted using SPSS Version 25 (IBM Armonk, NY; 2017). Comparisons of proportions employed odds ratios (OR) accompanied by

Figure 1
Distribution of scuba fatalities by age and sex (n = 126)

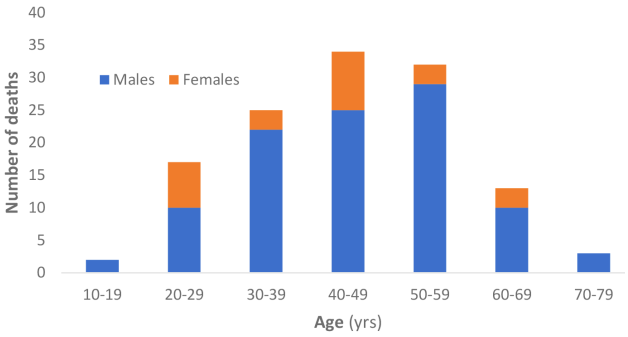
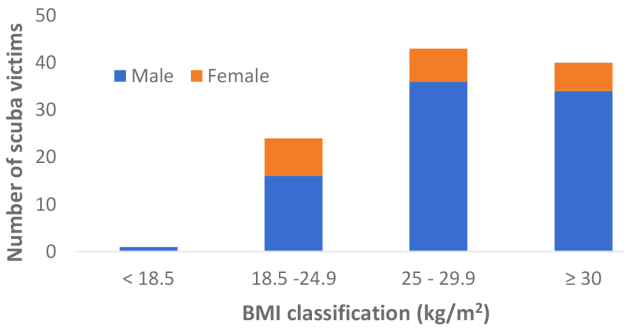


Figure 2
BMI classifications by sex for scuba victims (n = 108)



95% confidence intervals (95% CI). Trends in counts and proportions are reported as the average proportional change between time periods accompanied by 95% confidence intervals. The level of significance was 0.05.

Results

STUDY PERIOD 2001–2013

Demographics

There were 126 scuba diving fatalities during the study period. Despite annual fluctuations, there was little overall change over the study period in the mean age of the victims. The mean (SD) age was 44 (13) years and 99% (79%) of the victims were male (Figure 1).

The body mass index (BMI) data were available for 108 victims. The mean (SD) BMI was 28.5 (5.0) kg·m^2. Two thirds of victims were either overweight (BMI 25–29) or obese (BMI ≥ 30) (Figure 2).

Of note, 12 (38%) of the 32 (25%) deaths determined to be cardiac-related were among victims who were obese, with BMIs ranging from 30 to 42 kg·m^2. (The other major disabling agents were asphyxia (47%) and arterial gas embolism/pulmonary barotrauma (15%) which are discussed in a further paper).

Certifications and experience

The scuba diving certification levels were known for 90 victims (Table 1). Based on 74 cases with data on the period of diving, the median (IQR) was 3 (1–12) years. The median (IQR) number of dives performed prior to the fatal incident was 20 (5–100) dives.

In addition to, or in the absence of a reported number of dives, witness statements from their buddies and/or families were also used to help determine the divers' possible level of experience within the defined parameters. On this basis, 52 (41%) victims were defined as novices, 43 (34%) were experienced and 15 (12%) were very experienced. In 16 (13%) cases, indications of experience were unavailable. Nine (7%) victims died on their first dive and 15 (12%) within their first five dives. At least 12 of the scuba victims had not dived for periods ranging from six months to 11 years immediately prior to their fatal dive.

Location and setting

New South Wales and Queensland had the highest proportions of deaths with approximately one quarter of deaths each (Table 2). No deaths occurred in the Northern Territory or Australian Capital Territory. The mean (SD) age of the scuba diving victims varied little between states and territories.

Overall, 58 (46%) deaths occurred in a commercial setting, either on dive charters or under the supervision of a dive professional. More than 80% of scuba deaths in Queensland occurred in a commercial setting. The relative proportions by state and territory were Queensland (86), Victoria (50), New South Wales (41), Tasmania (33), Western Australia (26), South Australia (12), Northern Territory (0) and Australian Capital Territory (0).

Origin of victims

Overall, 35 (28%) victims were tourists. Twenty-four (83%) of the 29 scuba victims in Queensland were tourists, one from interstate and the remainder from overseas. The proportions of victims who were tourists in the other states and territories ranged from zero (Victoria, South Australia, Northern Territory, Australian Capital Territory), 11% (Western Australia), 16% (New South Wales) to 22% in Tasmania.

Buddy circumstances

Based on available reports, 13 (10%) victims had set out diving solo. Fifty-five (44%) had separated from their buddy or group, intentionally or unintentionally, prior to the incident and another 22 reportedly separated during the incident. No relevant information was available for one incident. Overall, only 35 (28%) victims were with their buddy or buddies at the time of their demise.

Table 1
Certification levels of victims (n = 126)

Certification level	*n* (%)
None	8 (6)
Under training	12 (10)
Open water (OW)	39 (31)
OW + other courses	14 (11)
Technical	10 (8)
Instructor	7 (6)
Not reported	36 (29)

Based on the relevant data available for 112 of the incidents, there was a higher frequency of separation prior to the incident when the divers were within a group larger than two, rather than within a buddy pair. This occurred with 23/33 (70%) of victims in groups and 32/79 (41%) of victims in buddy pairs. (OR = 3.4; 95% CI = 1.4 to 8.1, P = 0.006).

Dive purpose

No activity details were available for one diver. Seventy-nine (63%) victims were essentially sightseeing. Twenty-three (18%) were harvesting seafood (recreationally) and spearfishing. Another diver was collecting cuttlefish eggs for research.

In addition there were five divers who died during work-related activities. Four of these were amateurs who died during activities such as clearing an anchor, unfouling a propeller or cleaning their boat's hull. The last, an instructor, died while leading a deep technical dive using a closed-circuit rebreather (CCR).

Seventeen (13%) victims died while undergoing diver training or while being supervised by an instructor on an introductory scuba experience. One of these occurred while being trained by a friend, who was not a qualified instructor. The others occurred while under the care of certified instructors. The number of deaths in the various training programs were: introductory scuba (5), open water (6), advanced open water (2), rescue (1), technical (1), commercial (1) and unsanctioned (1).

Six of these deaths were associated with pre-existing medical conditions, another six were associated with poor supervision in adverse conditions, and at least two involved equipment problems precipitating a rapid ascent and subsequent cerebral arterial gas embolism (CAGE).

Depth of incident

The depth at which the incident occurred, or was believed likely to have occurred, was recorded in 116 incidents. The median (IQR) depth was 12 (6–21) metres (m), with a range of 0 to 125 m. Forty-six (37%) incidents occurred at a depth of ≤ 10 m, and 72% ≤ 20 m.

Table 2
Distribution and mean age of scuba fatalities by state or territory (n = 126). n/a = not applicable

Location	*n* (%)	Age (years) mean (SD)
New South Wales	32 (25)	47 (13)
Queensland	29 (23)	46 (15)
Victoria	20 (16)	41 (14)
Western Australia	19 (15)	43 (9)
South Australia	17 (13)	41 (12)
Tasmania	9 (7)	47 (12)
Northern Territory	0 (0)	n/a
Australian Capital Territory	0 (0)	n/a

Weighting

In 21 cases the weighting circumstances were unreported, and six victims had not set out wearing any weights. Of the 99 victims who were known to have been wearing a weight belt or integrated weights at the time of diving, 81 (82%) were found still wearing the weights. Only 18 of the victims were known to have ditched their weights.

The amount of weight carried was known for 63 victims. The median (IQR) weight was 9 (6, 12) kg with a range of 2 to 20 kg. Thirty of the 33 divers who carried 9 kg or more were diving in temperate waters (i.e., below the Tropic of Capricorn).

Buoyancy compensators

Data were available about the buoyancy compensator device (BCD) circumstances in 95 of the incidents. Overall, 45 (47%) victims were found with an uninflated BCD and in only two cases was the BCD later found to be faulty. Of note, 38 of the 45 victims (84%) found with an uninflated BCD were also still wearing their weight belt.

Breathing gas supply

Six (5%) of victims had been breathing a gas other than air. Five of these were using closed-circuit rebreathers and carrying air and oxygen cylinders to provide the 'bottom mix'. An open-circuit diver who died from fulminant DCS had been breathing air on the bottom and nitrox during decompression.

At least 42 (34%) of the victims had exhausted or near-exhausted their breathing gas supply. Sixty-four (51%) had sufficient gas to surface safely, with 40 (32%) having at least one quarter of their supply remaining. In 15 cases the gas supply circumstances were unstated or not applicable.

Equipment faults

There was no mention of any equipment examination in 13

Figure 3
Scuba diving deaths in Australia, 1965–2013 (n = 393)

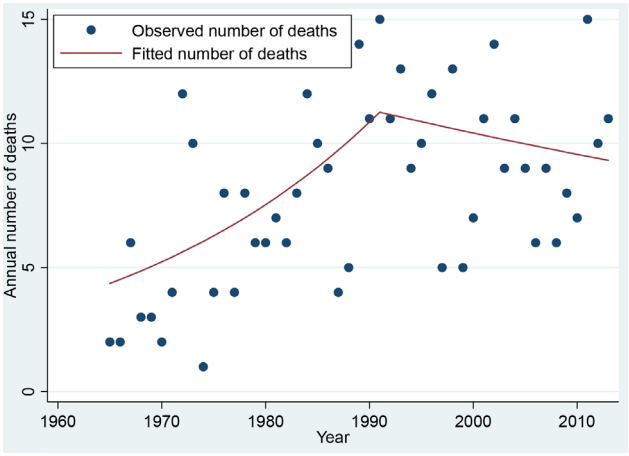
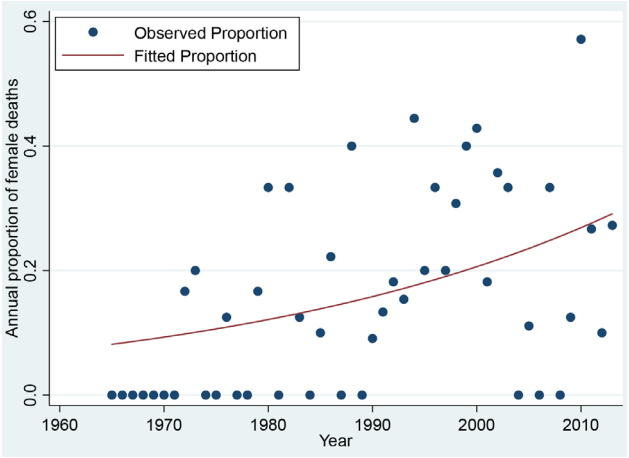


Figure 4
Annual female deaths as a proportion of total deaths 1965–2013



cases and nothing abnormal reported in 72 cases. Police examiners reported finding faults with the equipment in 41 (33%) cases although most of the faults were thought not to have been a significant contributor to the fatalities. The faults identified and their frequency included breathing gas impurities (11), regulator (9), BCD (9), pressure gauge (4), closed circuit rebreather (1) and others (7).

Faulty (high reading) cylinder contents gauges were associated with and thought likely to have contributed to four incidents, all of which involved the victim running out of breathing gas. Most of the regulator faults (7 cases) involved perforated mouthpieces which allowed water aspiration. Most of the identified BCD problems (8) involved malfunctioning inflator/deflator mechanisms.

Equipment-related problems were the likely triggers in at least eight of the incidents. These included a detached demand valve, faulty tank valve, faulty inflators on a BCD and a drysuit, and out-of-date and faulty oxygen sensors on a rebreather.

There were 11 cases where the breathing gas did not meet the relevant Australian Standards.[26] In 10 of these, the water vapour and carbon dioxide levels were high, although not believed to have been contributory. In one case, excess oil in the breathing air was thought likely to have caused nausea and led to a rapid ascent with consequent CAGE.

COMBINING STUDY RESULTS WITH HISTORICAL DATA – 1965 TO 2013

The DAN AP database indicates that from 1965 to 2000 there were a total of 267 recorded fatalities involving scuba divers in Australian waters. The additional 126 cases from this current study period makes a total of 393 deaths from 1965 to 2013, inclusive. Some demographic data and trends revealed from these combined data are presented in Figures 3 to 5 and Table 3.

The Joinpoint trend analysis as shown in Figure 3 identified a statistically significant change in the trend in average scuba deaths in 1991. On average scuba diving deaths rose by a factor of 1.44 (95% CI 1.08 to 1.91) each decade between 1963 and 1991. This means that each decade had, on average, 44% more deaths than the previous decade. After 1991, there was no statistically significant trend.

Figure 4 shows the number of female deaths in each year as a proportion of total deaths, along with the trend line fitted by the Poisson model. Overall 18% of the scuba victims were females, with the proportion rising from 0% in 1965 to 27% in 2013. On average, the proportion of female scuba diving deaths rose across this period by a factor of approximately 1.30 (95% CI 1.11 to1.53) each decade.

The mean (range) age for the scuba divers was 33.7 (13–72) years. Table 3 shows the age data for the scuba victims as a group, and by gender.

Figure 5 shows the mean age at death in each year, along with the trend line fitted by the weighted least squares regression model for the years 1965 to 2013. The mean age rose by an average of 4.6 years (95% CI 3.4–5.7) for each decade. Mean age was approximately constant following 2001, suggesting that it may have reached a plateau at this point.

Discussion

Among the 126 scuba diving-related fatalities, the victims were predominantly middle-aged males and more than three quarters were overweight or obese. The majority of the divers were certified but around 40% were still novices. Most of the victims in Queensland were international tourists. Three quarters of the victims were alone at the time of their incident.

DEMOGRAPHIC FACTORS

Various surveys since 2001 have indicated that around three

Table 3
Age measures of scuba victims, 1965–2013

Age (years)	All *n* = 393	Male *n* = 393	Female *n* = 70
Mean (SD)	37.7 (13.5)	38.1 (13.7)	35.8 (12.6)
Range	13–72	13–72	18–65
% > 50	21	22	16

quarters of active Australian scuba divers were male.[8,27] Our finding that 80% of victims were males likely reflects their high participation rate as well as the greater prevalence of chronic medical conditions, especially cardiac and cardiovascular, in the male population.[28] The increase in the proportion of female victims over the extended period likely reflects increased participation by females.

National sporting surveys have also indicated that 30% of Australian scuba divers were aged 45 years or older,[8] consistent with that of survey respondents of mainly relatively inexperienced divers.[27] However, survey respondents of more active divers (e.g., DAN members and PADI members) were older, with medians in excess of 45 years.[27] By comparison, the ages of scuba diving victims have increased substantially over the decades but appear to have plateaued since 2000, with a median of 45 years. This is similar to the survey respondent cohort of more active divers.[27]

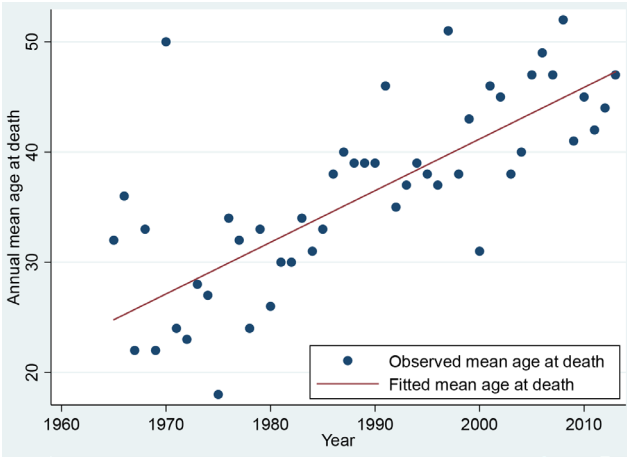
OVERWEIGHT OR OBESITY

The proportion of Australian scuba victims who were either overweight or obese (78%) was considerably higher than the 63% in the Australian adult population.[28] It was also much higher than the prevalence reported by three cohorts of Australian divers, which ranged from 40% in younger, less experienced divers, to 64% in older, active divers.[27]

The prevalence of obesity in the Australian scuba victims (35%) was also substantially higher than in the general community (28%)[28] and in the diver survey respondents.[27] This suggests that being overweight or obese may be a risk factor for a scuba diving fatality, which is often cardiac-related.[10–16,2,6] This is consistent with data from the general population that indicate an association between significant health conditions and being overweight or obese,[29–32] as well was an association between obesity and sudden cardiac death.[33,34]

Obesity *per se* can be a contributory factor to an incident.[35,36] The adverse effects of obesity on respiratory mechanics when immersed, the need for additional weights and increase in the hydrodynamic drag may act to compromise safety by adding to the cardiac workload. Undiagnosed obstructive sleep apnoea, diabetes, as well as hypertension and high cholesterol are more common in obese individuals and are all risk factors for coronary artery disease.[37]

Figure 5
Age of scuba victims, 1965–2013



ORIGIN OF VICTIMS, LOCATION AND SETTING OF DIVING

Diving in an unfamiliar setting can introduce additional risks due to the lack of local knowledge about factors such as sea conditions, underwater topography, differences in diving practices, cultural differences and language difficulty. This is exacerbated by inexperience.[10–18] Almost 30% of the scuba victims in this series were tourists, mainly from overseas. Queensland is a very popular destination for tourists and many dive or snorkel on the Great Barrier Reef, mostly in a commercial setting.[38] As such, it is unsurprising that almost one quarter of the scuba deaths occurred in this State, 80% of these involving tourists who were predominantly international.

Diving-related tourism is an important income source for Queensland and, in 1992, a regulated code of practice for diving activities was introduced in order to provide regulated safety guidelines for the industry in that state. This has been periodically updated, the latest version being released in 2018.[39] However, despite the comparatively high number of deaths, the estimated annual fatality rate for tourist scuba divers in Queensland is considerably lower than the overall rate for Australian residents.[8] The risk in Queensland may be partly mitigated by generally more favourable diving conditions and closer oversight as a result of enforcement of the code of practice. Although a code of practice has been created for Victoria, it is voluntary, likely unknown to many local dive professionals due to minimal promotion, and, in the long-time experience of these authors, is often not followed.[40]

One quarter of the scuba deaths occurred in New South Wales, most involving residents, and in non-commercial settings. New South Wales has a relatively large resident diving population, reportedly encompassing 27% of active Australian divers.[41] Its long and accessible coastline and variety of dive sites is conducive to independent diving and snorkelling.

Diving in the southern states can be more demanding with respect to colder water, rough and variable conditions, poorer visibility and the need for greater thermal insulation with its associated increased weighting requirements. This is reflected in the considerably higher annual scuba fatality rate estimated for Victoria.[8]

CERTIFICATION AND EXPERIENCE

Of the 90 scuba victims whose certification level had been recorded, almost all (91%) were known to be certified or were undergoing training at the time of their demise. This proportion was considerably higher than the 77% reported for scuba victims from 1972 to 1993[42] and suggests an improved recognition of the need for training and certification and/or possibly a greater ease of determining certification status due to the internet. Although, it is generally accepted that training reduces the likelihood of a mishap, there are few published data specifically supporting this. However, some reports indicate that diving fatality victims had often broken one or more of the general diving safety rules, so learning, and adhering to, such rules should provide a safety buffer.[43,44]

Although it was not investigated in this study, it is logical that, in general, competent swimmers are more likely to be more comfortable in the aquatic environment than poor swimmers. Swimming competency is assessed prior to diver certification, although the minimum requirements are basic[45,46] and some certified divers likely have poor aquatic skills. These do not hold them in good stead if challenged by difficult surface conditions.

Defining 'experience' in diving is extremely difficult, with the number of dives, time of accumulation, recency of activity and the nature of diving among the influencing factors. Given the varying definitions of experience used in previous fatality reports from Australia and elsewhere, it is difficult to confidently measure and compare changes in the level of experience of the victims over time. Although the defining criteria in this series were somewhat arbitrary, at least 46% of the victims had done a minimum of 30 dives, so experience *per se* is not necessarily protective. Factors such as recency of diving and relevance of experience were highlighted in this series, as was the need for specific skills and specialised training for more challenging environments.

It is likely that lack of recent diving affects the current competency of a diver and can be contributory to accidents. At least 12 of the scuba victims had not dived for periods ranging from six months to 11 years. A recurring theme in diving accident reports and also suggested in this series is 'experienced' divers getting into difficulties after an extended absence from diving. Unless practiced, it is inevitable that diving skills will deteriorate over time so re-familiarisation under controlled conditions is well-advised. Because certain medical conditions can affect diving safety, any potential change in an individual's fitness to dive needs to be carefully considered and acted upon. Equipment requires proper inspection and servicing, especially after not being used for an extended period as deterioration can occur.[12]

It is concerning that 16 (13%) deaths in this series involved student divers, 11 of these 16 being novices, while participating in sanctioned training or an introductory scuba experience. Poor planning and inadequate supervision, often in adverse conditions, was identified as major contributors to many of these.[11,13]

BUDDY SYSTEM

The absence, or breakdown, of the 'buddy system' is another recurring theme in dive fatality reports.[2,47,48] However, it can sometimes be difficult to determine exactly when the actual separation occurred and at what stage of the chain of events. In this series, little more than one quarter of the victims were reported to have been with a buddy at the time of their demise. Although this is higher than the 18% reported in an earlier Australian series,[2] it indicates an on-going problem that needs to be further addressed.

The buddy system is taught to trainees as an important part of diving safety in a hostile environment where a diver might need assistance with equipment, entanglement, breathing gas supply, managing sea conditions and a variety of other potential problems. Training in self-sufficiency and the carrying of redundant equipment is a valuable safety measure. However, a well-trained and vigilant buddy can be an important asset in an emergency.

Perhaps with the exception of a team trained in that manner, diving in buddy groups larger than two introduces additional challenges in monitoring and maintaining contact.[4] In this series, victims were more than three times as likely to have separated if they were diving in a buddy group larger than two.

BECOMING POSITIVELY BUOYANT IN AN EMERGENCY

Delay to resuscitation adversely affects the outcome and precious time is lost if it is necessary to locate and recover a victim underwater. Therefore, it is better for an unconscious diver to be at the surface, rather than requiring a search and recovery from underwater.[49] It follows that, if a diver is in danger of becoming unconscious, whether underwater or on the surface, it is important to attain positive buoyancy. This can be achieved by inflating the BCD, ditching weights, or both. The exception to this is during a overhead environment dive where the surface is not directly accessible.

In an earlier review of Australian compressed gas diving-related fatalities, almost three quarters of the victims were found wearing their weights.[2] In this 2001–13 series, at least two thirds (possibly up to 82%) of the divers still

had their weights *in situ* at the time of attempted rescue or recovery. This highlights an on-going problem of divers being reluctant, or unable, to effectively gain positive buoyancy in an emergency. In many cases it is probable that, by the time the divers recognised the need to do so they were too incapacitated to act effectively. Ditching the weight belt is an important emergency measure and training drill for divers, and needs to be practiced, embedded and periodically revisited.

ANNUAL SCUBA DIVING FATALITIES IN AUSTRALIA

Despite various progressive improvements over time, including the increasing and now ubiquitous use of BCDs, the authors are not aware of any clear 'intervention' such as new equipment, procedures or regulations that would explain the apparent plateauing of the annual fatalities as indicated in Figure 3. However, it appears likely that the trend resulted from reduced scuba diving activity and this will be discussed in a future report.

LIMITATIONS

As with any uncontrolled case series, the collection and analysis of the fatality data are subject to inevitable limitations and uncertainties associated with the investigations. Witness reports varied in their likely reliability. Police reports varied in their content, often related to the expertise of the investigators. Given that many incidents were unwitnessed, some of the assertions in the reports are speculative. Many data items were not available which rendered the study data incomplete, thus limiting the conclusions that can be drawn.

Conclusions

The older age of victims in this series may reflect a diving population with an increasing prevalence of age-related and chronic medical conditions. The high prevalence of obesity among these victims suggests that obesity may be a likely risk factor in such deaths, possibly due to cardiac stress and reduced fitness and mobility.

The higher frequency of deaths in NSW and Queensland likely reflects the greater participation in these states. The high proportion of deaths in overseas tourists diving with commercial operators in Queensland is consistent with the popularity of diving on the Great Barrier Reef but highlights an on-going need for appropriate screening and monitoring in what may be a higher risk cohort.

The substantial number of deaths that occurred during training or an introductory scuba experience highlights the importance of careful assessment of the dive site, the prevailing conditions, an appropriate instructor-student ratio and close supervision.

Mortality may also be reduced with improved buddy selection and monitoring, training in solo diving for those who do so, as well as greater emphasis and practice on the benefits of attaining positive buoyancy in the event of impending unconsciousness in the water.

Recommendation: Data collection for diving fatalities in Australia and elsewhere would be improved and standardised by the use of specific, dedicated proformas. An example of which can be found at: https://adsf.org.au/wp-content/uploads/2019/04/Data-Collection-1.pdf.

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Pupillometry is not sensitive to gas narcosis in divers breathing hyperbaric air or normobaric nitrous oxide

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Key words
Diving research; Nitrogen; Nitrous oxide; Physiology

Abstract
(Vrijdag XCE, van Waart H, Sleigh JW, Mitchell SJ. Pupillometry is not sensitive to gas narcosis in divers breathing hyperbaric air or normobaric nitrous oxide. Diving and Hyperbaric Medicine. 2020 June 30;50(2):115–120. doi: 10.28920/dhm50.2.115-120. PMID: 32557412.)
Introduction: Gas narcosis impairs divers when diving deeper. Pupillometry is sensitive to alcohol intoxication and it has been used in anaesthesia to assess nitrous oxide narcosis. It is a potential novel method to quantify narcosis in diving. The aim of this study was to evaluate pupillometry for objective measurement of narcosis during exposure to hyperbaric air or nitrous oxide.
Method: Pupil size in 16 subjects was recorded directly at surface pressure and during air breathing at 608 kPa (equivalent to 50 metres' seawater depth) in a hyperbaric chamber. Another 12 subjects were exposed to nitrous oxide at end-tidal percentages of 20, 30 and 40% in random order at surface pressure. Pupil size and pupil light reflex were recorded at baseline and at each level of nitrous oxide exposure.
Results: Pupil size did not significantly change during exposure to hyperbaric air or nitrous oxide. The pupil light reflex, evaluated using percentage constriction and minimum diameter after exposure to a light stimulus, was affected significantly only during the highest nitrous oxide exposure – an end-tidal level of 40%.
Conclusion: Pupillometry is insensitive to the narcotic effect of air at 608 kPa in the dry hyperbaric environment and to the effects of low dose nitrous oxide. Pupillometry is not suitable as a monitoring method for gas narcosis in diving.

Introduction

Divers venture underwater either for work or pleasure, and may experience nitrogen narcosis. The onset of narcosis symptoms is expected around 30 metres' sea water (msw) (4 atmospheres absolute pressure [atm abs], 405 kPa), when breathing air.[1] These symptoms influence the diver's capacity to make decisions and are a contributing factor in incidents.[2] Besides incidents, the reduced mental capacity affects the quality of work underwater, causing divers to make more mistakes or taking longer to complete their task.[3] Quantifying nitrogen narcosis has been investigated since Behnke first attributed the narcotic effects experienced during hyperbaric exposures to nitrogen.[4] Ideally narcosis could be continuously monitored in real time without interfering with diving activities.[5] Pupillometry is a potentially novel method to achieve this.

Pupillometry is the measurement of the pupil size and pupillary reflexes. The pupil size is subject to reflex responses to light/dark or pain stimuli. Pupil size and reflexes can be measured objectively with a portable infrared pupillometer with a light flash, which can calculate multiple variables such as latency of onset, magnitude of constriction and constriction velocities (Figure 1). Both the resting pupil size and light reflex are modulated by a feedback loop between the eye and the oculomotor nucleus in the pretectal area of the midbrain. Other areas in the brain influence this nucleus to dilate or contract the pupil based on general sympathetic and parasympathetic pathways.[6]

Previous research has shown that pupillometry can be used to screen workers for the influence of alcohol.[7] An increase in pupil diameter was measured directly after 0.6 $g \cdot kg^{-1}$ ethanol intake. The pupil light reflex has been used to quantify cognitive processes like attention, decision making and emotional arousal.[8–12] It has also been used to measure the effects of anaesthetic agents, including nitrous oxide,[6,13] and on the intensive care unit to monitor critically-ill patients.[14]

The behavioural manifestations of alcohol intoxication and nitrogen narcosis are quite similar.[15] Pupillometry has, to our knowledge, never been used to assess the narcotic effects of nitrogen in hyperbaric air breathing.

Figure 1
Metrics of the pupil light reflex. Maximum dilation (max) is the maximal pupil diameter before the light flash. Minimum dilation (min) is the minimal pupil diameter after the light flash. Latency is the time between the onset of the light flash and the onset of the pupil contraction. Constriction is calculated as (max-min)/max

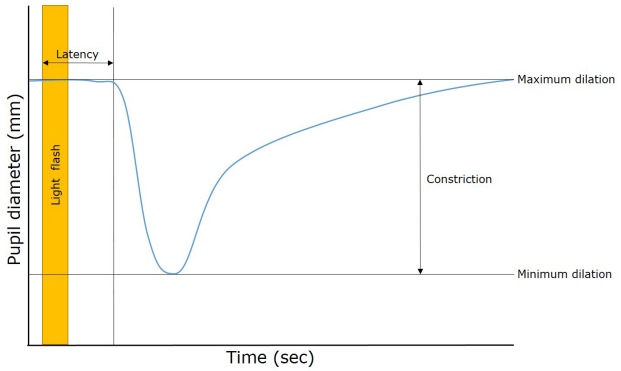
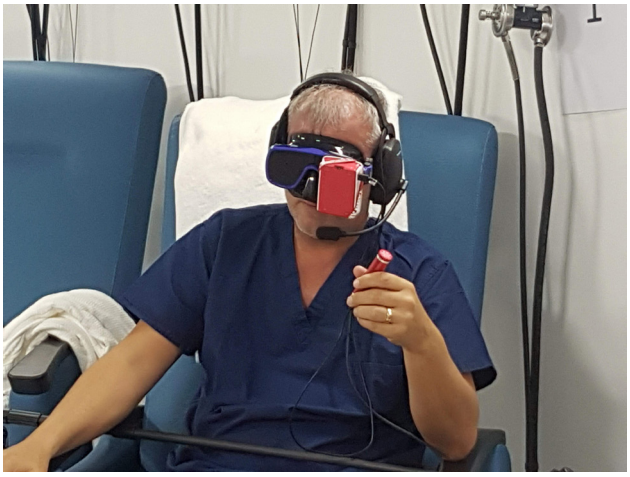


Figure 2
Participant wearing the pupillometry headset, while seated inside the hyperbaric chamber



Conducting narcosis experiments either during dives or inside a hyperbaric chamber are costly, labour and time intensive because of the incurred decompression and personnel involved to conduct hyperbaric experiments. In previous diving medical research, nitrous oxide has been used as a substitute to nitrogen narcosis.[16–19] Low dose nitrous oxide and hyperbaric nitrogen result in similar behavioural impairment measured with a range of psychometric tests.

The aim of this study was to evaluate the use of pupillometry for objective measurement of mild to moderate narcosis experienced during exposure to graded doses of nitrous oxide, and to hyperbaric air breathed at a pressure chosen to meet or exceed the maximum recommended depth for use of air during diving.

Methods

The study consisted of two experiments: pupillometry during air breathing at hyperbaric pressure; and during sea-level exposure to low-dose nitrous oxide.

TRIAL DESIGN AND PARTICIPANTS

The hyperbaric trial took place at the hyperbaric facility at Deep Dive Dubai, in March 2018. The study protocol was approved by the Dubai Scientific Research Ethics Committee of the Dubai Health Authority, United Arab Emirates (reference 10/2017_06).

The randomised, single-blind, cross-over, nitrous oxide trial took place at the Waikato Clinical School, University of Auckland, in July–August 2018. This study protocol was approved by the Health and Disability Ethics Committee, Auckland, New Zealand (reference 16/NTA/93) and was registered with the Australian New Zealand Clinical Trial Registry (ANZCTR) with Universal Trial Number U1111-1181-9722. These pupillometry measurements were a sub-study in a larger body of work investigating gas-induced narcosis that will be reported elsewhere.

Participants were eligible if they were certified, healthy adult divers, aged between 18 and 60 years and had normal visual acuity, either corrected or uncorrected. Participants were excluded if they were using recreational drugs, tobacco, psychoactive medication, excessive alcohol (> 21 standard drinks per week) or over five caffeine-containing beverages a day. All participants provided written informed consent. Participants abstained from any caffeinated drink on the measurement day, and from alcohol for at least 24 hours before the measurement. Participants had at least 6 hours of sleep the night before the measurement.

EQUIPMENT

A pupillometry device suitable for hyperbaric environments was built using an infra-red camera (PiNoir Camera V2, RS-components, the Netherlands) attached to a Raspberry Pi, a small computer (Raspberry Pi 3 Model B, RS-components, the Netherlands) mounted to a blacked-out scuba diving mask (Figure 2). The Pi was controlled by a web interface on a mobile device outside the hyperbaric chamber via a WiFi connection. The device took a picture of the eye, and the images were stored on a micro-SD card for off-line analysis. Unlike the proprietary pupillometer described below, the device built for the hyperbaric chamber did not measure pupillary reflexes in response to a light flash.

The pupil and iris diameter were determined from the stored images using Photoshop (Adobe, San Jose, CA, USA). Since distance between the camera and eye varied, the pupil diameter was corrected by using the fixed iris diameter, by calculating the ratio between them.

During the nitrous oxide experiments, a purpose-built PLR-200 Pupillometer (Neuroptics, Laguna Hills, CA,

Table 1
Demographic data for study subjects

Characteristic	Hyperbaric experiment *n* = 16	Nitrous oxide experiment *n* = 12
	Mean (range)	Mean (range)
Age (years)	35.3 (20–54)	36.3 (23–55)
Body mass index	23.8 (19.1–29.3)	26.3 (22.7–31.3)
Diving experience (years)	11.4 (1–29)	12.0 (1–36)
Dives	2,679 (15–10,000)	557 (20–1,500)
	n (%)	*n (%)*
Gender (male)	10 (62.5)	8 (66.7)
Certification:		
- Supervised diver	2 (12.5)	0
- Autonomous diver	2 (12.5)	6 (50.0)
- Dive Leader	1 (6.3)	0
- Dive instructor	11 (68.8)	6 (50.0)
- Technical diver	11 (68.8)	4 (33.3)
Experience (> 50 msw air)	8 (50.0)	7 (58.3)

USA) was used to record not just pupil diameter, but also pupillary reflexes in response to a 180 microwatt light flash administered for 154 milliseconds. Analysis of the recording was performed by the device to calculate the metrics mentioned below and stored for statistical analysis.

HYPERBARIC EXPERIMENTAL PROCEDURE

Sixteen divers volunteered to participate in this study. The chamber was a multi-place (10-person) rectangular hyperbaric chamber (Oxyheal 5000, National City, CA, USA). The lights inside the chamber were dimmed to 50% to minimise influence of ambient light on the pupillometry recording. Pupil size was then recorded inside the hyperbaric chamber at surface pressure immediately before compression. Participants were then compressed in groups of 2–4 persons to 608 kPa (equivalent to 50 msw depth) breathing environmental air. Upon arrival an acclimatisation period of five minutes was allowed to ensure onset of nitrogen narcosis before the recordings started. The infra-red photo of the eye was directly assessed for clarity and focus and if needed repeated to obtain one high quality image with the iris and pupil completely visible (not obscured by eye lids). After the measurements were finished, decompression was according to the US Navy decompression tables, including 100% oxygen breathing from 190 kPa to surface pressure.

NITROUS OXIDE EXPERIMENTAL PROCEDURE

Twelve divers volunteered to participate in this study. The experiment was conducted in a normobaric laboratory environment using a closed-circuit anaesthesia breathing loop (Vital Signs, Mexico) attached to an anaesthesia machine (S/5 Aespire, Datex-Ohmeda, Madison WI, USA). A normal scuba mouthpiece and a disposable anaesthetic antibacterial filter (Ultipor 25, Pall, Port Washington, NY, USA) constituted the interface with the participant. These two pieces of the breathing circuit were replaced for each participant which allowed use of the same breathing loop for multiple subjects. The nose was occluded with a nose clip. A gas sample line connected the mouthpiece filter to the anaesthetic monitor (GE Healthcare, Chicago, IL, USA) which measured the inspired fraction of oxygen, end-tidal pressure of carbon dioxide and end-tidal percentage of nitrous oxide breath by breath in real time.

The flow of nitrous oxide into the breathing circuit was titrated to maintain the desired end tidal percentage (20, 30 or 40%) of nitrous oxide. The remainder of the breathing gas was oxygen, with a continuous flow into the anaesthetic circuit of 2 $L \cdot min^{-1}$.

A baseline pupillometry measurement, before breathing nitrous oxide, was recorded while the participant was breathing 50% oxygen (balance nitrogen) on the circuit. Subjects then breathed nitrous oxide with an end-tidal level of 20, 30 and 40% in randomised order with a rest period of twenty minutes in between. The participants were blinded to the dose of nitrous oxide being administered. After 3–5 minutes of washing-in nitrous oxide at each dose, the device was placed in front of the right eye, covering the eye. While the camera was focussed on the pupil, the device applied one light flash to obtain the pupil light reflex. The measurement was repeated if, during the recording, the view of the pupil

Table 2
Pupillometry parameters in the nitrous oxide (N_2O) experiment (n = 12). Data are mean (SD)

Pupillometry parameter	Baseline	20% N_2O	30% N_2O	40% N_2O
Maximum dilation (mm)	5.5 (0.8)	5.4 (0.7)	5.4 (0.8)	5.6 (0.8)
Minimum dilation (mm)	3.9 (0.7)	3.9 (0.7)	4.0 (0.6)	4.2 (0.8)
Contraction (%)	-28.6 (5.6)	-27.8 (5.8)	-26.8 (3.8)	-24.8 (4.2)
Latency (ms)	237.5 (34.9)	237.5 (23.8)	239.2 (36.8)	225.8 (55.8)

Table 3
Mean differences with 95% confidence intervals (95% CI) in pupillometry parameters compared to baseline in pupillometry in the nitrous oxide (N_2O) experiment (n = 12). * indicates significant difference: $P < 0.05$

Pupillometry parameter	20% N_2O	30% N_2O	40% N_2O
Maximum dilation (mm)	0.1 (-0.2 to 0.4)	0.0 (-0.2 to 0.3)	-0.1 (-0.4 to 0.2)
Minimum dilation (mm)	0.0 (-0.3 to 0.3)	-0.1 (-0.3 to 0.2)	-0.3 (-0.6 to 0.0)*
Contraction (%)	-0.8 (-3.6 to 1.9)	-1.8 (-4.5 to 0.8)	-3.8 (-6.1 to -1.4)*
Latency (ms)	0.0 (-21.5 to 21.5)	-1.7 (-28.5 to 25.1)	11.7 (-29.7 to 53.0)

was blocked by a blink causing an inability to calculate the parameters. At the end of each exposure the nitrous oxide was washed-out of the anaesthetic circuit using oxygen with a fresh gas flow of 6 $L \cdot min^{-1}$.

OUTCOMES

The primary outcome was the absolute change in pupil diameter during exposure to nitrogen or nitrous oxide relative to baseline.[20] A secondary outcome of the nitrous oxide experiments was the pupillary light reflex, quantified by the percentage constriction (defined as maximum diameter (MAX) minus the minimum diameter (MIN) divided by MAX), and the latency (defined as the time between the light flash and the onset of the constriction) (Figure 1).

STATISTICAL ANALYSIS

Descriptive statistics were generated to characterise the study participants. All outcome measures were tested for normality and subsequently characterised by their mean and standard deviation (SD). Differences between baseline and intervention measures were analysed with paired t-tests and reported as mean difference with 95% confidence intervals. All data were analysed with SPSS version 25 (IBM, Armonk, NY, USA). Statistical significance was set at $P < 0.05$.

Results

HYPERBARIC NITROGEN EXPERIMENT

The 16 participants had between 15 and 10,000 dives, 11 were instructors or above, 11 were technical divers and half of the group had previous experience breathing air at 608 kPa or deeper (Table 1). The mean pupil-iris ratio did not change significantly while breathing air at 608 kPa (0.50 [SD 0.08]) compared to surface pressure baseline (0.51 [0.10]).

NITROUS OXIDE EXPERIMENT

The 12 participants had between 20 and 1500 dives, six were instructors or above, 11 were technical divers and seven had previous experience breathing air at 608 kPa or deeper (Table 1). The maximum pupil diameter (i.e., the resting state) and the latency did not change significantly during exposure to nitrous oxide at an end-tidal percentage of either 20, 30 or 40% compared to baseline (Table 2). However, the minimum diameter was significantly larger at the 40% nitrous oxide level (P = 0.047) compared to baseline with a mean difference of -0.3 mm, but not at lower concentrations. Pupil constriction was inhibited by nitrous oxide. This was only significant when baseline (-28.6%) was compared to nitrous oxide at an end tidal fraction of 40% (-24.8%) (P = 0.004) (Table 3).

Discussion

In this study pupillometry was insensitive to the narcotic effects of nitrogen in air breathed at 608 kPa, and low-dose nitrous oxide had only very small effects. Only during exposure to nitrous oxide at the end tidal = 40% level was there a small effect on the pupil reflex after a light flash. Since previous studies have clearly shown that similar nitrous oxide and hyperbaric air exposures cause significantly decreased cognitive performance[21] we conclude that pupillometry is unsuitable for monitoring the effects of this potentially dangerous degree of gas narcosis in divers.

Based on the effect previously reported in an alcohol study[7] we hypothesised that pupil diameter would be affected by

the exposure to air at 608 kPa. The doses used in the alcohol study (0.3 and 0.6 g·kg^{-1}) were approximately equivalent to 2 and 4 standard drinks for a 70 kg male and are in the same range as another alcohol study, in which oral alcohol (0.5 g·kg^{-1}) produced a similar reduction in cognitive performance as air dives to 40–45 msw.[15] Both alcohol and nitrogen have an impact on a wide variety of neural systems, and produce some similar effects. However, the lack of effect found with pupillometry in the present study using air breathing at 608 kPa, might indicate that alcohol and nitrogen influence the pupil feedback loop in the midbrain differently. A similar difference has been described, where the volatile anaesthetic agent ether affects pupil dilation, while other anaesthetic agents do not.[6]

The pupillary light reflex was slightly depressed when participants were exposed to sufficient inspired nitrous oxide to maintain an end tidal percentage of 40%. Others reported a similar result during general anaesthesia incorporating 60% nitrous oxide.[13] However, in the present study no light reflex depression was found at lower doses of nitrous oxide. Compared to air breathed at 608 kPa, an end tidal fraction of 40% nitrous oxide results in greater cognitive impairment.[18] This suggests that pupillometry is only sensitive to levels of impairment that exceed those likely to be produced in the air diving operational range of interest (surface to 50 msw; 101–608 kPa).

In addition, the small effect found at an end tidal nitrous oxide fraction of 40% was reliant on a light flash to measure the effect on the pupillary reflex. The need to expose the diver regularly to a light flash in order to assess the narcosis level, would also make it less suitable for continuous monitoring in a dive environment.

This study had a number of strengths, including exposure of subjects to both air at 608 kPa and nitrous oxide at surface pressure, the use of multiple concentrations of nitrous oxide to evaluate pupillometry response in a graded narcotic dose-response, and the use of two different pupillometry devices that showed similar results.

Some limitations need to be acknowledged. During the hyperbaric experiment only pupil dilation could be measured. Due to different face shapes, some divers had to push the mask to their face in order to get the camera in front of the eye causing some variation in the distance between eye and camera. We compensated for this, by measuring the iris diameter and calculating the ratio between the two of them. Narcosis is hard to measure objectively and accurately. In this trial there was no method available to monitor the narcotic effects systematically, and therefore it could be argued that perhaps these participants did not experience any narcosis However, impairment caused by breathing air at 608 kPa was assumed, given the extensive literature on nitrogen narcosis at this depth.[2,22] Similarly, we expected the nitrous oxide to have a cognitive impairment based on literature.[17] This was supported by informal observations of change in behaviour in all participants. Finally, we did not evaluate pupillometry in actual diving where other influences (such as hypercapnia) may enhance gas narcosis at any given depth. It is conceivable that pupillometry might be more sensitive under these circumstances.

Conclusion

In conclusion, pupillometry is insensitive to nitrogen narcosis in the operational depth range of interest (surface to 50 msw) and to the narcotic effects of low dose nitrous oxide. Hence, this method is not suitable as a monitoring method for gas narcosis in diving.

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Conflicts of interest and funding

Professor Mitchell is the Editor of *Diving and Hyperbaric Medicine* Journal and had no role in managing the review process or the decision to accept this manuscript. These matters were managed by the European (deputy) Editor, Dr Lesley Blogg. There were no other conflicts of interest.

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Deep anaesthesia: The Thailand cave rescue and its implications for management of the unconscious diver underwater

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Key words
Cave diving; Ketamine; Anaesthesia; Anesthesia; Unconsciousness; Full face mask; Equipment

Abstract
(van Waart H, Harris RJ, Gant N, Vrijdag XCE, Challen CJ, Lawthaweesawat C, Mitchell SJ. Deep anaesthesia: The Thailand cave rescue and its implications for management of the unconscious diver underwater. Diving and Hyperbaric Medicine. 2020 June 30;50(2):121–129. doi: 10.28920/dhm50.2.121-129. PMID: 32557413.)
Introduction: In 2018 12 children and one adult were anaesthetised before being extricated through over a kilometre of flooded cave in Thailand. Full face dive masks (FFMs) putatively capable of maintaining constant positive airway pressure (CPAP) were employed. Here we describe the anaesthetic intervention and investigate the CPAP capability of the FFM.
Methods: Pressure was measured inside and outside the Interspiro Divator FFM during 10 tidal and 10 vital capacity breaths in divers at the surface and submerged with the mask deployed on open-circuit scuba (10 divers); and a closed-circuit rebreather (five divers). Relative in-mask pressure was calculated as the difference between inside and outside pressures. We also measured the in-mask pressure generated by activation of the second stage regulator purge valve in open-circuit mode.
Results: When submerged in open-circuit mode the mean relative in-mask pressure remained positive in normal tidal breathing (inhalation 0.6 kPa [95% CI 0.3–0.9]; exhalation 1.1 [0.8–1.4]) and vital capacity breathing (inhalation 0.8 [0.4–1.1]; exhalation 1.2 [0.9–1.4]). As expected, the relative in-mask pressure was predominantly negative when used on closed-circuit with back mounted counter-lungs due to a negative static lung load. Mean in-mask pressure during purge valve operation was 3.99 kPa (approximately equal to 40 cmH_2O) (range: 2.56 to 5.3 kPa).
Conclusions: The CPAP function of the Interspiro Divator FFM works well configured with open-circuit scuba. This may have contributed to the success of the Thailand cave rescue. Caution is required in generalising this success to other diving scenarios.

Introduction

There is consensus among diving medicine experts that prolonged attempts to maintain a safe airway in an unconscious diver underwater carry a high risk of drowning. The corollary is that if a diver loses consciousness underwater, they should be returned to the surface expeditiously even if the breathing system mouthpiece is retained when using a half-mask, or despite use of a full-face mask that appears not to be flooded. In discussing the related conundrum of a diver with a significant decompression obligation who becomes unconscious underwater (due, for example, to a hyperoxic seizure), a recent guideline on the rescue of an unconscious diver at depth highlighted the difficulty of protecting the airway for any length of time and concluded: "*Any attempt to do so might result in drowning, which […] would likely represent a greater threat to life than decompression sickness (DCS) arising from a direct ascent*".[1]

Even where equipment specifically designed to enhance airway protection underwater is used, the same guideline maintained a conservative stance, stating: "*It is evident that under some circumstances the airway could be protected adequately to allow a period of decompression under ideal conditions, and this would be even more likely if the victim were using a full-face mask or a properly designed and deployed mouthpiece retaining device. Any decision to attempt this would depend entirely upon context, and it is reiterated that the path of least risk in the majority of circumstances will be to bring the victim directly to the surface*".[1]

Concerns about airway management in an unconscious diver are not limited to scenarios in which unconsciousness ensues during a dive. There are other plausible scenarios in which an unconscious person may need to be taken underwater. For example, a cave explorer who is rendered unconscious by an accident or medical event in a dry section of a cave may subsequently need to be extricated through an underwater sump. Another improbable but now famous example is detailed in this paper.

While it has been suggested that mouthpiece retaining straps may be effective in preventing drowning after loss of consciousness underwater,[2] discussion of this issue has largely taken place in the absence of relevant evidence. This unsatisfactory situation was recognised at the 2012 'Rebreather Forum Three' meeting where a consensus of participants identified the study of airway protection by mouthpiece retaining straps and full-face masks as research priorities.[3] At the time this consensus was published there was little confidence that the question would ever be addressed in an experimental setting given the obvious ethical concerns with testing airway protection in immersed unconscious humans.

Remarkably, in 2018 an international rescue team was effectively forced into conducting such an experiment in attempting to save the lives of 12 boys and their soccer coach who had become trapped several kilometres inside a flooded cave in Thailand. It is a matter of record that all 12 boys and their coach were successfully brought out through approximately 2.6 km of cave, of which approximately 1.1 km was fully flooded, whilst anaesthetised and wearing full face masks.[4] We use the term 'anaesthetised' intentionally in this setting because the boys were asleep, unresponsive to voice, and not exhibiting purposive movement. In the aftermath of this event it seems appropriate to revisit the issue of managing the unconscious diver underwater, and to address the question of whether the success of this rescue implies a change in the previously quoted 2012 guidelines is appropriate.

The full-face mask (FFM) used in the Thai cave rescue was an Interspiro Divator Full Face Mask (Interspiro, Täby, Sweden), chosen for several reasons including a function designed to maintain a degree of constant positive pressure (referred to by the manufacturer as "*safety pressure*"), throughout the respiratory cycle. In medical terms, this effectively translates to a form of constant positive airway pressure (CPAP) and is henceforth referred to as such. CPAP could serve both to discourage water ingress, and to splint the airway open in an unconscious subject. We found no data available in the public domain which confirmed CPAP is maintained during use of the Interspiro mask, and given the important implications to practice around unconscious diver management, this was investigated in the present study.

This paper begins with an account of the anaesthetic administered to those rescued in Thailand. One of the present authors (RJH) was the anaesthesiologist who performed the initial anaesthetic procedures, while another author (CJC) was prominently involved in supplementary dosing during the rescue. It continues with the methods and results of the Interspiro FFM CPAP-function evaluation. The nature of both the anaesthesia and breathing apparatus are highly relevant to the rescue's implications for management of unconscious divers underwater. These implications are subsequently discussed.

Anaesthesia in the Thailand cave rescue

Reporting of this de-identified rescue narrative and outcome information was deemed out of scope by the New Zealand Health and Disability Ethics Committees but approved by the Chief Medical Commander of the Thailand Cave Rescue.

The relevant author (RJH) is an Australian consultant anaesthetist with over 30 years experience in cave diving. He has a longstanding professional and personal interest in search and rescue operations including some experience in training other cave divers in the practical aspects of through-sump rescue (a 'sump' is a completely flooded section of cave tunnel bounded at each end by areas of dry cave). RJH was called by the British cave divers in Thailand on 05 July 2018, 12 days after the Thai soccer team entered the cave and three days after they were found. The author and his regular dive partner (CJC) flew to Thailand that same day as a small Australian Medical Assistance Team (AUSMAT).

It rapidly became apparent that the best chance of a successful rescue lay in the option of anaesthetising the 13 stranded team members (including the coach). All other viable alternatives had been tried or considered, then eliminated. There were six sumps to traverse and it was felt by the rescuers that any attempt to extricate the team without anaesthesia would result in panic, and the subsequent drowning of either the boy and/or the rescue diver. Although the depths were trivial (≤ 4.5 metres' fresh water) diving conditions were hazardous with moderate strength flow of opaque turbid water, multiple tight restrictions, 'line traps' (places which permit the guiding line followed in the cave to pass, but are too narrow for a diver to follow), and a predicted egress duration of three hours.

Whilst developing the anaesthesia plan, RJH consulted widely with other specialists including paediatric anaesthetists and psychiatrists. With the exception of Edgar Pask during the Second World War,[5] no precedent could be found for immersing anaesthetised humans. Numerous medications were considered including benzodiazepines, clonidine and chloral hydrate. Ketamine hydrochloride was the obvious final choice due to its significant track record in austere environments and developing nations. It has a wide therapeutic index making it forgiving in less skilled hands and can be administered by multiple routes including by intramuscular injection. It has the advantage of maintaining spontaneous respiration in appropriate

dosage, with anaesthetised patients protecting their own airway better than with other sedative drugs and anaesthetic agents.[6] It offers good cardiovascular stability in volume replete patients, and may offer advantages in protecting from hypothermia compared to alternative agents due to the sympathetic activation of peripheral vasoconstriction.[6] Disadvantages of ketamine in the proposed setting included increased salivation, dysphoria and unpleasant emergence phenomena, and difficulty determining the end point of successful anaesthesia, especially by lay persons.

A final plan for the anaesthesia was derived. It was known that the weights of the rescuees were between 30 and 65 kg. For simplicity, especially for the non-medical cave divers who would need to supplement the initial loading dose of ketamine with 'top-up doses' further down the cave, the rescuees were classified as 'large' (approximately 50 kg) or 'small' (approximately 40 kg). Any individual less than 45 kg would be treated as 'small', and anyone over 45 kg would be treated as 'large'. Whilst RJH was able to adjust loading doses of ketamine for the initial induction of anaesthesia at the back of the cave, the other cave divers were instructed to simply give a pre-prepared 'small' (100 mg) or 'large' (125 mg) intra-muscular top-up dose to the rescuees when required (typically indicated by resumption of purposive movement). This meant that some of the rescuees were slightly under- or over-dosed (on a weight basis) during extrication, but this approach avoided the considerable risk of mis-dosage by cave divers trying to tailor dosing more precisely.

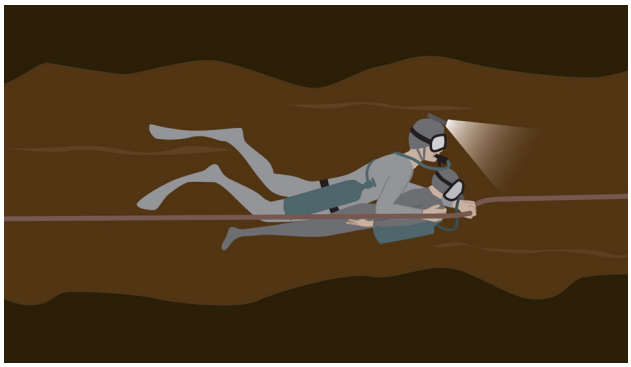
The operation began 15 days after the team entered the cave, and took place over three days (8–10 July) with four individuals rescued on days one and two, and five on day three. On each rescue day, those selected for rescue were fasted from 0600 with the knowledge the rescue team would be in place at approximately midday. First, the child was given 0.5 mg oral alprazolam for anxiolysis and to decrease the risk of awareness and dysphoria. Larger doses or longer acting benzodiazepines were avoided due to concerns around respiratory depression. Over the ensuing 15 minutes the child was dressed in their wetsuit (if not already on) and a horse collar style buoyancy compensation device. Elastic bungees were placed around the child's chest and pelvis which would later be used to retain a front mounted scuba cylinder.

The child then descended the 15 m muddy slope to the waiting anaesthetist (RJH) who administered an intramuscular injection of atropine (as an anti-sialagogue) in the anterolateral thigh with a 23 g hypodermic needle, penetrating the child's wetsuit. Atropine dosage was 20 $mcg \cdot kg^{-1}$ based on a body weight of either forty or fifty kilograms. Ketamine 5 $mg \cdot kg^{-1}$ was then injected into the contralateral thigh with body weight estimated from recently recorded weights with allowance for loss of several kilograms over the starvation period. Once it was clear that a deep level of general anaesthesia had been achieved (the child was unresponsive) with spontaneous respiration maintained, a rescue diver assisted RJH in placing the FFM on the child. The FFM straps were tightened to a degree perceived to exceed that which would normally be comfortable for a diver. The scuba cylinder providing gas to the mask was attached frontally to the two bungee cords placed earlier. The cylinder contained 80% oxygen and 20% nitrogen; the high oxygen fraction chosen to prolong safe apnoea time if the latter were to occur, with the smaller nitrogen fraction (in theory) reducing the extent of any absorption atelectasis. Additionally, the maximum inspired partial pressure of oxygen (PO_2) of 1.2 atmopheres absolute at the greatest depth was within acceptable limits for the expected exposure duration to avoid both pulmonary and cerebral oxygen toxicity. The FFM CPAP function automatically engaged when breathing commenced.

With the mask and cylinder in place and respiration established, the child was turned to the prone position with their faced immersed in the water. Occasionally at this point, or earlier when the mask was first placed, the child appeared to breath-hold / become apnoeic. On several such occasions a positive pressure breath was given by briefly pressing the FFM second stage regulator purge valve, which appeared to stimulate breathing. Spontaneous respiration with subjectively adequate tidal volumes was monitored by observing the volume of expiratory bubbling from the exhaust valve on the mask. After approximately thirty seconds, the child was sat up again to confirm the FFM remained dry. This was repeated two more times until both RJH and the rescue diver were satisfied with the mask seal. In the prone and immersed position, the child's hands were then clipped together with cable ties and a carabiner behind his back, and the ankles were loosely bound with more bungee cord. These restraints were performed both to prevent entanglement of the limbs on cave projections, and to stop the child pulling at the FFM should the anaesthesia wear off unexpectedly.

After performing final checks, the rescue diver submerged and left the chamber with the child positioned face down and beneath the rescuer as represented in Figure 1. Each traverse out to the dive base (from which point no further diving was required) would take approximately 3 hours on rescue day one, but only around 90 mins by day three as systems and procedures improved. On-route, support divers stationed at various chambers within the cave offered assistance to the rescue diver. One author (CJC) with his extensive veterinary experience of administering ketamine, was able to make an initial medical assessment in one of the between-sump dry chambers further out, and help supervise the other divers perform their first injections. Each child received 3 or 4 'top-up doses' of ketamine during the egress, successfully given by the cave divers when it was judged the child was rousing. These doses were administered in the cave sections between sumps, often in very difficult conditions (such as floating in the streamway, trying to deploy the medications whilst not losing hold of the child).

Figure 1
Depiction of the approximate configuration of the rescue diver and unconscious diver during passage through the flooded sections of cave



On reaching the chamber from which no further diving was required, a limited medical assessment was performed by a Thai Navy underwater medicine team and a full medical assessment was performed in the Thai Army field hospital outside the cave. Table 1 shows the initial observations of the rescuees measured during their brief stay in this field hospital. A protocol was developed which addressed airway, breathing, circulation, and hypothermia management (ABC+H).[7] Hypothermia was identified on day one as a critical issue. The water and air temperature in the cave was 23°C.

Resuscitation roles (ABC) were assigned to an anaesthesiologist, respirologist, and paediatric cardiologist, respectively. The hypothermia management was formalized and assigned to the anaesthesiologist (author CL). It is noteworthy that case nine (Table 1) exhibited severe hypothermia (29.6°C as measured by tympanic membrane temperature) without shivering. He was rewarmed before being transferred by air ambulance to Chang Rai hospital and suffered no adverse consequences. The management of hypothermia in this event is discussed in more detail elsewhere.[7]

At the field hospital, 12 of the subjects required no airway management beyond simple airway manoeuvres and supplemental oxygen via non-rebreather mask. One boy suffered a brief episode of laryngospasm which was managed with a bag-valve-mask device. Remarkably, none of these subjects, who spent considerable time underwater whilst unconscious, drowned or aspirated significant amounts of water. Three showed chest X-ray changes consistent with minor aspiration or infection. None of those rescued recalled any events between the induction of anaesthesia and emerging from the cave which is further evidence of a deep level of anaesthesia.

Methods

The study evaluated the pressure changes inside the Divator FFM with the safety pressure feature (Interspiro, Täby, Sweden) relative to the ambient water pressure during the respiratory cycle in immersed and fully conscious divers. The protocol was approved by the University of Auckland Human Participants Ethics Committee (Reference 022486). All participants provided written informed consent.

TRIAL DESIGN AND PARTICIPANTS

This was an observational cohort study conducted in the Exercise Physiology Laboratory at the University of Auckland during March 2019. Participants were certified healthy divers aged 18 to 60 years. Five rebreather divers and five open circuit divers were recruited. Their current health was assessed using the Recreational Scuba Training Council (RSTC) screening questionnaire at the beginning of their visit. All participants performed a spirometry test. Forced vital capacity (FVC) and the forced expiratory volume in one second (FEV_1) were measured, from which we calculated the FEV_1/FVC ratio (%).

EQUIPMENT

A circular frame pool (3 m diameter and 0.76 m depth) was installed in the laboratory and filled with fresh water (~20°C) to a standardised depth of 60 cm. The same model of FFM as used in the Thai cave rescue (specified above) was studied in its stock configuration for operation with an open circuit scuba pressurised gas supply, and in modified configuration (described below) for operation on a closed-circuit rebreather.

In operation with an open circuit gas supply the low-pressure hose on the FFM second stage demand valve was connected via a 2 m hose to a first stage scuba regulator (Apeks, Blackburn, England) on a pressurised scuba cylinder (as was done in Thailand). Air was supplied to the mask at an intermediate pressure of 9 bar (0.9 MPa) when the cylinder was pressurised to 200 bar (20.3 MPa). The scuba cylinder was situated adjacent to the pool. Non-compliant pressure tubing was potted through a bung fitted to the communication port of the mask to measure the pressure inside the mask via a physiological pressure transducer (MLT844, AD Instruments, Dunedin, New Zealand). Another non-compliant tube was affixed to the left side of the mask at a position we estimated to correspond with the mask centroid in a horizontal face-down diver, to measure the external water pressure via a second (identical) physiological pressure transducer. The pressure transducers were calibrated with a manometer (PM-9100HA, Lutron Electronic Enterprise, Taipei, Taiwan) using two-point calibration against atmospheric pressure and atmospheric pressure + 6 kPa. Reference atmospheric pressure was measured using a barometer (GPB330, Greisinger Electronic, Regenstauf, Germany).

In operation with a closed-circuit rebreather, the FFM second stage regulator was detached and the hole sealed with a plastic blinding plug that was 3D-printed locally. The mouthpiece valve assembly of an Inspiration Evolution

Table 1

Initial observations made at the cave field hospital. The salient feature of these data in relation to the issue of airway management is the lack of any evidence of gross water aspiration. T0 = temperature on arrival. T1 = temperature on departure for Chang Rai Hospital. NIBP = non-invasive blood pressure. RR = respiratory rate. SpO_2 = peripheral oxygen saturation (breathing supplemental oxygen via a non-rebreather mask). N = normal. RLL = right lower lobe. LLL = left lower lobe

Patient	Temp T0	Temp T1	Heart rate	NIBP	RR	SpO_2	Chest X-ray
1	35.0	36.4	78	128/78	14	97	N
2	34.0	34.8	96	130/90	32	100	RLL opacity
3	35.5	35.5	75	137/83	24	99	N
4	35.0	34.9	78	128/78	14	97	R hilar opacity
5	37.5	35.6	75	138/60	18	98	N
6	36.4	34.9	100	140/104	20	100	N
7	35.5	36.4	95	134/98	16	87	N
8	34.4	35.5	71	126/97	22	100	N
9	29.6	36.4	96	172/124	12	100	N
10	33.8	36.9	110	132/62	16	100	LLL opacity
11	33.4	38.5	102	128/100	18	94	N
12	32.9	37.0	82	140/100	20	100	N
13	34.0	36.9	89	135/81	12	96	N

Plus rebreather (Ambient Pressure Diving, Helston, Cornwall) with back mounted counter-lungs was attached via a 3D-printed plastic adaptor to the FFM communications port. A non-compliant tube penetrated the adaptor and was connected to a pressure transducer (as above) to measure pressure in the mask. The positioning of the external pressure sensor tubing on the mask was as described above. The rebreather was worn on the subject's back and operated with air diluent with an inspired PO_2 set point of 0.7 atmospheres absolute (atm abs) (70.9 kPa). In keeping with normal rebreather diving practice, participants were instructed to maintain 'minimum loop volume'; that is, sufficient gas in the loop such that respiratory excursions just avoid emptying the counter-lungs during inhalation.

Voltage signal from the two transducers was sampled at 1 kHz using a Powerlab 16/35, acquired and filtered using a 10 Hz low pass filter via LabChart data acquisition software (AD Instruments, Dunedin, New Zealand).

EXPERIMENTAL PROCEDURE

All 10 participants completed the experiment using the FFM connected to the pressurised open circuit scuba gas supply, and five divers trained on rebreathers also completed the experiment using the FFM connected to the closed-circuit rebreather.

For each equipment configuration, the experiment was conducted during spontaneous breathing at the surface and while submerged. For the surface measurements participants knelt upright with their face and all diving apparatus above the water. For the submerged measurements, they lay face down, horizontal in all planes, on the bottom of the pool with their face at ~50 cm depth. Under each of these conditions, resting participants were asked to breathe at normal comfortable tidal volume and frequency for 10 breaths ('normal breaths'), then take 10 vital capacity breaths. Finally, whilst submerged and in open circuit mode only, participants were asked to depress the purge button on the FFM second stage regulator for a burst of approximately two seconds.

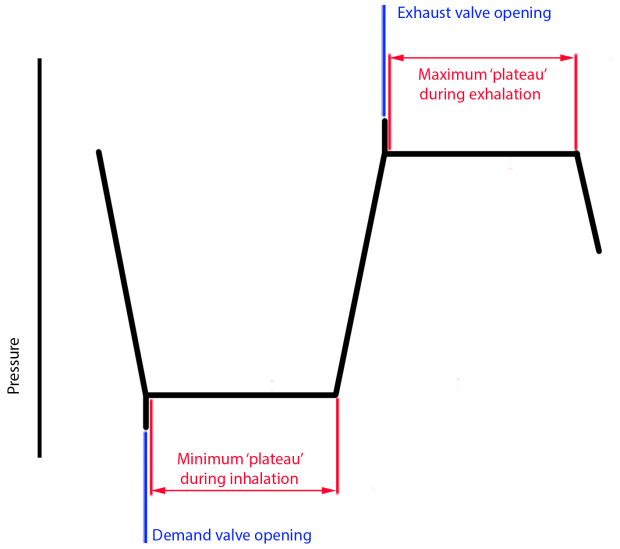
Lastly, we assessed the ability to create a leak across the mask flange seal using only auto-manipulation of the facial muscles.

OUTCOME MEASURES

The primary outcome measure was the minimum and maximum pressures inside the mask relative to ambient pressure at the mask centroid ('relative in-mask pressure') during the inhalation and exhalation phases of the respiratory cycle respectively. Because of the differing characteristics of the pressure vs time curves, the approach to calculating these pressures differed between open- and closed-circuit experiments.

In the open-circuit experiments breathing typically produced identifiable pressure 'plateaus' during inhalation and exhalation. The start of the 'minimum plateau' during inhalation began just after the negative pressure spike preceding demand valve opening and was considered to last until the beginning of the steep pressure rise. The 'maximum plateau' during exhalation began just after the positive pressure spike preceding exhaust valve opening and was considered to last until the beginning of the steep pressure fall (Figure 2). During these plateaus the relative in-mask pressure was calculated automatically at the same rate of the data sampling (see earlier) by subtracting the ambient

Figure 2
Schematic depiction of inhalation and exhalation pressure 'plateaus' during use of the FFM with open circuit scuba equipment



pressure from the in-mask pressure. All relative in-mask pressures captured during the inhalation and exhalation plateaus over 10 breaths were averaged to derive the average minimum and maximum relative in-mask pressures respectively. These minimum and maximum means for individual subjects were then averaged across the 10 subjects to give the average minimum and maximum relative in-mask pressure for the particular condition.

In the use of the rebreather the valve opening spikes were absent. The pressure-time curve was more sinusoidal and typically devoid of identifiable plateaus. We therefore took the simpler approach of recording absolute peak (exhalation) and trough (inhalation) pressures over 10 breaths in each subject in calculating the average minimum and maximum relative in-mask pressure for the particular condition, as described for the open-circuit experiments above.

The peak pressure generated during the use of the purge button at the surface and at depth was recorded and means were calculated from pooled data.

Mask leaks during facial muscle manipulation were described in a qualitative manner.

STATISTICAL ANALYSIS

Means and standard deviations (SD) were calculated for data describing subject characteristics. Means and 95% confidence intervals were calculated for the relative in-mask pressures measured under all conditions and equipment configurations. Data were tested for normality, all pressures were normally distributed. One-sample *t*-test, with α set at 5%, was used to test the hypothesis that the mean relative in-mask pressure in the various conditions was different from zero.

Table 2
Characteristics of the FFM study participants. Note that the CCR group is a subset of the total group. Data are mean (SD) unless otherwise indicated. BMI = body mass index. CCR = closed circuit rebreather group. FEV_1 = forced expiratory volume in one second. FVC = Forced vital capacity

Parameter	Total *n* = 10	CCR *n* = 5
Age (years)	32.6 (10.5)	34.0 (7.5)
Female gender	*n* = 4	*n* = 1
Beard	*n* = 3	*n* = 3
Small/long face	*n* = 2	*n* = 0
BMI ($kg \cdot m^2$)	24.6 (4.0)	25.9 (4.8)
FEV_1 / FVC	0.84 (0.12)	0.82 (0.11)

Results

Participant characteristics are described in Table 2. Mean relative in-mask minimum and maximum pressures during inhalation and exhalation in open and closed-circuit equipment configurations are given in Table 3 (surface measurements) and Table 4 (measurements while submerged).

The key finding was that when operated underwater with a pressurised open-circuit gas supply, the Divator FFM with the 'safety pressure' feature maintained positive pressure inside the mask relative to the surrounding water, even during maximal inhalations (Table 4 and Figure 3). This validates the manufacturer's claim of an effective CPAP function. Predictably, CPAP was not evident when the mask was operated with a non-pressurised closed-circuit gas supply. Indeed, when submerged in this configuration the mean relative in-mask pressures remained negative throughout the respiratory cycle, even during exhalation. The latter finding, not seen during operation at the surface where exhalation against the resistance of the circuit generated positive pressures (Table 3), almost certainly reflects the negative static lung load that exists when an immersed diver is horizontal and face-down during use of a rebreather with back mounted counter-lungs.[8]

Use of the purge button resulted in a mean in-mask pressure of 3.99 kPa (approximately equal to 40 cmH_2O) (range: 2.56 to 5.3 kPa), during submergence. At the surface it delivered on average a mean pressure of 3.05 kPa (~31 cmH_2O) (range: 1.20 to 5.31 kPa).

All participants were able to create leaks with purposefully excessive facial movements. No water ingress was noted in open circuit configuration (where relative in-mask pressure remained positive and outward bubbling was the principal manifestation of a leak), while some water entered the mask during the closed-circuit trials. Five participants had a small but constant leak of bubbles leaking out of the mask in open circuit configuration, all with potential explanations: two having small/long faces; and three having a beard.

Table 3
Mean relative in-mask pressures during inhalation and exhalation in open and closed-circuit equipment during surface measurements. CI = confidence interval. VC = vital capacity

Breathing mode while submerged	Inhalation In-mask relative pressure kPa [mean (95% CI)]	*P*-value	Exhalation In-mask relative pressure kPa [mean (95% CI)]	*P*-value
Open circuit				
Normal breaths	0.4 (-0.7 to 1.6)	0.42	0.3 (0.3 to 0.4)	< 0.001
VC breaths	-0.1 (-0.1 to 0.0)	0.004	0.3 (0.3 to 0.4)	< 0.001
Closed circuit				
Normal breaths	-0.6 (-1.3 to 0.1)	0.06	0.7 (0.3 to 1.1)	0.009
VC breaths	-1.7 (-4.1 to 0.6)	0.11	1.0 (0.7 to 1.3)	0.001

Table 4
Mean relative in-mask pressures during inhalation and exhalation in open and closed-circuit equipment configurations during submerged measurements. CI = confidence interval. VC = vital capacity

Breathing mode while submerged	Inhalation In-mask relative pressure kPa [mean (95% CI)]	*P*-value	Exhalation In-mask relative pressure kPa [mean (95% CI)]	*P*-value
Open circuit				
Normal breaths	0.6 (0.3 to 0.9)	0.002	1.1 (0.8 to 1.4)	< 0.001
VC breaths	0.8 (0.4 to 1.1)	< 0.001	1.2 (0.9 to 1.4)	< 0.001
Closed circuit				
Normal breaths	-1.9 (-2.9 to -1.0)	0.005	-0.6 (-1.4 to 0.2)	0.09
VC breaths	-2.0 (-2.9 to -1.0)	0.004	-0.3 (-1.1 to 0.5)	0.34

Discussion

The most striking aspect of the Thai cave rescue narrative is that 12 unconscious non-diver children and one adult were rescued through 1.1 km of flooded, tortuous cave passage in near zero visibility, and none drowned. These were not the 'ideal conditions' in which 'a period' of underwater airway management might be achievable according to the current rescue guideline.[1] Indeed, the Thai cave scenario represented the antithesis of 'ideal conditions' and seen through that lens, the success of the operation was remarkable. One obvious question arising is 'what are the implications of this event for recommendations about attempting airway management in an unconscious diver underwater'? In particular, should related guidelines be less discouraging of managing an unconscious diver underwater where lessons learned from the Thailand cave rescue are applied, and where there are compelling reasons to attempt it?

Any effort to address these questions must take account of the degree to which the factors associated with the success in Thailand are generalisable to other diving situations in which loss of consciousness may occur. These factors are enumerated and discussed in that context below.

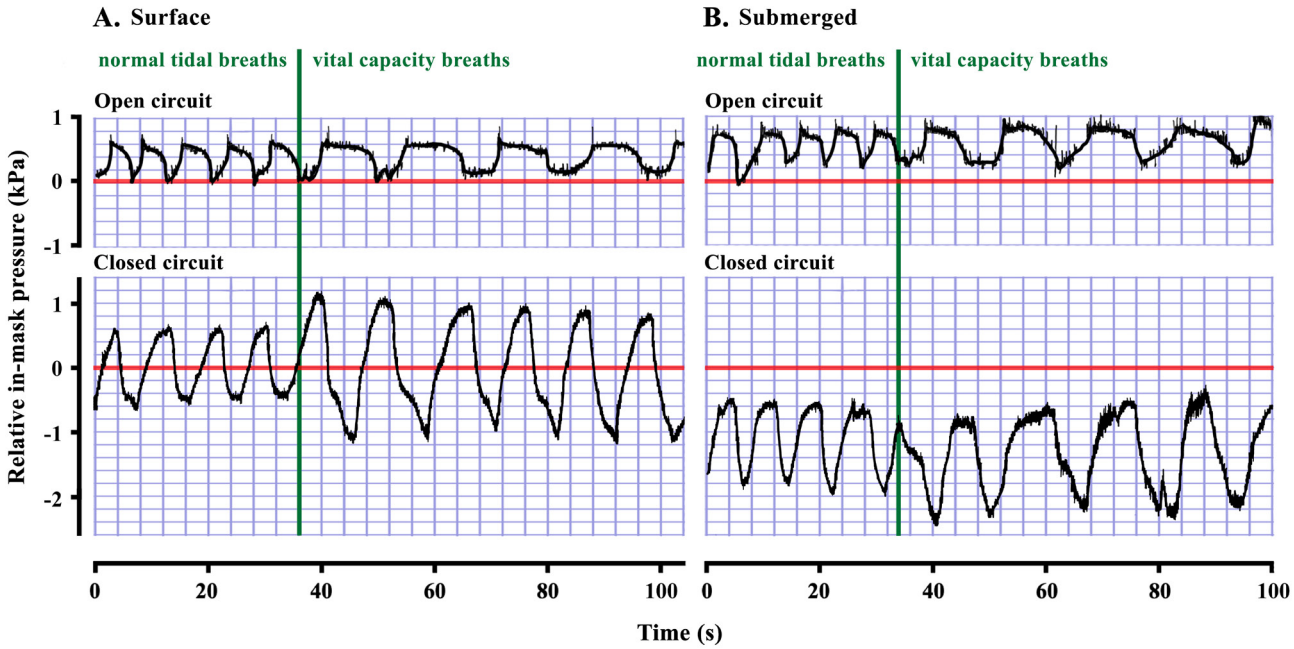
First, in the Thailand cave rescue, unconsciousness was induced with a drug rather than by a medical event such as a seizure, and the unconscious state in these respective settings may be qualitatively and quantitatively different. The principal anaesthetic drug (ketamine) was explicitly chosen for certain specific properties. It is effective when administered intra-muscularly, and the relatively long time-course of its action when absorbed from an intramuscular depot suited the needs of this scenario. Ketamine is sympathomimetic and is much less likely than other intravenous anaesthetic agents to cause hypotension.[6] It was therefore suited to this scenario in which no haemodynamic monitoring was available or possible. Perhaps most importantly, ketamine preserves respiratory drive and airway protective reflexes to a greater extent than other anaesthetics.[6] This was vital in a situation in which there was no means of continuously assisting ventilation, and in which it was possible that water could contaminate the airway if the FFM leaked.

It is difficult to evaluate the extent to which the depth of anaesthesia induced by ketamine administered to the rescuees equates with the depth of unconsciousness that, for example, would follow a hyperoxic seizure. It is clear from the earlier account that they were effectively anaesthetised (non-responsive and not moving) at least at the start of the diving transit. Nevertheless, the potential qualitative and quantitative differences between ketamine anaesthesia and unconsciousness induced by other events that might occur in diving potentially confound judgements on the generalisability of the Thai rescue outcomes.

Second, the Interspiro FFM with the safety pressure

Figure 3

Representative pressure – time waveforms showing relative in-mask pressure where zero represents the ambient pressure at the mask centroid under the various experimental conditions



feature investigated in this study appears to have prevented significant aspiration of water in the rescuees. The results presented here confirmed that when operated in open circuit mode the mask will provide a small degree of CPAP throughout the respiratory cycle. However, this was not a comprehensive evaluation. We only tested one mask, and the efficacy of the CPAP function was only tested at shallow depth in the prone horizontal position. It may behave differently in other attitudes. For example, allowing the neck to extend into a face up position in a prone subject could place the mask centroid several centimetres shallower than the expiratory diaphragm, and the degree of CPAP relative to surrounding water pressure would be correspondingly greater. Users of this mask in cave rescue training scenarios have noted that the buoyancy of this FFM tends to promote this attitude change in a prone subject.

The extent to which CPAP in the FFM influenced the positive outcome in Thailand is unknown but it is a highly plausible contributor. This can be inferred from the fact that our subjects could provoke water ingress through facial movement when the mask was used without CPAP on a rebreather, but not when used with CPAP in open circuit mode. Therefore, in considering generalisability of the Thailand outcomes, it must be observed that these FFMs do not provide CPAP to a prone diver in a horizontal attitude using a rebreather with back-mounted counter-lungs. We acknowledge that a prone diver using a rebreather with front-mounted counter-lungs or a supine diver using back-mounted counter-lungs (conditions not tested here) may experience a positive static lung load, and therefore CPAP. With these facts in mind, it seems plausible to conclude that the efficacy of an FFM – rebreather combination in preventing water ingress and aspiration in an unconscious diver may depend on the positions of the diver and the rebreather's counter-lungs. In some combinations a FFM may not be as effective as they appeared in Thailand. Our results also suggest that FFM efficacy in preventing leaks can also be degraded by facial shape and beards. Leaks could be a particular disadvantage in use of a rebreather with a small gas supply, but less problematic in use of open circuit apparatus with a larger gas supply.

Third, the Thailand rescue was conducted with the rescuees breathing from open circuit scuba. The consequent exhalation of bubbles with each breath facilitated monitoring of the children's respiration by the rescuing divers. In contrast, another potential problem with managing an unconscious rebreather diver underwater is that it could be difficult for the rescuer to tell if the victim is actually breathing because there are no bubbles. Direct observation of visible counter-lungs might be the only form of monitoring, and this is not possible in most rebreather models.

Fourth, the young age and lean body habitus of the rescuees may have been an advantage in preventing airway obstruction when they were unconscious. Irrespective of whether the airway remains dry it is possible that mechanical airway obstruction could occur more easily if the head were not held in an ideal position during rescue of an adult diver, particularly if they were obese. If the rescuee were held in a horizontal prone position, the positive buoyancy of a FFM may help keep the airway open by lifting the head into a face-up position.

Finally, the skill and expertise of the divers who managed each boy through the underwater sections of the cave, and the time afforded them to plan the approach, were surely contributors to the positive outcome. The rescue of unconscious divers underwater in other settings may involve less experienced and skilled divers who find themselves in an unexpected unplanned situation with extremely high levels of stress.

It is clear from the above there are several considerations which potentially confound interpretation of the Thailand cave rescue in revising general recommendations for managing unconscious divers underwater. We do not believe the positive outcomes in Thailand challenge the fundamental principle that the safest place for an unconscious diver is on the surface. Nevertheless, the Thailand event and the FFM investigation reported here do provide a degree of enhanced confidence that a FFM could prevent drowning during rescue of a diver who unexpectedly becomes unconscious underwater, but who continues to breathe. This would be especially relevant to a scenario where direct ascent to the surface is either not possible or is hazardous, such as an unconscious diver being swum out of a cave, or where completion of some decompression time might prevent fatal decompression sickness. To be in a position to accrue these advantages, divers would need to adopt an FFM for all their diving (so that they are wearing one if an adverse event were to occur), and this opens up legitimate debate about their cost, complexity, potential hazards and the need for proper training in their use.

We note a particular caveat in relation to divers who are not only unconscious, but also not breathing. The apparent success of using the purge button to provide a positive pressure stimulus to breathing in the Thailand cave rescue should not be interpreted as evidence that sustained positive pressure ventilation is feasible underwater using this approach. Our data show the highest relative in-mask pressure generated on purge button activation was high enough (54 cm H_2O) to potentially cause pulmonary barotrauma. The purge button is not designed to facilitate positive pressure ventilation, and must be employed cautiously if used for this purpose. Moreover, a diver in respiratory arrest may also be in cardiac arrest, and in that setting their only hope for survival is proper cardiopulmonary resuscitation at the surface.

Leaving aside the issue of whether FFMs are practical or desirable for general use, the Thailand cave rescue and the findings of the present study strengthen the argument for elective use of an FFM (particularly in an open-circuit CPAP mode) during procedures carrying an enhanced risk of loss of consciousness due to oxygen toxicity (such as in-water recompression).[9] Similarly, it seems justified to conclude that if faced in future with a similar situation to the Thailand cave rescue (such as rescue of an injured unconscious caver through a sump), the methods employed by the Thailand rescue team could be utilised again with a reasonable expectation of success. Such a conclusion would have seemed very implausible before the Thailand event.

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Anxiety impact on scuba performance and underwater cognitive processing ability

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Abstract
(Tsai F-H, Wu W-L, Liang J-M, Hsu H-T, Chen T-Y. Anxiety impact on scuba performance and underwater cognitive processing ability. Diving and Hyperbaric Medicine. 2020 June 30;50(2):130–134. doi: 10.28920/dhm50.2.130-134. PMID: 32557414.)
Introduction: Anxiety is a substantial consideration in scuba diving and may influence a diver's performance and cognitive activities. This study aimed to simultaneously observe the effect of anxiety trait on actual diving performance and underwater cognitive processing ability.
Methods: Twenty-seven scuba divers completed the STAI-T component of the State-Trait Anxiety Inventory, and were subdivided into two groups on the basis of trait anxiety scores ≥ 39 and < 39. Scuba diving performance was measured in a pool. The completion time of four standardised scuba skills was recorded by a diving instructor. The correct completion rate and response time for a cognitive function assessment (number-Stroop test) were measured both on land ('dry') and underwater at 5 metres' fresh water.
Results: Anxiety trait was associated with prolonged mask clearing: mean completion time 7.1 (SD 3.2) s vs. 10.8 (5.4) s in low and high anxiety trait divers respectively ($P = 0.04$). Low (vs high) anxiety trait divers had reduced response times for the number-Stroop test: 49.8 (3.0) s vs. 53.3 (5.4) s ($P = 0.04$) dry, and 64.4 (5.0) s vs. 72.5 (5.5) s ($P < 0.01$) underwater. Performance of other skills was not significantly affected by trait anxiety nor correlated with the number-Stroop test results.
Conclusions: Personal anxiety trait prolongs mask clearing and underwater cognitive processing ability but the latter did not affect execution of other underwater scuba diving skills.

Introduction

The physical performance and cognitive function of scuba divers are affected by factors such as hydraulic pressure and water temperature.[1,2] Anxiety is a also substantial consideration in scuba diving and can influence divers' physiological and cognitive activities.[3] Anxiety can be defined as a negative emotional experience caused by stress[4] and can be exacerbated by equipment problems, poor physical conditions, or psychological factors.

In previous studies, it has been observed that levels of personal anxiety influence underwater motor skills[5–15] and that it is a good predictor of panic behavior underwater.[13] Panic occurs more often in participants with a trait anxiety score above 39 on the State-Trait Anxiety Inventory (STAI). This is a useful threshold score and can be employed *a priori* for the prediction of panic behavior in novice scuba students with an overall prediction rate of 83%.[13]

In addition, anxiety typically produces negative effects on the performance of cognitive tasks. Cognitive performance of divers in open water was significantly worse than that of subjects in a chamber; this finding was attributed to the anxiety caused by the uncertainty of being in an open sea situation.[16] Impaired attentional processes are among the primary cognitive functions involved in anxious individuals.[17] Attentional control theory (ACT) assumes that anxiety impairs the efficiency of the central executive component of the working memory system and proposes that anxiety impairs processing efficiency more than performance effectiveness.[18] Executive functions are a set of cognitive processes. The core executive functions include inhibition, working memory, and cognitive flexibility.[19] Higher order executive functions require the simultaneous use of multiple basic executive functions and include planning and fluid intelligence. The basic central executive functions most affected by anxiety include shifting and inhibition.[17] Anxious individuals are noted to have impaired inhibitory abilities and show poorer concentration than do non-anxious individuals. Coombes further reported that efficiency of motor planning was compromised only when the motor task to be performed needed increased attentional resources and greater precision.[20]

Table 1

Relevant characteristics of the study groups with a trait anxiety score < 39 (low anxiety trait) vs. ≥ 39 (high anxiety trait). Data are mean (SD). M = male; F = female; OWD = open water diver; AOWD = advanced open water diver; SR = stress rescue diver

Characteristic	Low anxiety trait *n* = 13	High anxiety trait *n* = 14	*P*-value
Gender	M = 10; F = 3	M = 7; F = 7	0.15
Age	21.5 (2.4)	24.1 (6.0)	0.17
Height (cm)	172.5 (7.2)	166.5 (7.9)	0.05
Weight (kg)	67.4 (10.2)	56.1 (10.7)	0.01
Trait anxiety score	32.8 (6.0)	44.9 (6.0)	< 0.01
Logged dives	12.5 (20.8)	38.0 (56.3)	0.13
Certification level	10 OWD; 3 AOWD	10 OWD; 3 AOWD; 1 SR	–

Inhibition, known as interference control, is often checked by the Stroop test.[21] This test asks subjects to cite the colour of the ink that a word is written in; the words all represent colours and it has been found that when a conflict between the word colour and the ink colour exists e.g., the word 'yellow' printed in red ink instead of yellow ink, the subjects take longer to answer correctly and are more prone to errors than when name matches the colour of the ink. This conflict represents an intense interference effect; in some tasks, the correct response requires inhibition to suppress the competing automatic response.[22] The Stroop test has been used to check cognitive function in different groups such as children,[23] the elderly,[24] and athletes,[25] has been proven helpful in investigating selective attentional processes in anxiety disorders[26] and has been applied in some scuba-diving-related studies in which the participants performed the test after diving in open water or within a chamber simulating the underwater environment.[27,28]

To simultaneously observe both the effect of an anxiety trait on actual diving performance and underwater cognitive processing ability to clarify their relationship, this study sought to confirm the following issues: (1) whether anxious divers would exhibit slower diving skills performance; (2) whether anxious divers would have inefficient cognitive processing ability in underwater conditions; and (3) whether the cognitive processing ability predicts execution ability of underwater skills.

Methods

The present study was approved by the Institutional Review Board of Kaohsiung Medical University Chung-Ho Memorial Hospital, and written informed consent was obtained from all participants before the initiation of the experiment.

PARTICIPANTS

Twenty-seven scuba divers (mean age 22.9 (SD 4.7)) years were enrolled in this study and asked to complete a dive history questionnaire to determine their diving experience. The mean logged dives were 25.7 (44.2) for those divers. In accordance with trait anxiety thresholds predictive of panic[13] and respiratory distress,[29] the group was subdivided into participants with an anxiety trait score above 39 (STAI-T ≥ 39) and those with a score below 39 (STAI-T < 39). The Chinese version of State-Trait Anxiety Inventory was applied, which consists of 20 items rated on a 4-point Likert scale. There are seven anxiety-absent statements and 13 anxiety-present statements in the Trait scale. By summarizing all single scores for each item, the scale has a total score of between 20 and 80.

Relevant group characteristics are presented in Table 1.

TEST LOCATION

An outdoor diving pool was selected for the Stroop test. The pool had a length of 25 m and a depth of 10 metres' fresh water (mfw) at the deepest part; the water temperature was maintained at 27°C. Several platforms at different depths (2 and 5 mfw) were installed for training purposes. For safety reasons, only the shallow area of the pool (5 mfw) was used to conduct the Stroop test. To ensure that the instructor could fully observe underwater activity, scuba diving skills performance was measured in a general swimming pool.

EXPERIMENTAL PROCEDURE

The participants were told that the purpose of the experiment was to examine their perception and scuba performance. They completed the trait-anxiety part of the STAI before the formal test. The formal test included the diving skill tests (underwater) on one day, and on another day they undertook the Stroop tests on land and underwater in a random order).

NUMBER-STROOP TEST

During the underwater Stroop test, the participants wore standard recreational scuba equipment, working on the platform of the diving pool with a depth of 5 mfw. They observed a 10.1-inch liquid crystal display screen, placed 30 cm ahead at eye level, on which the experimental task was displayed. At the beginning of each trial, a cross at the center of the screen appeared for 1 s. The participants were instructed to fix their eyes on this cross until it disappeared. One to three numbers (numerals 1, 2 or 3) then appeared

in the center of the screen for 2 s. The participants were asked to count the number of numerals (for example '33' would equal 2 numerals), as quickly as possible, and press the appropriate keys on a wireless keypad that they held. They pressed the different keys using fingers of one hand. The participants kept looking at the screen while using the keyboard. Response speed and accuracy were emphasized. If the response was too slow (> 2 s), the program moved onto the next trial. Each subject performed 84 trials. Several practice trials were administered before the real task. For the land-based ('dry') Stroop test, the participants were asked to complete the Stroop test under conditions similar to the underwater ones with the same display screen placed 30 cm ahead at eye level. The participants were in a sitting position.

DIVING PERFORMANCE TESTS

The diving skills completed were as follows:

Mask clearing: The instructor recorded the time taken by the participants to complete the task of removing, flooding, replacing and then completely clearing the mask underwater.

Buddy breathing: The instructor recorded the time taken for the participants to hold the wrist of a diving partner, take two to three breaths, and then pass the regulator over to their partner, who took two breaths and then handed it back.

Regulator recovery: The instructor recorded the time taken for the participants to remove the breathing regulator from their mouths, retrieve it, and insert it back into their mouths, after which they cleared it before taking their next breath.

Buoyancy control: The instructor recorded the total time taken to release all the air in the buoyancy compensator and then re-establish neutral buoyancy so as to remain in a hovering position for 3 s.

Performance times were measured by an instructor with a Dive Control Specialist Instructor license. The participants were provided with an opportunity to practice three times before each test. They were then required to perform the four skills in a random order. They were asked to perform these skills as quickly as possible after initiation by a hand sign from the instructor who observed from a position directly in front of the subject. The instructor decided when each performance was completed, and 'approved' each test before the participant moved to the next skill by giving an OK signal.

DATA ANALYSIS

The participants' trait anxiety levels were calculated as the sum scores of the items of the trait anxiety scales. Scuba performance was represented by the times taken for the diver to complete each of the diving skills (see above). The Stroop test response time was defined as the time to identify the number of characters by pressing the relevant button on the keypad and was measured after target appearance. Regardless of the accuracy of the answer, the time was recorded and summed. If the response was slower than 3 s, then 3000 ms was recorded. Response times less than 100 ms were omitted. The correct answer rate was defined as the percentage of accurate responses.

SPSS 20.0 statistical software (IBM Corp., Armonk, New York, USA) was used to analyse the study data. Means and standard deviations were calculated for all variables. The chi-square test and independent *t*-test were used to test the group differences (high-anxiety vs low-anxiety divers) in basic characteristic data. A two-way mixed ANOVA was used to determine the effect of anxiety state (≥ 39 and < 39) and environment (water vs. land) on Stroop test performance. The relationships between cognitive processing ability and scuba performance were measured using the Pearson correlation test. Statistical significance was set at $P < 0.05$.

RESULTS

The average completion times for each scuba skill and the average response times and accuracy for the Stroop test for the different STAI-T-level groups are listed in Table 2. The high-anxiety divers exhibited significantly longer completion times in the mask clearing skill ($P = 0.04$) and longer response times in the Stroop test on land (dry) ($P = 0.04$) and underwater ($P < 0.01$) than did the low-anxiety divers. The ANOVA results in Table 3 indicate that being underwater had a significant effect on accuracy and response times in the Stroop test. In addition, the participants' trait anxiety levels significantly affected response times. No significant interaction between underwater effect and trait anxiety level was observed. There were no strong or significant correlations between scuba skills performance and either speed or accuracy in the Stroop test.

Discussion

Increased anxiety has been observed to impair the psychomotor and cognitive performance of divers.[11,12] In the present study, the high-anxiety group took more time in mask clearing tests than did the low-anxiety group; hence, anxiety could affect some underwater performance. The reason for this result could be due to the trigeminocardiac reflex, which is triggered by facial immersion in cold water and causes an automatic gasp. Therefore, the participants could be at risk of inhaling water while clearing their masks. Such situations increase the anxiety state of people with relatively high trait anxiety levels. This result suggests that personal anxiety is a contributing factor for underwater performance of mask clearing.

In previous research, inhibitory control ability measured using the Stroop test was not affected by shallow water immersion at 5 mfw.[28] However, the reaction times of Stroop tests at a 20 mfw were increased without statistically significant changes in error rates,[28] suggesting that the

Table 2
Scuba skill performance and number-Stroop test results in the study groups with a trait anxiety score < 39 (low anxiety trait) vs. ≥ 39 (high anxiety trait). Data are mean (SD). D = dry; U = underwater

Skill	Low anxiety trait (*n* = 13)	High anxiety trait (*n* = 14)	*P*-value
Mask clearing (s)	7.1 (3.2)	10.8 (5.4)	0.04
Buddy breathing (s)	34.8 (25.7)	43.3 (38.3)	0.50
Regulator recovery (s)	9.8 (3.0)	11.1 (3.5)	0.32
Buoyancy control (s)	22.3 (11.4)	26.9 (12.4)	0.33
Number-Stroop test			
Correct rate (D) (%)	95.6 (2.0)	95.4 (2.7)	0.84
Correct rate (U) (%)	88.9 (3.9)	86.1 (9.3)	0.33
Response time (D) (s)	49.8 (3.0)	53.3 (5.4)	0.04
Response time (U) (s)	64.4 (5.0)	72.5 (5.5)	< 0.01

Table 3
Two-way ANOVA evaluation of the effect of anxiety trait and environment (dry or underwater) on number-Stroop test results

Parameter	Underwater effect		Anxiety effect		Interaction	
	F	*P*	*F*	*P*	*F*	*P*
Percent accuracy	33.10	< 0.01	0.92	0.35	0.87	0.36
Response time	146.41	< 0.01	22.33	< 0.01	2.63	0.12

participants' cognitive systems slowed but accuracy remained constant. Inhibitory control ability is strongly involved in behaviour, which is crucial to safety and accident prevention in extreme environments and enables individuals to choose how to respond to special situations and how to behave.[19] In the present study, cognitive processing abilities, such as accuracy and response time, measured using the Stroop test, declined at a depth of 5 mfw. This negative result may have resulted from a lack of experience of the divers in the present study. In comparison, the participants in a previous study were all experienced and the result was that executive functions were not affected at a depth of 5 mfw.[28] Attentional control theory suggests that high-anxiety individuals sometimes show weak performance on tasks that demand attention because processing cognitive information is impaired by high anxiety levels.[17] This theory uses the working memory model, which is proposed to include a central executive and two slave systems.[30] Anxiety occupies resources of the central executive system leading to fewer processing resources being available for work demands. The high-anxiety group took significantly more time to complete the Stroop test in the present study, whereas their accuracy was not affected. This result was consistent with attentional control theory in that while anxiety might not influence the final decision, it nevertheless slows down the cognitive processes.

The results of the present study suggest that scuba skill performance was not significantly associated with cognitive processing ability as measured by the Stroop test, a basic psychological measure of inhibitory control.[19] However, it measures only a part of the executive function. In addition, the participants had all completed basic diving training, which included the diving skills tested in this study. A repetitive practice effect could have masked any effect of cognitive processing ability.

There are several limitations to our study. First, the number of subjects was small and there was a trend toward greater diving experience in the higher anxiety trait divers (Table 1). This could (arguably) bias against showing an effect of anxiety on diving skill performance. Second, performance time is not an ideal measurement parameter. Other methods of evaluating performance should be considered, such as the performance rating on a 7-point Likert scale.[14] Finally, this study was performed under relatively safe conditions, and so the results cannot be completely applied to true open-water conditions. To determine the relationship between activities performed underwater in real-world conditions and anxiety, more complex tasks should be designed. Testing in controlled conditions in open water might be required.

Conclusion

According to attentional control theory, working effectiveness should not be affected by anxiety whereas efficiency may be reduced. The present study verified that the high-anxiety group took more time in completing the underwater Stroop test tasks; however, the accuracy of that group was not affected. However, there was no correlation between diving skill performance and the Stroop test task. Further complex cognitive function tests may be required in future studies to completely represent the required real-world diving capabilities. The present study also indicated that high-anxiety divers took more time clearing their mask, so in regard to practical application, coaches may need to give these students more opportunities to practice this technique.

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Review article

Recreational diving in persons with type 1 and type 2 diabetes: Advancing capabilities and recommendations

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Abstract

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Diving by persons with diabetes has long been conducted, with formal guidelines published in the early 1990s. Subsequent consensus guidelines produced following a 2005 workshop helped to advance the recognition of relevant issues and promote discussion. The guidelines were intended as an interim step in guidance, with the expectation that revisions should follow the gathering of additional data and experience. Recent and ongoing developments in pharmacology and technology can further aid in reducing the risk of hypoglycaemia, a critical acute concern of diving with diabetes. Careful and periodic evaluation remains crucial to ensure that participation in diving activity is appropriate. Close self-monitoring, thoughtful adjustments of medications and meals, and careful review of the individual response to diving can assist in optimising control and ensuring safety. Open communication with diving partners, support personnel, and medical monitors is important to ensure that all are prepared to effectively assist in case of need. Ongoing vigilance, best practice, including graduated clearance for diving exposures and adverse event reporting, are all required to ensure the safety of diving with diabetes and to promote community understanding and acceptance.

Introduction

Diabetes has the potential to promote acute changes in consciousness, making it at least a relative contraindication for many activities. While the non-breathable medium does increase the risk of scuba diving for all participants, and even more for persons susceptible to altered levels of consciousness, there is also a long history of persons with diabetes participating in scuba diving activities with a reasonable record of safety. Societal pressure to eliminate unnecessary barriers to participation has led to expanded consideration of risk and risk mitigation.

Healthy individuals maintain plasma glucose in a range from 4–8 mmol·L^{-1} (72–144 mg·dL^{-1}), relying in large part on endogenous insulin to regulate the movement of glucose into cells. Diabetes mellitus is a chronic disease characterised by insufficient insulin production (type 1 diabetes; T1D) or ineffective response to this hormone (insulin resistance; type 2 diabetes; T2D). Combinations of insulin resistance and reduced insulin production are also possible. It is currently estimated that there are 420 million people suffering from diabetes worldwide, with the number expected to exceed 500 million by 2030. Approximately 90% of the individuals are diagnosed with T2D and 10% with T1D, however, there are regional differences in the prevalence of diabetes.[1] Diabetes management involves the individualised application of diet, physical activity, and medical treatment to promote good glucose control, intended to avoid both acute and chronic complications.

T1D is treated with multiple daily injections, a mixture of basal and prandial insulin boluses, or through continuous subcutaneous insulin infusion (CSII) with insulin pumps. Most individuals with T1D benefit from rapid-acting insulin analogs in combination with basal insulin analogs to reduce

the risk of hypoglycaemia.[2] Education and awareness are important to help patients manage ongoing challenges of variable meal content, physical activity, sickness, stress, and for women, menstrual cycle effects. Dose adjustments may be needed to manage the variables, and the delay between subcutaneous injections and the glucose lowering effect requires individuals to both understand the physiological relationships and plan ahead to effectively maintain glucose control.[3]

T2D is characterized by insulin resistance in combination with a progressive loss of β-cell insulin secretion. Metformin is the preferred initial pharmacologic treatment agent. Once initiated, metformin therapy should be continued as long as it is tolerated and effective. Other agents, including insulin, can be added to a metformin regimen as required. Early introduction of insulin is considered if there is evidence of ongoing catabolism (weight loss), frequent symptoms of hyperglycaemia, or frequent periods of plasma glucose ≥ 16.7 $mmol \cdot L^{-1}$ (300 $mg \cdot dL^{-1}$) or HbA_{1C} levels > 86 $mmol \cdot mol^{-1}$ (10%).[2] Patients with newly diagnosed T2D with poor glycaemic control, evidenced by HbA_{1C} ≥ 113 $mmol \cdot mol^{-1}$ (12.5%) or 5 $mmol \cdot mol^{-1}$ (1.5%) above their target, may benefit from initiated treatment with metformin in combination with insulin.[3]

A patient-centered approach guides the individual prescription of glucose lowering agents. Considerations include different comorbidities. Patients with T2D who have established cardiovascular or chronic kidney disease may benefit from modern glucose lowering agents such as sodium glucose cotransporter-2 inhibitors (SGLT-2 inh) or glucagon-like peptide-1 receptor agonists (GLP-1 RA). GLP-1 RAs are preferred to insulin for most patients with T2D who need the greater glucose-lowering effect of an injectable medication while avoiding an elevated risk of hypoglycaemia.[4]

The obvious primary acute concern regarding physical activity of persons with diabetes who require glucose lowering agents for control is hypoglycaemia, which can impair cognitive function and consciousness. Hyperglycaemia can also impair cognitive function, and persistent hyperglycaemia can lead to an increase the risk of long-term diabetes complications, including cardiovascular disease, renal failure, and vision impairment.

The risk of cardiovascular events must also be taken under consideration as a critical component of surveillance and screening in persons with diabetes. Candidates with significant co-morbidities or secondary complications should not be cleared to participate in diving activities. This applies to those with either T1D or T2D. Discussion of selection and surveillance is beyond the scope of this paper.

Improving glycaemic control in patients with T1D substantially reduces the risk of microvascular complications and cardiovascular disease.[5,6] Weight loss and exercise or, in more extreme cases, bariatric surgery will assist in achieving better glycaemic control in patients with T2D.

Diabetes treatment guidelines emphasise the value of good glycaemic control to mitigate the risk of cardiovascular complications.[7] Good glycaemic control is also important for T2D individuals to reduce the risk of cardiovascular disease.[8] It has been shown in persons with T2D that despite a mean glycated haemoglobin level of 7% or less (≤ 52 $mmol \cdot mol^{-1}$) and an age of less than 55 years, the excess risk of death was still approximately twice as high as the risk among controls.[9]

The risk of cardiovascular events should be evaluated in both T1D and T2D subjects, weighing the separate risk factors for cardiovascular disease, in the overall fitness to dive assessment in divers with diabetes.

Innovations in diabetes care

The last two decades have seen an increase in pharmacological options, particularly new insulin formulations that are less likely to result in hypoglycaemia. Examples include insulin Glargine U300, insulin degludec, oral SGLT-2 inh, and injectable GLP-1 RAs. In parallel, technological innovations include downloadable insulin pens and insulin pumps paired with continuous glucose monitoring (CGM). Reducing the frequency and severity of hypoglycaemic events can reduce the development of hormonal regulation impairment and thereby lead to more stable glucose levels.[10]

A transition from direct blood glucose monitoring to sensor-based glucose monitoring of the interstitial fluid with CGM is ongoing. In many countries, CGM, either real-time (rtCGM) or intermittent scanning (isCGM), is now considered standard of care. In addition to current glucose values, CGM can provide profiles on past and recent glucose trends and rates of change.[11] Thus, a more comprehensive picture is available for the individual with diabetes. Practically, rtCGM can incorporate user-adjusted alerts, alarms and follower notification functions to aid in glucose management. CGM can be used either as a stand-alone system with multiple daily injection therapy or with an insulin pump/CSII.

Emerging technologies include insulin pumps with autonomous insulin infusion stops, low and predicted low glucose suspension (LGS and PLGS, respectively) functions, and hybrid closed loop (HCL) pumps. LGS and PLGS functions can reduce glucose variability and minimise hypoglycaemic events.[12,13] HCL pumps adjust the insulin dose based on glucose sensor readings to further reduce glucose variability and the risk of hypoglycaemia.[14]

Diabetes and diving

Diabetes is at least a relative contraindication for diving since impaired consciousness affects the diver's ability

to take care of himself/herself or a diving partner and potentially confer a significant risk to health or life. The risk can be increased if the condition contributes to a lack of cardiovascular fitness. It has historically been described as an absolute contraindication prompting blanket bans, but more recent experience has moved it into the relative contraindication class for those without significant co-morbidities or secondary complications.

Diving with diabetes was banned in the UK after an unfortunate case of misdiagnosis ultimately led to a diver committing suicide.[15] Similar prohibitions existed internationally, with the timeline varying based on when it was flagged as an area of concern. Despite the existence of the general prohibition, it became clear that many individuals were diving, and that they were often hiding their condition to avoid disqualification, potentially increasing risks through a lack of group preparedness. The ban on diving by persons with diabetes was reversed in the UK in 1991, but this did not reflect a common pattern of relaxation internationally. A workshop was organized by the Undersea and Hyperbaric Medical Society (UHMS) in 1996 to discuss the possibility of liberalizing guidance on diving with diabetes. The weakness of the evidence base made it impossible for agreement to be reached, but the meeting was successful in promoting international efforts to gather relevant data. The substantial body of evidence produced over the following decade suggested that diving by persons with diabetes could be conducted with notably few adverse events related to the condition.[15–18]

The research evidence provided the foundation for a 2005 UHMS-Divers Alert Network (DAN) workshop.[19] Consensus guidelines for recreational diving with diabetes were generated, primarily focusing on T1D cases. There were three discrete components to the guidance: selection and surveillance, scope of diving, and glucose management on the day of diving.[19,20]

Selection and surveillance play a critical role in determining if candidates should be allowed to dive. Problematic co-morbidities, serious secondary complications, and poor physical fitness can all serve to disqualify candidates (see 'Medical clearance'). Our focus in this review is largely on individuals who are generally healthy, with well managed diabetes as the primary health issue.

The scope of diving guidance was intentionally conservative as a starting point: a maximum depth limit of 30 metres; all dives within no-decompression limits; no overhead environments; and duration no longer than 60 minutes. There are concerns, primarily related to hypoglycaemia, that justified a conservative starting point. Symptoms associated with hypoglycaemia could be confused with those arising from other factors related to diving. For example, fatigue could result from effort expended during a dive; reduced cognitive focus or lethargy could result from cold stress; confusion or mental absence could result from nitrogen narcosis; and malaise or other physical compromise could result from decompression illness.[21] An erroneous assessment could lead to delays in securing the correct treatment, be it carbohydrate or glucagon administration or hyperbaric oxygen therapy. Concerns that hyperbaric exposure would specifically alter glucose utilization was set aside following a chamber study of exercising divers with and without T1D,[22] but there remains a concern that the distractions of diving could increase the risk of hypoglycaemic unawareness. The diving environment changes many normal stimuli; altering sensations due to immersion, buoyancy, wetsuit and/or hood use; compromised verbal and auditory communication; and creating unusual visual effects and physical demands. Symptoms might be more easily missed. Cautious experience could help to minimise the risk of complications.

Medical clearance for recreational divers with T1D and T2D

Diving can be physically demanding, and individuals with diabetes who want to start this activity must demonstrate sufficient knowledge and control over their condition in addition to meeting requirements for diving and medical fitness. This includes a cardiology screening[23–26] and medical clearance regarding glucose control.

CARDIOLOGY SCREENING TO EVALUATE IF THE INDIVIDUAL IS 'FIT TO DIVE'

The goals are to:

- Identify individuals with serious cardiac conditions that could pose a danger during diving, for example, hypertrophic cardiomyopathy or aortic stenosis.
- Identify individuals with cardiovascular disease that could cause disablement during diving, for example, acute myocardial infarction or angina pectoris.

Recommendation:

- Screening is recommended of all individuals with T1D or T2D.
- History including information on heredity and symptoms of cardiovascular diseases.
- Clinical examination including monitoring and evaluation of blood pressure, heart rate and heart sounds, and a 12-lead resting ECG.
- In case of abnormal findings, referral to a cardiologist is recommended.

MEDICAL CLEARANCE REGARDING GLUCOSE CONTROL

Candidates must be able to adjust insulin doses and carbohydrate intake prior to dive.[27] Patients with T2D should use an oral glucose lowering agents that best control their diabetes but must bear in mind that some of these medications may increase the risk of hypoglycaemia (Table 1).[4]

Table 1

Glucose lowering agents, cardiovascular effects, and risk of promoting hypoglycaemia.[4] CHF = congestive heart failure; CVD = cardiovascular disease; DPP-4 inh = dipeptidyl peptidase inhibitors; GLP-1 RA = glucagon-like peptide-1 receptor agonists; MI = myocardial infarction; SLGT-2 inh = sodium glucose cotransporter-2 inhibitors

Class of glucose lowering agent	Representative agents	Cardiovascular benefit and risk	Risk of hypoglycaemia
Biguanide	Metformin	Reduction MI and coronary deaths	Low
DPP-4 inh	Sitagliptin Saxagliptin Vidagliptin Linagliptin	CV risk neutral, may increase risk of CHF	Low
SLGT-2 inh	Canagliflozin Dapagliflozin Empagliflozin	Reduction CVD risk	Low
GLP-1 RA	Liraglutide Exenatide Dulaglutide Semaglutide	Reduction CVD risk	Low
Thiazolidinediones	Pioglitazone	Increased risk of cardiac failure	Low
Insulin	Human insulin Mealtime analogues NPH insulin Basal analogues	CV risk neutral	High
Sulfonylureas	Glimepiride Glibenclamide Glipizide	CV risk neutral or may be increased	High

Medical clearance to dive requires a consensus between clinicians with expertise in diabetology and diving medicine, and the diver. The hypoglycaemia definitions used are in accordance to the American Diabetes Association (ADA) classification.[28] The following simplified criteria for dive qualification have been proposed:[27]

- Glycaemic control, HbA1c < 63 mmol·mol^{-1} (8%), without any symptomatic long-term diabetic complications (cardiovascular, nephropathy, neuropathy, or substantial retinopathy). Mild non-proliferative background diabetic retinopathy is accepted.
- No history of severe hypoglycaemia (defined as that requiring intervention by a third party)[28] during the last year and no evidence of hypoglycaemia unawareness.
- Knowledge on how to manage the diabetes disease, monitor glucose levels, adjust insulin doses effectively, and to gauge carbohydrate intake prior to physical activity.
- CGM is recommended for risk evaluation and hypoglycaemia prevention prior to diving and for subsequent evaluation of outcomes.
- Yearly evaluation should be performed by a specialist in diabetology in consultation with a physician with training in diving medicine.

The focus of the 2005 UHMS-DAN guidelines[19] was on the recreational diver, but the same guidance for selection and surveillance, glucose management on the day of diving and team awareness and readiness could apply if the diver with diabetes was a dive leader or professional diver. One of the areas that would benefit most from refinement is the scope of diving. A concept not included in the 2005 UHMS-DAN guidelines is graduated clearance, which would work well for all divers with diabetes, and particularly well to define the limits for professional divers.

A model for graduated clearance can be found in scientific diving. Divers are initially approved to dive to the shallowest depths with experienced partners. After gaining experience and demonstrating competence at a step they may be cleared for the next depth increment. A recommendation was made to the American Academy of Underwater Sciences to consider formalising the depth clearance of scientific divers with diabetes using a similar graduated scheme,[29] but qualification for this group currently remains solely based on medical clearance. In any case, a practice of formal or informal graduated clearance to dive could provide additional safety buffers, reducing stress and concerns for all. Questions of the duty of care of persons with diabetes responsible for others deserves additional consideration, but it is beyond the scope of this paper. It is important to acknowledge that restrictions on professionals vary and that there is no unified position or solution.

Preventing hypoglycaemia through glucose monitoring

The goal to maintain stable or moderately rising pre-dive glucose levels is to minimise the risk of hypoglycaemic

Table 2

Illustration of different continuous glucose monitoring devices: Dexcom G4/G5/G6 (rtCGM); Freestyle Navigator (rtCGM); Abbott Libre (isCGM); Eversense XL (implantable sensor, rtCGM); and Medtronic (rtCGM). These enable automatic frequent measurements of glucose concentration (every 5–15 min) and a simultaneous indication of the direction of changing glucose values and how fast this is happening, indicated by different trend arrows. The table illustrates various combinations (glucose value and type of trend arrow) where the individual can predict a low glucose value during the next 15 minutes, which also allows preventive measures to be taken

Product	Interval mmol·L^{-1} / mg·dL^{-1}	Trend arrow	Interval mmol·L^{-1} / mg·dL^{-1}	Trend arrow	Interval mmol·L^{-1} / mg·dL^{-1}	Trend arrow
Dexcom (G4,G5,G6)	> 6.5 / > 117	↓↓	5.7–6.5 / 103–117	↓	4.8–5.7 / 86–103	↘
Freestyle Navigator; Libre; Eversense (XL)	Not applicable (n/a)	n/a	> 5.7 / > 103	↓	4.8–5.7 / 86–103	↘
Medtronic (Enlite 2,3)	> 6.5 / > 117	↓↓↓	5.7–6.5 / 103–117	↓↓	4.8–5.7 / 86–103	↓

events[28] during dives. Frequent glucose monitoring is required to be aware of state and changes in state. If a glucometer is used, measurements taken at least four to eight times per day during one to two weeks prior to and throughout periods of diving are recommended.

Use of CGM gives additional information and a monitoring period of at least two weeks is recommended prior to diving. This type of glucose monitoring could be beneficial to identify and address unexpected deviations in glucose management that could potentially increase the risk during subsequent diving. Episodes can be more reliably detected by CGM to warn of unwelcome deviations and the potential need for additional regimen adjustments.

Real-time CGM can help to prevent hypo- and hyperglycaemia day and night and to register possible hypoglycaemia unawareness. In addition to the use of alerts and alarms, trend data can help guide good practice. The beneficial effects of rtCGM on glucose control has been verified with insulin injection therapy[30,31] and CSII treatment.[32] Individuals demonstrating hypoglycaemia unawareness had a lower frequency of hypoglycaemia when using rtCGM in comparison with self-monitored blood glucose.[33] Additionally, rtCGM was associated with decreased frequency of nocturnal hypoglycaemia and improved glycaemic variability.[34] The use of rtCGM in connection to recreational diving has been shown to be beneficial for individuals with T1D.[34] Table 2 lists combinations of glucose levels from CGM systems with the use of trend indicators predicting imminent hypoglycaemia hazards.

While CGM and pump technologies provide additional insight and protection for patients, it must be recognized as an important limitation that they cannot currently be used while diving. Pumps are generally described as water-resistant, but only allowing exposures to depths of 2.5–3.5 metres for short periods. Neither the structural integrity nor delivery performance is tested beyond these near-surface conditions. All insulin pumps should be disconnected and removed before dives and then reconnected as soon as possible after diving in order to reinstate insulin delivery, glucose monitoring and control.

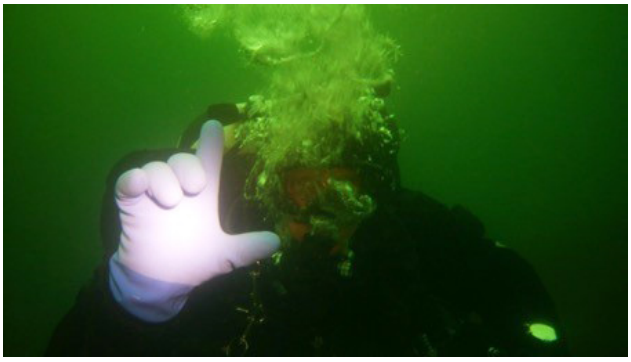
Preventing hypoglycaemia by insulin adjustments and carbohydrate intake

Individuals using multiple daily injection treatment should reduce the long acting (< 24 h effect) insulin dose to minimise the risk of hypoglycaemia during diving. A 20% reduction may be reasonable for moderately long, strenuous, or repetitive dives. Individual response must be assessed, progressing from the most benign conditions, to determine the most appropriate adjustments. When a more pronounced long-acting insulin is used (insulin degludec), a dose reduction results in a postponed effect which may not reduce the risk of hypoglycaemia during the day of the dive. Instead, in these cases, large pre-dive carbohydrate boluses (30 g or more) without additional insulin may be necessary to prevent hypoglycaemia.[35]

Employing CSII treatment, the risk of hypoglycaemia during diving may be reduced by adjusting the basal rate (90–120 min before diving) and/or the intake of carbohydrates prior to diving.[27,34] During a series of relatively modest dives (maximum depth 18–22 metres; 42–52 min duration), the glucose level was reduced by 1.7 (SD 3.8) mmol·L^{-1} (31 (68) mg·dL^{-1}) when the insulin dose was reduced prior to dive and a carbohydrate bolus was ingested with no extra insulin 10 min pre-dive.[29] Using CSII with an autonomous insulin infusion stop and PLGS function before and after diving could further improve safe management,[12,13,36] but this has not been studied during diving conditions.

In order to lower the glucose variability during diving a meal should be consumed at least 1.5–2 h and preferably 3 h prior to the dive. The insulin concentration levels will then be lower at start of diving and thus reduce the risk of

Figure 1
The 'L-signal' – sign of hypoglycaemia – posed as a question or offered as a statement. Photo by Peter Adolfsson



hypoglycaemia during the dive. The insulin dose prior to a meal may need to be reduced, especially when diving is conducted 1.5–2 h after a meal. The glucose target prior to diving should be 7–12 mmol·L^{-1} (126–216 mg·dL^{-1}). The pre-dive intake of a modest carbohydrate bolus, 15–30 g per 70 kg body weight (depending on glucose value), is recommended just before diving with no accompanying insulin.

When repeated dives are planned, insulin dose adjustment may be needed before meals to compensate for the increased insulin sensitivity seen during repeated physical activity comparable to the planned scuba diving.

Detailed personal logbooks should be maintained, recording multiple pre- and post-dive glucose levels, carbohydrate boluses, insulin dose, and description of dives (depth, duration, thermal stress, levels of physical exertion, and any adverse events). When rtCGM is used, calibration or confirmation of readings is recommended before and after each dive. The records and downloads can be used to understand the individual glucose response to multiple factors to optimise future glucose regulation.

Managing suspected hypoglycaemia during a dive

It is important for all divers and dive support personnel to be familiar with the needs of persons with diabetes, to be informed of protocols for monitoring and managing blood glucose deviations, and to be able to recognize acute signs and symptoms. Dive teams should practice signaling and treating hypoglycaemia. The 'L-signal', with the index finger and thumb forming the letter L, is used to ask a diver about his or her glycaemic status or to alert the partner of concerns regarding hypoglycaemia (Figure 1).[19,20,33] If a problem is suspected, the protocol based on the 2005 UHMS-DAN guidelines[19] is for both diver and partner to immediately surface and end the dive. No-decompression and no overhead diving is recommended to allow direct ascent to the surface at least until divers with diabetes gain substantial experience and are fully in control of their condition. Obligatory decompression and dives in overhead environments make surfacing more complicated. The default advice for divers concerned with possible hypoglycaemia is to surface with safe but expeditious dispatch. Once on the surface, affected divers should establish positive buoyancy and then ingest a modest carbohydrate bolus (as gel or glucose/fructose solution).

Carbohydrate formulations should be carried by both divers with diabetes and their partners on all dives. Divers should practice buddy procedures: communicating with the 'L-signal' and ingesting gel, first on the surface and then underwater for advanced practice if appropriate. A high level of comfort and competence in underwater ingestion of carbohydrate is required before divers conduct dives that do not allow direct access to the surface, either as decompression ceilings or overhead obstructions. Dive partners, divemasters, instructors, and surface support staff should understand both the concerns and management strategies for acute problems linked to diabetes. All should be sufficiently informed to be able to appropriately assess potential issues and have access to carbohydrate and glucagon to assist divers in need.

Protocols for underwater management of suspected hypoglycaemia have been developed and implemented by divers, primarily relying on the ingestion of glucose paste. Individuals with additional experience and the inability to surface directly and immediately in a safe manner would have to ingest carbohydrate before surfacing. Discussion of specific protocols for underwater ingestion of glucose is beyond the scope of this paper.

Patient obligations

- Consult a physician and diabetes/diving specialist to ensure medical and physical fitness to dive before making an attempt to dive.
- Inform dive partners and support personnel of the condition and the ways to provide aid in case of emergent need.
- Ensure that both diver with diabetes and partner carry appropriate and accessible carbohydrate formulations on all dives.
- Ingest a modest bolus of carbohydrate immediately pre-dive to minimise the risk of hypoglycaemia.
- Measure plasma glucose levels repeatedly in the hour pre-dive and immediately post-dive or consider using a CGM system to monitor glucose levels in real-time outside of diving.
- Ensure a stable or rising pre-dive glucose level (SMBG or CGM) of ≥ 7 mmol·L^{-1} (127 mg·dL^{-1}).
- Be aware of conditions that may be confused with symptoms of hypoglycaemia. Dive depth should be limited to 30 metres until diving competence, diabetes control, and emergency management capabilities are well established. Any subsequent increase in exposure depth should be thoughtful and graduated.

- Avoid dives longer than 60 minutes until glucose response under realistic conditions is fully understood and can be well managed. Any subsequent increase in dive time should be thoughtful and incremental.
- All dives should be logged, including glucose monitoring results, carbohydrate ingestion, medication use, and any adverse events. This information should be used for dive planning and shared with medical advisors to ensure optimal practices.
- Stay well hydrated in conjunction with diving.

Considerations for youths

A small group of inexperienced but motivated teenage drivers with diabetes was shown to be able to maintain plasma glucose levels and avoid hypoglycaemia associated with diving,[37] but diving by youths with diabetes must be carefully considered. Appropriate medical evaluation and clearance is necessary prior to participation. Candidates must have the physical ability, mental maturity, and motivation to manage all the hazards of diving with diabetes and the assessing physician should take into account possible parental or peer pressure to dive. Divers with diabetes must have a thorough understanding of their disease, reliably demonstrate good management practice and risk assessment, and be sufficiently mature to take personal responsibility for their safety, fully understand the risk that they may pose to themselves and other divers and demonstrate appropriate diving skill. As recommended in the 2005 UHMS-DAN guidelines,[19] a minimum age of 16 years is likely reasonable, and only then for individuals who have been well oriented in training programs that incorporate both dive training and diabetes management.

International aspects of diving

Divers often travel widely to dive, and it is important to comply with local regulations and requirements for diving and diabetes. Responsible practice is important to protect the safety and freedom of all divers. Relevant training materials and guidelines are available for review and to share with local authorities.[19,38–40]

Limitations

It is understood that the availability of treatment options and medical care is not consistent. Direct consultation with experts in either diving medicine or diabetes may not be practical. The expense associated with scuba diving may limit the pool of candidates who are in a better position for optimised care, but it is unclear that all who may wish to participate can be appropriately evaluated. A conservative approach, in lieu of comprehensive medical support, is warranted.

Where possible, candidates for diving with diabetes would be best trained through organised programs dedicated to providing this familiarisation. Unfortunately, such programs are not widely available currently. Additional effort is needed to develop training materials and programs to facilitate dissemination of critical information to divers, diving leaders, and medical monitors. Resource materials made available to community-based groups could minimise the background work required by such groups and ensure a higher standard for consistent and best practice. Clearance to dive professionally for persons with diabetes must be established through consultation that includes medical monitoring and professional oversight involving various authorities as well as based on national rules and regulations.

Conclusions

Diving, even in the absence of diabetes, is a potentially dangerous activity. Mindful and safe practice is required for all divers, particularly for those who have additional medical challenges. There is a substantial evidence base indicating the diving with diabetes can be conducted safely by qualified individuals but ongoing vigilance and best practice is required. Recent and ongoing developments in pharmacology and technology can aid in reducing the risk of hypoglycaemia, the critical acute hazard in diving with diabetes. Open communication with diving partners, support personnel, and medical monitors is important to ensure that all are prepared to effectively assist in case of need. Ongoing vigilance, best practice, including graduated clearance for diving exposures and adverse event reporting, are all required to ensure the safety of diving with diabetes and to promote community understanding and acceptance.

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Technical report

Evaluation of the Abbot FreeStyle Optium Neo H blood glucose meter in the hyperbaric oxygen environment

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Key words
Blood glucose level; Glucose monitoring; Diabetes; Hyperbaric oxygen treatment

Abstract
(Baines CR, Cooper PD, O'Rourke GA, Miller C. Evaluation of the Abbot FreeStyle Optium Neo H blood glucose meter in the hyperbaric oxygen environment. Diving and Hyperbaric Medicine. 2020 June 30;50(2):144–151. doi: 10.28920/dhm50.2.144-151. PMID: 32557416.)
Introduction: This study investigated the effects of hyperbaric oxygen treatment (HBOT) on the accuracy and reliability of point-of-care fingertip capillary blood glucose values in euglycaemic non-diabetic participants compared against venous serum blood glucose samples processed in an accredited pathology laboratory.
Methods: Ten non-diabetic hyperbaric staff members (age 35–55 years) underwent a standard 243 kPa HBOT exposure for 95 minutes. Blood glucose levels were measured via (i) finger-prick capillary test using the FreeStyle Optium™ Neo H glucometer and (ii) venous serum test using the Cobas 6000 laboratory analyser. Samples were taken at (T1) 0 minutes (pre-HBOT), (T2) 25 minutes, and (T3) 55 minutes into HBOT.
Results: All participants were euglycaemic at T1 (BGL 3.8–5.4 mmol·L^{-1}). The highest venous serum value was 5.90 mmol·L^{-1} at T3 and the highest capillary value was 6.30 mmol·L^{-1} at T1. *Post hoc* tests showed a statistically significant difference between the mean capillary result pre-dive (T1) and readings at T2 ($P = 0.001$) and T3 ($P < 0.001$) while differences between T2 and T3 capillary results were not statistically significant, illustrating the effect of HBOT on capillary beds. Differences in venous values across the time points were not significant.
Conclusions: Venous serum glucose samples processed in an accredited laboratory may be more consistently accurate, but capillary point-of-care testing avoids delays in sample processing and provides glucose data that are of clinical relevance. The FreeStyle Optium™ Neo H glucometer is safe to use and provides a reliable measurement of blood glucose in the HBOT environment.

Introduction

Since the 1970s there has been a gradual increase in the applicability of hyperbaric oxygen treatment (HBOT) which has seen it move away from the sole domain of diving-related disorders. It is now used in the management of a number of medical and surgical conditions, including wounds that fail to heal due to diabetes or radiation injury.[1]

Accurate identification of blood glucose level (BGL) particularly at levels lower than 4.0 mmol·L^{-1} is extremely important in the clinical hyperbaric oxygen environment. A growing proportion of the hyperbaric patient population now has diabetes either as a causative factor (e.g., non-healing wounds) or a concurrent medical co-morbidity to their treatment indication. A number of studies have noted HBOT to acutely lower blood glucose, particularly in patients using insulin or insulin secretagogue therapy.[2–11] These changes in blood glucose levels could potentiate a clinically significant hypoglycaemic episode whilst at pressure. The exact physiological reasons for blood glucose fluctuations in the HBOT environment have yet to been fully elucidated.[3,12] Furthermore, the symptoms of hypoglycaemia may mimic those of cerebral oxygen toxicity, a potentially serious side-effect of HBOT.[5,13] Management of oxygen toxicity is quite different from that for hypoglycaemia therefore it is important for clinicians to be able discern which clinical event is occurring.[13–15]

Venous serum glucose measurements performed in central laboratories remain the reference method for the evaluation of glucose levels, especially for diabetic patients. However, due to their point-of-care (POC) availability, speed of analysis, and minimal blood volume requirements glucose

Table 1
Relevant characteristics of the study group

Characteristic	*n* = 10
Gender (% male/female)	50/50
Age range (years)	35–55
Weight range (kg)	70–95
Non-diabetic (%)	100
HbA1c range (%)	4.50–5.90
Cholesterol range (mmol·L^{-1})	3.90–7.40

meters are frequently used as a substitute for venous glucose assay.[16–18] The POC glucometer, using capillary blood is now a routine method for glucose analysis in the normobaric setting as well as in the hyperbaric chamber.[6,13] It is notable that the hyperbaric environment exposes the POC device to a variety of atmospheric, technical and patient factors that may potentially adversely affect the analytical accuracy of the device.[19–21]

Several studies demonstrate a consistent bias towards overestimation of the blood glucose from capillary samples when compared to venous serum measurements performed under standard laboratory conditions at one atmosphere absolute (atm abs) (101.3 kPa) pressure.[6,13] Vasomotor changes experienced during HBOT further complicate comparison of capillary to venous samples.[19–21] Additionally, the immediate milieu of modern HBOT requires careful consideration of which electronic devices are permitted within the hyperbaric chamber due to fire and implosion risk.[22–25] These factors can be coupled with the potential for analytic inaccuracies when used outside the manufacturers' specified environmental conditions.[25] Performance of POC glucometers has been demonstrated to be unpredictable under hyperbaric conditions, due to either the direct effects of pressure on engineered components or the effects of hyperoxia on biochemical machinery (e.g., enzymes in test strips) or a combination of both.[22]

This study aimed to assess the accuracy and reliability of POC fingertip capillary blood glucose values in euglycaemic non-diabetic participants under hyperbaric conditions and compare against contemporaneous venous serum samples processed in a recognised pathology laboratory.

Methods

This study was prospectively approved by the Tasmanian Human Research Ethics Committee (UTAS HREC No: H0015770) and conducted in accordance with National Health and Medical Research Council (NHMRC) guidelines and relevant institutional governance procedures. All participants (n = 10) were hyperbaric staff members at a tertiary referral hospital for the state of Tasmania, Australia. Each participant gave written informed consent to the inclusion of their data in this study. Participants were non-diabetic and had a valid medical certificate for diving.[26,27] Each participant was additionally screened for dyslipidaemia and diabetes mellitus as the presence of elevated venous serum triglyceride levels and modest increases in HDL cholesterol levels can influence long-term glycaemic control.[28,29] Participant characteristics are reported in Table 1.

Immediately prior to hyperbaric exposure each participant had an intravenous cannula inserted aseptically into their preferred antecubital fossa. They then underwent a standard 243 kPa (14 metres' seawater [msw] equivalent) hyperbaric exposure according to a '14:90:20' treatment table (Figure 1). Each participant received oxygen via an Amron™ oxygen treatment hood (Amron International, Inc., California, USA) in a multi-place chamber with an accompanying attendant as per Australian and New Zealand Standards.[26–30] None of the participants consumed food whilst undergoing this treatment. All participants had consumed a normal breakfast prior to exposure.

Blood was drawn from the intra-venous cannula at three time-points for laboratory processing, with simultaneous capillary sampling undertaken by lancing the finger-tips of each participant for POC blood glucose testing. POC capillary blood testing was performed using three separate FreeStyle Optium™ Neo H glucometers (Abbott Healthcare, Massachusetts, USA). At each time-point the finger-prick capillary sample was divided between the three glucometers to evaluate inter-glucometer variability. The FreeStyle Optium™ Neo H glucometer was used to process both the pre-exposure POC BG value and the intra-exposure (in-chamber) BG values. The FreeStyle Optium™ Neo H glucometers are designed for use with the glucose dehydrogenase (GDH-NAD) enzymatic test-strips which were used in this study according to manufacturer's instructions for all POC testing.[31]

Time-point 1 (T1) was immediately prior to compression; (a normobaric sample at 101.3 kPa), time-point 2 (T2) was at the start of the first air-break after 25 minutes of hyperbaric oxygen at 243 kPa; and time-point 3 (T3) was at the start of the second air-break after 55 minutes of hyperbaric oxygen at 243 kPa. Glucometer testing for capillary blood glucose values at T2 and T3 testing was undertaken within the hyperbaric chamber under pressure. All POC capillary glucose results were compared with venous serum glucose results obtained from a reference laboratory (see below).

Venous serum samples were drawn from each participant into a blood collection tube containing sodium fluoride, and a glycolysis inhibitor used to limit the *ex vivo* consumption of glucose.[32] The rate of glycolysis varies with the glucose concentration, temperature, white blood cell count, and other factors. All samples were retained in-chamber until the end of the hyperbaric exposure from whence all samples were taken to the laboratory. The elapsed time from collection-to-separation of the blood sample did not exceed the test site's laboratory recommendations.[33]

Figure 1
Illustration of a '14:90:20' hyperbaric treatment profile showing respired gas and BGL sampling times

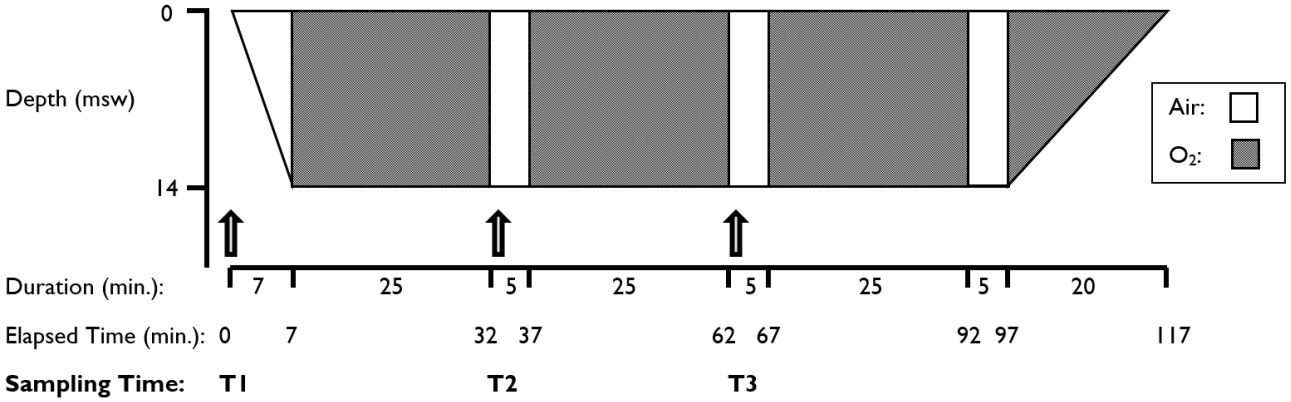
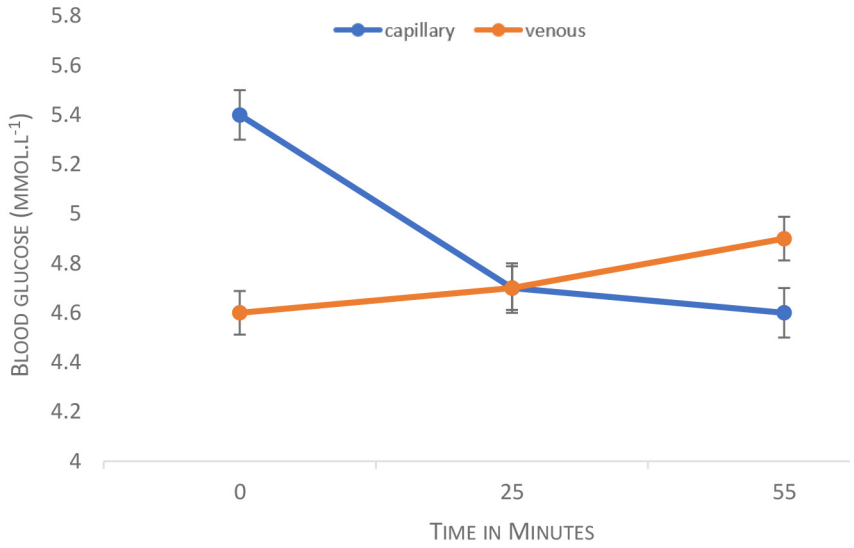


Figure 2
BGL trends in capillary and venous serum samples during hyperbaric oxygen treatment. Values are means and the error bars display standard deviation



Laboratory testing of venous serum samples was performed using the hexokinase enzymatic reference method with the GLUC3 kit of the cobas 6000 laboratory analyser's c501 module (Roche Diagnostics GmbH, Mannheim, Germany) in the on-site biochemistry laboratory of the research facility. The laboratory is accredited as a human pathology testing provider by the National Association of Testing Authorities, Australia (NATA).[34] The cobas 6000 system has a standard deviation of 0.05 mmol·L^{-1} when the mean is 5.1 mmol·L^{-1}. The precision diminishes at a higher glucose level of 14.2 (considered a pathological level) with a standard deviation of 0.16 mmol·L^{-1}.[35]

STATISTICAL ANALYSIS

Mean absolute relative difference (MARD) calculation is currently the most widely accepted measurement for comparing the performance and accuracy of multiple meters in a single study.[36] MARD is determined by dividing the difference between the measured and reference values by the reference value and multiplying by 100 to generate a percentage.

For this study MARD was calculated by using two sets of data collected simultaneously. MARD was chosen for this review due to the requirement for direct clinical applicability of results.[37] The first set of data was obtained using the average of the three repeated capillary measures from the Freestyle Optium Neo H, while a standard laboratory measurement on venous samples provided the second dataset. The percentage reflects the average difference between capillary and the reference value. The mean of the readings from the three

Table 2
Venous serum and capillary blood glucose mmol·L^{-1} (n = 10)

Time-point	Mean (SD)	Range
T1: pre-dive		
Venous Serum	4.60 (0.50)	3.80–5.40
Capillary Average	5.39 (0.42)	4.77–6.07
Capillary 1	5.33 (0.42)	4.70–6.20
Capillary 2	5.36 (0.48)	4.70–6.30
Capillary 3	5.47 (0.43)	4.80–6.10
T2: 1st air-break		
Venous Serum	4.72 (0.68)	3.30–5.50
Capillary Average	4.72 (0.41)	4.00–5.23
Capillary 1	4.78 (0.43)	4.20–5.30
Capillary 2	4.66 (0.48)	3.90–5.30
Capillary 3	4.71 (0.43)	3.90–5.30
T3: 2nd air-break		
Venous Serum	4.94 (0.55)	4.10–5.90
Capillary Average	4.60 (0.38)	4.00–5.23
Capillary 1	4.59 (0.36)	3.90–5.10
Capillary 2	4.67 (0.35)	4.10–5.20
Capillary 3	4.54 (0.52)	3.90–5.40

glucometers was compared to the venous laboratory sample. The similarity of these measures was confirmed first using a repeated-measures one-way analysis of variance (ANOVA) for each time point. The influence of time point was further examined by conducting repeated measures ANOVA using the mean capillary results as well as the venous serum values. *Post hoc* tests were examined to determine between which time points the differences were statistically significant.

Results

The average capillary and venous serum values obtained across the hyperbaric oxygen exposures are presented in Figure 2. Of note, at 25-minutes the cross-point blood glucose level was 4.7 mmol·L^{-1}.

Descriptive data for capillary and venous serum values for the sample are shown in Table 2. The venous serum data demonstrate that all participants were euglycemic at baseline (BGL 3.8–5.4 mmol·L^{-1}). The highest venous serum value was 5.90 mmol·L^{-1} at T3 and the highest capillary value was 6.30 mmol·L^{-1} obtained at T1.

ASSESSING DIFFERENCES BETWEEN REPEATED MEASURES

Variables were normally distributed and an examination of absolute skewness and kurtosis revealed that all model variables fell within skewness ≤ ± 2.0 and kurtosis ≤ ± 7.0.[38] The results were interpreted descriptively and clinically as comparable and this was confirmed by the repeated measures ANOVA which found no statistically significant differences between capillary measures pre-dive (T1) [Wilks Lambda = 0.711, $F(2,8) = 1.626$, $P = 0.255$], during the first air break (T2) [Wilks Lambda = 0.773, $F(2,8) = 1.176$, $P = 0.357$], and during the second air break (T3) [Wilks Lambda = 0.799, $F(2,8) = 1.004$, $P = 0.408$].

A statistically significant effect was found for time for the mean capillary values [Wilks Lambda = 0.117, $F(2,8) = 30.068$, $P < 0.001$]. The multivariate Partial Eta squared result was 0.883 suggesting a moderate to large effect as per Cohen's (1988) classification.[39] *Post hoc* tests were examined to determine between which time points the differences were statistically significant. These tests suggest that the difference between the mean capillary result at T1 and both subsequent readings at T2 ($P = 0.001$) and T3 ($P < 0.001$) were statistically significant but the differences between T2 and T3 capillary results were not statistically significant. Differences in venous serum values across the time points were, however, not statistically significant [Wilks Lambda = 0.651, $F(2,8) = 2.142$, $P = 0.180$].

Table 3
Mean absolute relative difference (MARD) (n = 10)

Sample time	MARD (%) Mean (SD)	MARD (%) Range
T1	-17.94 (11.48)	-40.35–3.85
T2	-1.39 (14.09)	-35.35–11.11
T3	6.44 (7.00)	-6.52–15.82

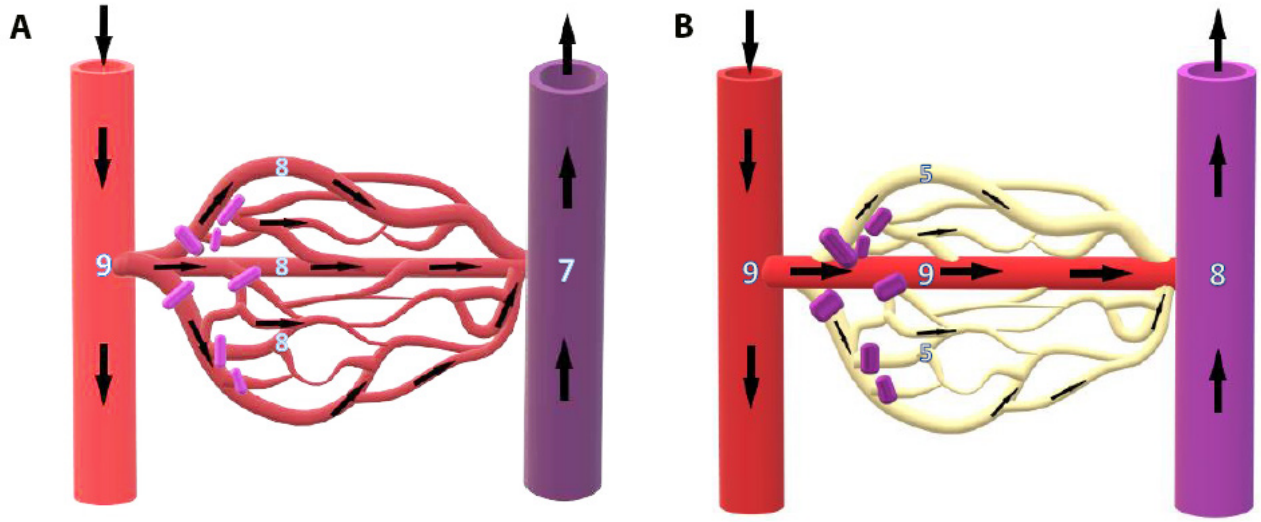
MEAN ABSOLUTE RELATIVE DIFFERENCE

The MARD reflects the percent variation that the capillary average differs from the reference value. Table 3 details descriptive data for the sample with respect to MARD percentages. As capillary scores were subtracted from the venous results, a negative MARD suggests that the venous serum value was less than the capillary value, and vice versa. As such, the results in Table 3 indicate that, on average, the venous serum BGL results were lower than capillary results at T1 and that this changed over the course of the HBOT. There was less average difference at T2 during the first air-break in the HBOT, although the range in values indicates that imprecision remains for some participants. In contrast, noting that the variation around the mean is reduced at T3 during the second air-break, the differences between the samples were not statistically significant.

Individual variability was evident from the large standard deviations and minimum and maximum values. These MARD results suggest relatively poor correlation between venous and capillary results, especially pre-exposure and after 55 min of HBOT. Although this appears to hamper the clinical use of capillary readings (since scores not only have a low accuracy but the direction of agreement is inconsistent), this finding must be interpreted in the context of the physiological changes occurring in the capillary bed during HBOT.[40]

Figure 3

A. Resting microvasculature. Progressive reduction in BGL is evident as blood passes from arterial delivery side, through the metabolically active capillary bed, to the venous side of the circulation. **B.** Hyperoxic vasoconstriction. Closure of precapillary sphincters causes shunting of blood away from metabolically active capillary bed through metarterioles or other arteriovenous anastomoses. Constant metabolic demand in capillary bed therefore leads to increased glucose extraction from capillary blood and reversal of the usual capillary-venous BGL ratio. Numbers are notional BGLs in mmol·L^{-1}



Discussion

This study assessed the accuracy and reliability of blood glucose values obtained from fingertip capillary samples in 10 euglycaemic non-diabetic participants tested by POC glucometer in hyperbaric conditions against contemporaneous venous serum blood glucose samples.

Self-monitoring blood glucose systems are regulated under several parameters including safety and reliability testing, analytical performance evaluation, and accuracy. The International Organisation for Standardisation stipulates the minimum accuracy criteria required from these systems. The latest version released in 2013, ISO 15197 is more rigorous than the first version of the standard; 95% of blood glucose results must fall within ± 0.83 mmol·L^{-1} at glucose ≤ 5.55 mmol·L^{-1} and within ± 15% at glucose ≥ 5.55 mmol·L^{-1}.[41] The FreeStyle Optium™ Neo glucometer meets the ISO 15197:2013 standard and was assessed in accordance with this tertiary referral hospital's institutional risk-assessment procedures[24] and was therefore deemed safe for use under therapeutic hyperbaric conditions.

In this study it was noted that capillary BGL measurements by POC glucometer were higher than laboratory-measured venous serum levels under normobaric conditions. This was consistent with the expected physiological decrement in BGL when sampling at different points along a metabolically active vascular bed. Although venous serum levels are often viewed as the 'gold standard' against which other measurements are validated, this is misleading as there is no single absolute BGL within the body at any given point in time. Glucose is the primary fuel source for most cellular activity under normal conditions and, as would be anticipated from first principles, the BGL progressively falls from the arterial 'delivery' side of the vascular bed to the venous 'drainage' side, with the capillary BGL being somewhere in between (Figure 3A). From a clinical perspective, it is the glucose delivery to the end-user tissues that is of paramount importance – and, it can be argued, this is better represented by the capillary sample than a downstream venous sample.

Hyperoxia, as encountered in HBOT, is a profound systemic vasoconstrictor. Constriction of pre-capillary arterioles diverts blood away from the capillary bed through arteriovenous anastomoses, increasing the shunt fraction, and resulting in arterial blood more directly reaching the veins. In the context of blood glucose levels this will lead to a relative elevation of the venous BGL in comparison to the capillary level (Figure 3B). This expected reversal of the normal capillary:venous BGL ratio during HBOT, secondary to vasoconstriction in the fingertips, has been previously reported in a limited sample (n = 4) during HBOT.[2]

From a technical perspective POC glucometers require a single use test strip to be inserted into the glucometer, one of three enzymes (glucose dehydrogenase, GD; glucose oxidase, GO; or hexokinase, HK) impregnated into the strip.[42,43] The enzyme acts on the whole blood obtained from a finger-prick taking 4 seconds to display a digital figure representative of capillary glucose on the POC glucometer.[44] All manufacturers of POC glucometers work within ISO 15197:2013 which requires that test strips should demonstrate 95% accuracy.

The effectiveness of the enzymatic reaction and thus accuracy can be influenced by many factors. Specifically – haematocrit or contaminant interference, difference in strip batches and strip rotting (an issue if the strips are stored in-chamber due the repeated adiabatic temperature changes during compression/decompression).[45] However, a significant consideration for the hyperbaric physician is the effect of partial pressures of oxygen (PPO_2) in the HBOT environment. A bench study by Tang et al., on venous serum from diabetics indicated oxygen can lower glucose measurements obtained with GO-based amperometric test strips.[18] This supports evidence from multiple studies that note issues with the enzyme reaction of the test strips used in a glucometer in hyperbaric and hypobaric environments and the respective PPO_2.[9,20,46,47] Test strips at this institution's hyperbaric unit, including those used in this study, are therefore routinely stored outside the chamber and taken inside only as required to minimise this potential confounder.

Conclusion

The FreeStyle Optium™ Neo H glucometer meets clinical requirements and provides a reliable measurement of blood glucose in the HBOT environment. The results of this work are consistent with the changes anticipated (from first principles) in BGL measurements under HBOT conditions. Capillary BGL is higher than venous pre-HBOT but falls to the same or lower levels during HBOT as peripheral vasoconstriction leads to an increased shunt fraction and the diversion of blood away from skin capillaries. This results in (i) the 'arterialisation' of venous blood glucose levels and (ii) increased glucose extraction from the reduced blood supply in the capillary bed in order to meet the stable metabolic demands of end-organ tissues, leading to a reversal of the normal venous:capillary BGL ratio.

The concept of what constitutes the 'gold-standard' BGL needs to be reconsidered in the hyperbaric context. Ultimately, it is the adequacy of glucose supply to end-user tissues that is of clinical relevance. Venous samples processed in an accredited laboratory may be more consistently accurate, but (i) delays in sample processing may cause artefactual reduction in BGL and (ii) measurement downstream of the metabolically-active end-organ vascular beds (and/or vascular shunts) renders interpretation of results problematic. Arterial samples provide a better assessment of the adequacy of glucose supply but are technically more difficult to perform, and painful for the patient. We would argue strongly that point-of-care testing of capillary samples should be regarded as the gold-standard in clinical practice. Capillary POC testing avoids delays in sample processing and provides the single most relevant blood glucose value irrespective of microvascular flow changes: the BGL present at the level of the end-user tissues.

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Short communication

Experimental use of flow cytometry to detect bacteria viability after hyperbaric oxygen exposure: Work in progress report

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Bacteria; Flow cytometry; Hyperbaric oxygen treatment; Hyperoxia; Wounds

Abstract
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Introduction: Hyperbaric oxygen treatment (HBOT), based on inhaling pure oxygen under elevated ambient pressure, is used as adjuvant intervention to promote healing in infected wounds. Despite extensive clinical evidence of beneficial effects of HBOT in soft tissue infections the mechanism of action remains to be elucidated. The aim of this study was to evaluate the use of flow cytometry as a novel method to assess the viability of pathogenic bacteria after hyperbaric oxygen (HBO) exposure.
Methods: Bacterial strains associated with soft tissues infections: *Escherichia coli, Klebsiella pneumoniae, Pseudomonas aeruginosa, Staphylococcus aureus* were exposed to oxygen at 2.8 atmospheres absolute (atm abs) (283.6 kPa) pressure for 45, 90, or 120 min, then stained with propidium iodide and thiazole orange and analysed by flow cytometry.
Results: *Escherichia coli and Staphylococcus aureus* showed no change in viability, nor morphology, the viability of *Pseudomonas aeruginosa* reduced in a dose-dependent manner and *Klebsiella pneumoniae* also showed dye uptake after HBO.
Conclusions: These initial results, indicate diverse sensitivity of bacteria to HBO, and suggest that flow cytometry can be used to monitor viability and morphological changes triggered by HBO exposure in bacteria.

Introduction

Hyperbaric oxygen treatment (HBOT) involves inhaling pure oxygen under elevated ambient pressure, and is a treatment option for wound healing, carbon monoxide intoxication, arterial gas embolism, and decompression sickness.[1–3] It is used, together with proper wound debridement and antibiotic therapy, as adjuvant treatment for various soft tissue infections, mainly anaerobic, mixed or necrotising.[2,4] Despite the clinical evidence, the underlying mechanisms are still not fully understood. Elevated generation of oxygen reactive species impeding bacteria metabolism and supporting the immune system in bacteria elimination coupled with denaturing bacterial toxins seem to be the most applicable.[5,6]

Accurate determination of live, dead, and total bacteria plays a critical role in clinical and experimental microbiology. In addition, the knowledge of bacterial cell morphology is required for the understanding of the probable mechanisms of action of biocides. Traditionally, the viability in bacteria is determined by the ability to form colonies on solid growth medium and to proliferate in liquid nutrient broths. These traditional, culture-based tests work poorly in slow-growing and non-culturable organisms and are time-consuming. Flow cytometry, a technique first applied to eukaryotic cells, has been previously adapted for quantification, viability and single cell analysis of bacteria.[7–9]

Despite extensive clinical evidence of beneficial effects of HBOT in soft tissue infections the mechanism of action remains to be elucidated. To do so, applicable molecular and analytical methods should be employed. The aim of this prelimnary study was to evaluate the use of flow cytometry as a method for the specific quantification of viable and non-viable bacteria after hyperbaric oxygen exposure.

Methods

BACTERIAL STRAINS

Four bacterial strains associated with soft tissue infections were chosen: *Escherichia coli, Klebsiella pneumoniae, Pseudomonas aeruginosa, Staphylococcus aureus*. These strains can behave as opportunistic pathogens, particularly in burns, septicaemia, endocarditis, soft tissue infections, urinary tract infections, and pneumonia (see e.g.,[10]). Bacterial strains were obtained either from the Czech National Collection of Type Cultures (CNCTC) or were isolated from patients. Strains isolated from patients underwent determination in our department, which serves as the Czech National Anaerobic Bacteria Reference Laboratory in the Czech Republic.

Escherichia coli

Gram-negative, facultative anaerobe, commensal microorganism of the gut microbiota. Various serotypes of *Escherichia coli* are human pathogens.

Klebsiella pneumoniae

Gram-negative, facultative anaerobe, routinely found in the human nose, mouth, and gastrointestinal tract as a part of the normal microbiota. *Klebsiella* species are often associated with nosocomial (hospital) infections.

Pseudomonas aeruginosa

Gram-negative, aerobe, *Pseudomonas aeruginosa* is a multidrug-resistant pathogen, often causing nosocomial infections.

Staphylococcus aureus

Gram-positive, facultative anaerobe, member of the normal flora of the body, it can cause a wide variety of diseases in humans including skin infections, abscesses, respiratory infections, and food poisoning. *Staphylococcus aureus* is the primary cause of bacteraemia in humans.

HYPERBARIC OXYGEN (HBO) EXPOSURE

Each bacterial strain was inoculated four times at 1×10^8 CFU·ml^{-1} in Schaedler Broth (HiMedia, India). One sample served as a control and three other samples were exposed to 100% oxygen at 2.8 atmospheres absolute (atm abs) (283.6 kPa) in an experimental hyperbaric chamber (HAUX Testcom 400, Germany). Three exposure times (45, 90 and 120 min) were used to identify possible dose-response effects on bacteria viability. One culture of each bacterium was subjected to HBO for each of these exposure times. The compression and decompression phases were each completed over two minutes. The inner chamber temperature was monitored, and maintained at room temperature throughout the exposure. The whole sample was subsequently analysed by flow cytometry after HBO exposure.

FLOW CYTOMETRY

Flow cytometry (FC) is commonly employed for quantification of viable bacteria in laboratory cultures, environmental, clinical and food samples.[11,12] Bacterial populations can be characterized by analysing their forward scatter (FSC), as well as their side scatter (SSC). The general assumption is that FSC is correlated to cell size and SSC represents cell density or granularity. In addition, staining with a variety of probes allows for a deeper understanding of bacterial membrane integrity, membrane potential, enzymatic and metabolic activity, culture structure and dynamics.[13]

Viability of bacteria is usually determined with FC after culture staining with various fluorescent dyes. This includes DNA binding dye probes, such as propidium iodide, which is impermeable in cells with an intact membrane, and only leaks into cells with compromised membranes.[14] Detection of non-viable bacteria with propidium iodide is often coupled with thiazole orange staining, which is a permeable nucleic acid dye, which stains live and dead bacteria.[7,9]

In our study, the viability of bacterial strains was analysed by flow cytometry (FACSCalibur, BD Biosciences, USA) after each HBO exposure. To do so, 0.5 ml of each sample was mixed with a combination of propidium iodide and thiazole orange, 0.5 µL each (BD Biosciences, USA), incubated for 20 min at room temperature and processed. The FSC and SSC together with propidium iodide absorption were used to detect bacteria viability and cell membrane integrity. Data were displayed as a dot-plot diagram. Briefly, more dot-plot diagram distribution in FSC and SSC parameters indicates bacterial subpopulations with changed cell morphology (e.g. cell swelling), and when associated with corresponding heterogeneity in propidium iodide absorption indicates cells with compromised membranes (e.g., dying bacteria). The data were analysed by FACSDiva (BD Biosciences, USA) software.

Results

Bacterial strains exposed to HBO exhibited differing patterns in the measured parameters. In *Escherichia coli* and *Staphylococcus aureus* no obvious difference was observed in FSC or SSC between the control sample (normobaric air, room temperature) and samples exposed to 45, 90 and 120 min of O_2 at 2.8 atm abs. *Pseudomonas aeruginosa* reacted to increased doses of HBO with the appearance of a visible population of bacteria with changed morphology, also associated with increased propidium iodide absorption (Figure 1). HBO exposure in *Klebsiella pneumoniae* led to the appearance of a visible population of bacteria with increased propidium iodide absorption, but without

Figure 1

Flow cytometric analysis of *Pseudomonas aeruginosa* before (K) and after 45, 90 and 120 min of exposure to HBO (2.8 atm abs O_2). **A:** X axis – size of bacteria (forward scattered light, FSC), Y axis – cell granularity (side scattered light, SSC); **B:** X axis – bacteria stained with propidium iodide, Y axis – SSC; **C:** X axis – bacteria stained with thiazole orange, Y axis – SSC. Morphological and viability changes are highlighted (black squares)

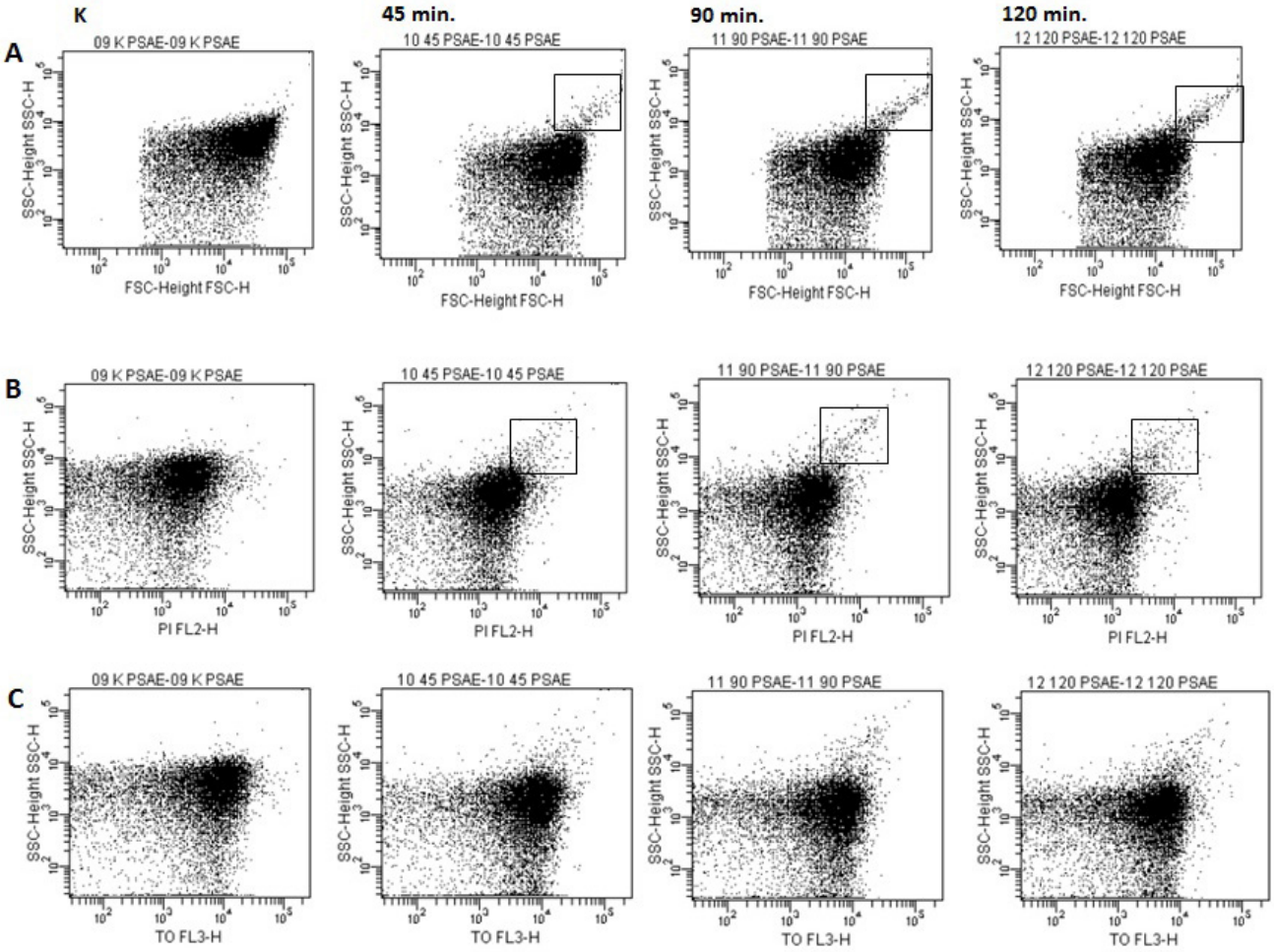

Table 1

Summary of changes in bacterial morphology and dye absorption after HBO exposure

Bacterial strain	Morphological change	Absorption of fluorescent dye
E. coli	Not visible	Not visible
S. aureus	Not visible	Not visible
P. aeruginosa	Dose-dependent	Dose-dependent
K. pneumoniae	Not visible	Increased

associated changes in bacterial morphology. The results summary is showed in Table 1.

Discussion

USE OF FLOW CYTOMETRY FOR BACTERIAL VIABILITY DETERMINATION

The conventional gold standard test for microbial contamination, a plate count method, can take up to several days to complete. Bacterial cell concentrations obtained by FC enable quantification of the entire bacterial community instead of the fraction of cultivable bacteria detected with the plate count method (typically < 1% of all bacteria). Flow cytometry measurements are reproducible with relative standard deviations below 3% and can be available within 15 min of samples arriving in the laboratory. The main advantages of FC are relevance, speed, accuracy, costs and automation potential.[15,16] Flow cytometry also facilitates understanding of physiological diversity in seemingly likewise acting populations when additional staining is applied. For practicality in microbiology, repeated division of a cell on an agar surface to produce a visible colony is usually taken as evidence of viability. Interpreting the

situation where there is an absence of colony formation is not clear-cut. Nowadays we understand that there is a discrepancy between the presence of bacteria and its ability to replicate under given conditions, so-called 'viable but non-culturable' bacteria. In this regard, it is crucial to further focus on reproductive growth, membrane potential, metabolic activities, and membrane integrity to better understand culture heterogeneity and viability. The advance of a direct single-cell examination using FC measurements accompanied by staining of bacteria with a variety of fluorescent probes facilitates this aim.[13,17]

THE SENSITIVITY OF BACTERIA TO HYPERBARIC EXPOSURE

The aim of this study was to demonstrate the use of flow cytometry in assessing viability of pathogenic bacteria after HBO exposure. In doing so, we identified apparent inter-specific differences in cytometric responses, possibly indicating diverse sensitivity of bacterial species to HBO (though see the study limitations below). These preliminary results are broadly confluent with the findings of others authors, who did not use flow cytometry to investigate effects of oxidative stress on bacteria.[18–20] Despite the relatively wide use of HBOT in soft tissue infection, data on the sensitivity of different bacterial strains to elevated partial pressures of oxygen are sparse and further work is required. We have identified FC as a plausible outcome measure in related future HBO studies.

LIMITATIONS

This study reports an attempt to utilise FC as a universal tool allowing for rapid detection of alive, impaired and dead bacteria after HBO exposure. Despite our promising results, some cautions should be taken. Firstly, we used bacterial isolates while the infected wound is usually colonised by a variety of normal and pathogenic bacterial flora. Thus, the observed reaction of bacteria to HBO might be over- or underestimated, since multi-strain colonies might behave differently. Secondly, some of the observed results might be transitory, thus evaluating the cultures at longer time points might be needed to fully understand the effect of the elevated partial pressure of oxygen on bacteria. Finally, due to the study design (one sample per bacterial species per exposure time) the results can only be considered hypothesis-generating. Further work with greater number of samples and possibly with sampling at different time intervals after HBO exposure is required to further determine the response of these bacteria to HBO.

Conclusions

To our knowledge, this is the first attempt to measure the direct effect of HBO exposure on bacteria viability with flow cytometry. The use of flow cytometry, originally designed for eukaryotic cells, has been extended to quantification, viability assessment and single cell analysis when staining with different fluorochromes. Our initial results suggest that flow cytometry can be used to monitor viability and morphological changes triggered by HBO exposure in bacteria. Nevertheless, further research is required to further investigate our results as well as to test other capabilities of this method while studying the effect of hyperbaric oxygen on bacteria.

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Assessment of a dive incident using heart rate variability

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Key words
Diving; Incident; Holter monitor; ECG; Autonomic nervous system; Heart rate variability

Abstract
(Zenske A, Koch A, Kähler W, Oellrich K, Pepper C, Muth T, Schipke JD. Assessment of a dive incident using heart rate variability. Diving and Hyperbaric Medicine. 2020 June 30;50(2):157–163. doi: 10.28920/dhm50.2.157-163. PMID: 32557418.)
Introduction: Scuba diving likely has an impact on the autonomic nervous system (ANS). In the course of conducting trials of underwater ECG recording for measurement of heart rate variability, there was an unexpected stressful event; one participant's regulator iced and began to free-flow.
Methods: A custom-made, water- and pressure-tight aluminum housing was used to protect a portable Holter monitor. ECGs were recorded in three experienced divers who witnessed an unplanned moderately stressful incident during diving. The ECG signals were analysed for measures of heart rate variability (HRV).
Results: Analysis for different short-term HRV measures provided consistent results if periods of interest were appropriately time-aligned. There was improvement in sympatho-vagal balance. One diver unexpectedly exhibited an increase in both sympathetic and vagal activity shortly after the incident.
Conclusions: A conventional open-water dive affected the ANS of experienced recreational divers as measured by HRV which provides a global evaluation of the ANS and alterations in its two branches. The heart rate variability data gathered from several participating divers around the time of this event illustrate the potential utility of this variable in quantifying stress during diving. HRV data may be useful in addressing relevant diving related questions such as effects of cold, exercise or different breathing gases on ANS function.

Introduction

Only a few studies have addressed the effects of scuba diving on the autonomic nervous system (ANS) of recreational divers.[1–3] Given that there are as many as six million[4] to seven million[5] active scuba divers worldwide with 500,000 more training every year,[5] studies on recreational divers seem relevant.

Heart rate is characterised by an inherent variability, i.e., the healthy heart is not a metronome.[6] Heart rate variability (HRV) is the result of neuronal control by the ANS,[7,8] and HRV is of extreme importance for adaptation and flexibility, which is lost in some cardiac and non-cardiac diseases in which heart rate becomes increasingly periodic.[9–11] On the other hand, persistently high HRV in the elderly represents a predictive marker of longevity.[12] More than 160 review articles in the last five years alone forcefully underline the importance and utility of measuring HRV.

Submersion and scuba diving cause physiological stress thereby activating the sympathetic nervous system.[13,14] In turn, the diving response activates the parasympathetic nervous system,[1,3,15] which, in addition, is activated by hyperbaric hyperoxia.[16,17]

So far, several systems for collecting underwater ECG data of recreational[18] or professional[19] divers have been described but no simple ways seem to exist to collect physiological data.[2] Thus, knowledge about the effects of deep dives on the ANS is limited. We have used a custom-made system to assess ECG data for wet and dry dives down to depths of 40 m.

In the course of conducting trials of underwater ECG recording for measurement of heart rate variability, there was an unexpected stressful event; one participant's regulator iced and began to free flow. This case, along with commentary on measurement and interpretation of heart rate variability in divers is presented in this report.

Table 1
Subject characteristics. BMI = body mass index in kg·m^2

Diver	ECG system	Age years	BMI	Dives
Leader	No	28	24	600
Victim	No	36	27	300
A	Yes	42	26	650
B	Yes	38	24	26
C	Yes	48	36	1,100

Methods

PARTICIPANTS

The group consisted of five divers. All divers were experienced and familiar with the dive site. Only three wore the ECG system. Personal and dive data of all five members are presented in Table 1.

The dive was performed within the framework of a study to investigate possible different effects between breathing air and nitrox on the ANS. The protocol of this study was approved by the ethical committee of Charité – University Medicine Berlin. The divers were informed about the study via written information. Informed consent was obtained from each of the participants.

UNDERWATER ECG SYSTEM

A Holter monitor was used for ECG recording. The Holter (Lifecard CF, Spacelabs Healthcare, Nürnberg, DE) uses a 12-bit resolution system to ensure accurate recordings. Lifecard can record seven days of ECG data on removable compact flash cards with a memory of 64 GBytes. These cards can be read on personal computers.

The recorder was placed in a custom-made water- and pressure-tight aluminum cylinder. The ECG leads had a length of 1.5 m. Push buttons on their distal ends permitted connection with adhesive electrodes. They were positioned on the chest wall of each participant (subclavicular right, subclavicular left and position V6). This equipment was deployed inside a dry suit.

The cylinders were equipped with a handle to allow them to be attached to the front of a belt worn around the waist within the dry suits (Figure 1A). However, they could also be worn outside the wet suits in which case the electrodes should be sealed using elastic, waterproof polyethylene plasters (Figure 1B).

ECG ANALYSIS FOR HRV

ECG signals were recorded continuously before, during and after diving. ECG analysis to evaluate dive-induced changes to the ANS was performed in the facilities of the German Naval Medical Institute (Maritime Medicine, Kronshagen, DE) using conventional software (Pathfinder SL, HRV-Tools, Spacelabs Healthcare, Feucht, DE).

Beside the heart rate (HR), two short-term HRV measures from the time domain were employed: standard deviation of NN periods (SDNN); and root mean square of successive RR interval differences (RMSSD). In addition, low frequency (LF) power, high frequency (HF) power, and their ratio (LF/HF) from the frequency domain were employed.

The ECG signals were analysed over a 5-min period shortly after the onset of the dive and another period of 5-min before surfacing. Another two 5-min periods were analysed: one before: and another after the incident. Finally, two 2-min periods were analysed: one before and the other after the incident. All employed measures were intended to reflect short-term HRV, that is, changes within a period of between 2 and 5-min.[20]

DIVE EQUIPMENT AND BREATHING GAS

The divers wore dry suits. They used 15 L cylinders, two independent, cold water-suitable regulators and a dive computer. This equipment complies with the standards of safe diving in cold water. The cylinders were filled with 3,000 L nitrox 40 (40% oxygen and 60% nitrogen).

DIVE LOCATION AND PARAMETERS

Lake Walchensee is located 805 m above sea level in the Bavarian alpine upland. Water visibility was up to 20 m and the temperature was 10°C at the surface and 5°C at a depth of 25 metres' fresh water (mfw). Dive tables of the trade associations were employed to determine the no-decompression time[21] that permitted a bottom time of 25 min at a depth of 25 mfw. However, correction for the altitude resulted in a decompression of 5 min at 3 mfw. For safety reasons, two more safety stops were to be completed: 1 min at 9 mfw and 3 min at 6 mfw. Taking the ascent rate (max. 10 m·min^{-1}) into account the total planned dive duration was 39 min.

Because of a regulator icing in one of the group, the dive was truncated at 14 min run time, but the intended stops were accurately observed (Figure 2).

Results

INCIDENT

After an uneventful descent to 25 mfw over 5 min, the dive continued for another 9 min, when the first stage of the victim's regulator iced, and there was uncontrolled free-flow from the second stage. The victim was unable to close the valve of the first regulator and swap to his second regulator. The group leader successfully closed the valve and handed the victim the second regulator permitting him to commence

Figure 1
The water- and pressure-tight cylinder contains the Holter monitor. The device can be worn inside a dry suit (**A**) or with a wetsuit by using waterproof dressing-protected electrodes (**B**)

breathing again. Because the group had performed the dive in close contact and with good visibility, the three other divers (A, B, and C) witnessed the incident straightaway. It was decided to abort the intended dive plan and to return to the entry, strictly observing the planned ascent schedule. On their way back, the victim was closely accompanied by the leader of the group on one side and by diver C on the other side. Divers A and B closely followed this trio.

ELECTROCARDIOGRAM

Physical activity associated with putting on and taking off the dive gear, as well as by entering/leaving the water, did affect the signal quality due to motion artifacts. However, during diving, the proportion of regular beats was ≥ 80%, enabling reasonable analyses for HRV measures. As a consequence, data sets from participants A, B and C could be analysed.

Heart rate

The average HR in divers A, B and C clearly decreased (in comparison to pre-dive) during the dive, from 87 to 73 min^{-1} (16%), 127 to 101 min^{-1} (20%), and 123 to 92 min^{-1} (25%), respectively. However, there were major fluctuations in rate during the dive (Figure 3).

After the incident HR quickly changed to a different degree and for a different duration. For the 5-min post-incident periods HR increased from 82 to 92 min^{-1} in diver A, from 103 to 124 min^{-1} in diver B, and from 84 to 95 min^{-1} in Diver C.

Tachogram

The RR intervals increased between the beginning and the end of the dive. Shortly after the onset of the incident, the average RR intervals decreased in divers A, B, and C. Figure 4 is presented as representative example (diver A). This tachogram additionally exhibits rhythmic oscillations. Before the incident their frequency was about 6 min^{-1} and was slightly increased to about 7 min^{-1} after the incident. The frequency comparably increased from 8 to 9 min^{-1} and from 6 to 7 min^{-1} in divers B and C, respectively. The amplitude of the oscillations immediately after the incident was decreased in all three divers compared with the amplitude before the incident (see diver A in Figure 4).

Heart rate variability

Because of the small sample size, only qualitative changes in the two time-domain and the three frequency-domain measures are presented. When analysed over the entire dive,

Figure 2
Planned dive profile. After 14 min runtime (arrow) the dive was terminated with observance of the planned stops as shown

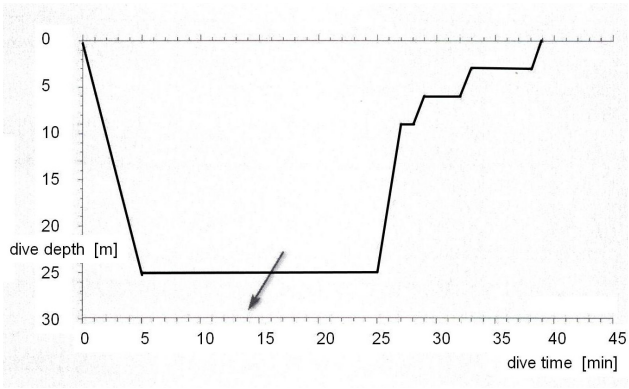
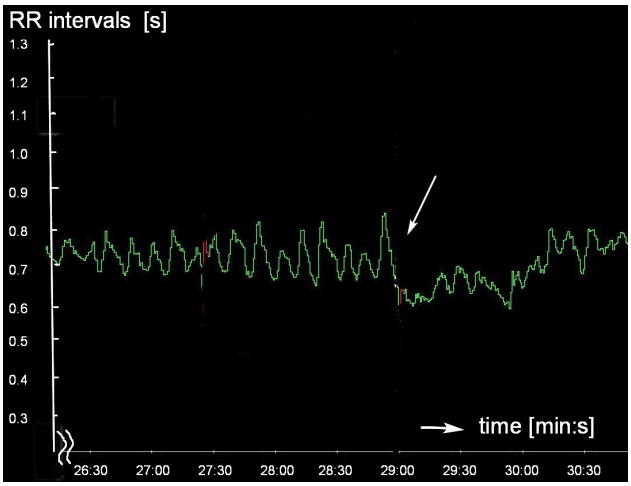


Figure 4
Tachogram of diver A. The arrow indicates the incident. Shortly after it, the RR intervals decreased together with their amplitude. Also, the frequency of the rhythmic oscillations was somewhat increased after the incident compared with before the incident



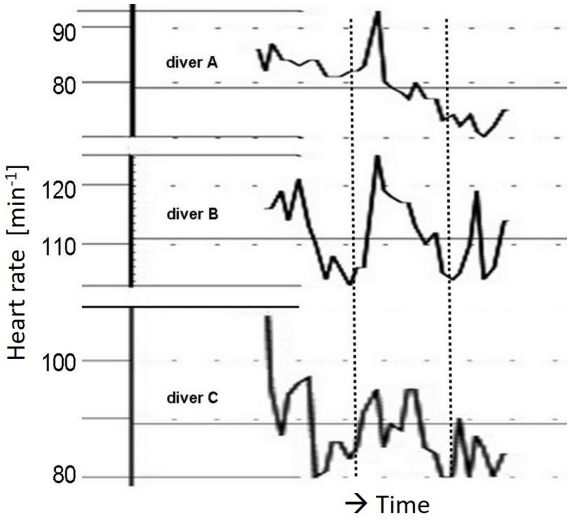
SDNN and RMSSD increased in all monitored divers. LF and HF were also increased but to differing extents such that LF/HF was decreased in all three divers (Table 2).

When analysed for the 5-min periods before and after the incident, the two time-domain and the three frequency-domain measures had changed less uniformly. When comparing these specific periods LF/HF was decreased in divers A and B and was increased in diver C (Table 2).

Finally, when analysed for the 2-min periods, the two time-domain measures were all increased after the incident, as well as LF and HF. Surprisingly, LF/HF was decreased in diver A but increased in divers B and C (Table 2).

Because the 2-min data for diver A were unexpected, an original diagram of the time course of the LF- and HF-power as well as the LF/HF ratio is provided (Figure 5). The diagram

Figure 3
Heart rate (HR) of divers A, B and C. HR clearly decreased between the start of the dive (leftmost point on the charted line) and prior to surfacing (right dotted line). The left dotted line indicates the onset of the incident following which the HR in all three divers rapidly increased. The horizontal dashes indicate five-minute intervals



shows a simultaneous increase of both the LF- and the HF-power at the time of the incident that lead to a moderate decrease in the LF/HF ratio. In both of the other divers LF/HF was increased.

Discussion

Within the framework of the underlying study, about 200 dives were performed without Holter housing failures. Moreover, the system did not impede the divers, as it was worn on a belt around the waist inside of dry suits. The unexpected recording made during a diving incident illustrated the potential for the system to detect major alterations of ANS function during diving.

HR was decreased in all three divers between the onset and the end of the dive. This result agrees with previous studies on recreational scuba diving, demonstrating an activated vagal system in experienced divers;[1,3,22] a finding also reported in children.[23]

Analysis of shorter ECG periods showed a transient increase in HR in response to the witnessed incident. Divers A, B and C witnessed all events described above, and the resulting stress lead to increases in individual HR.

The tachograms of divers A, B and C exhibited cyclic variations with a frequency between 6 min^{-1} and 9 min^{-1}. These variations are associated with breathing and are known as respiratory sinus arrhythmia.[24] The respiration rate was only moderately increased after the incident in divers A, B and C. Still, tachograms provide the respiration rate as one valuable physiological measure.

Table 2

Changes in HRV parameters over the entire dive, and when comparing the five- and two-minute periods before and after the incident in divers A, B and C. LF = low frequency power; HF = high frequency power; RMSSD = root mean square of successive RR interval differences; SDNN = standard deviation of NN periods

Measure	Periods	A	B	C
SDNN	entire	↗	↗	↗
	5-min	↘	↗	↗
	2-min	↗	↗	←→
RMSSD	entire	↗	↗	↗
	5-min	↘	↗	↘
	2-min	↗	↗	↗
LF	entire	←→	↗	↗
	5-min	↘	↗	←→
	2-min	↗	↗	↗
HF	entire	↗	↗	↗
	5-min	↘	↘	↘
	2-min	↗	↗	←→
LF/HF	entire	↘	↘	↘
	5-min	↘	↘	↗
	2-min	↘	↗	↗

HRV allows the cardiovascular system to rapidly adjust to sudden physiological and psychological challenges to homeostasis.[25] In this regard, HRV is a reliable indicator of health status and a sensitive index of autonomic stress reactivity.[26] Thus, this non-invasive tool provides information about the sympathetic and the parasympathetic (vagal) systems and the interaction of both ANS branches.

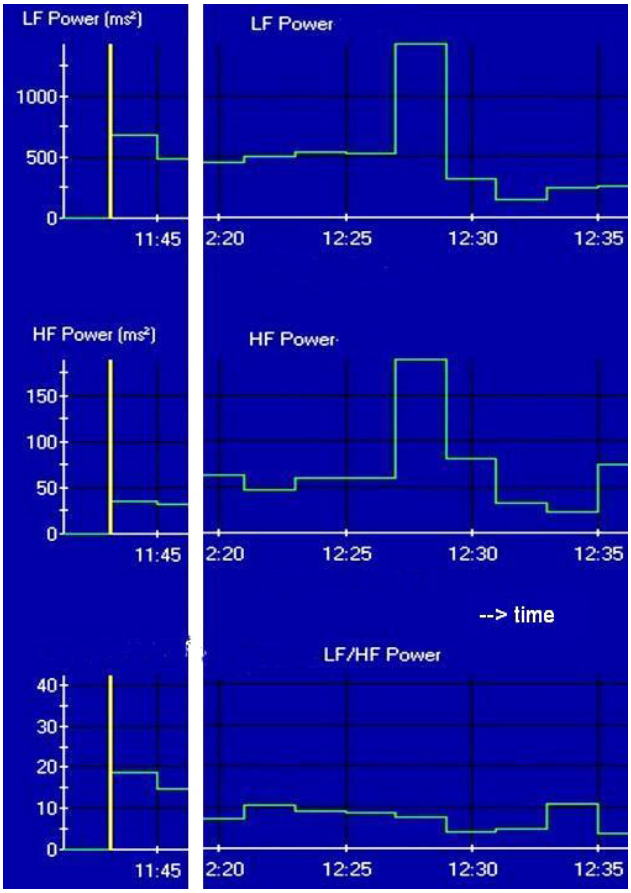
Submersing the face into water elicits the 'dive response'[15,27] including a bradycardia as the best-known manifestation.[28,29] On the other hand, as man is not made to live under water, submersion and diving will likely present stress. Thus, both the vagal and the sympathetic nervous system should be activated during diving.[3] Stress will further increase if the face is submersed into cold water. The strong and simultaneous activation of the two ANS branches has been termed autonomic conflict and may account for arrhythmias and (potentially) death in some vulnerable individuals.[14]

We chose two measures from the time domain and three measures from the frequency domain to assess ANS activity resulting from the incident: The mean SDNN reflects all cyclic components responsible for variability during the interval of recording. In contrast, the RMSSD is a measure of vagal activity.

The low frequency power spectrum (LF: 0.04 to 0.15 Hz) overwhelmingly reflects sympathetic activity. However, vagal modulations are also involved, although at a much lower power. The high frequency power spectrum (HF: 0.15 to 0.40 Hz) is considered to reflect vagal activity. HF components are associated with the respiratory sinus

Figure 5

Left panel: LF Power, HF Power and LF/HF ratio for diver A with frequency domain measures starting after correct placement of the ECG-electrodes, i.e., in a dry environment. Right panel: Tracings start approximately seven minutes before the incident. The drastic and simultaneous increase in both LF and HF after the incident are of surprise. The resulting decrease in the LF/HF ratio is suggestive for an improved sympatho-vagal balance



arrhythmia where respiration is normal and varying between 9 and 24 breaths·min^{-1}.[30] In our three divers, maximum rate was 9 min^{-1} (0.15 Hz) and was thus located in the LF power spectrum. It had been previously reported that scuba diving is associated with surprisingly low respiration rates.[31] The LF/HF ratio is considered to present the sympatho-vagal balance.[32,33]

Not only can HRV describe overall changes in the ANS activity, but it also can differentiate which of the two ANS branches is activated and to what degree. HRV also permits measurement of the breathing rate.[24] Because of these advantages, the HRV has established its usefulness in describing health status.[26] HRV is also suited to assess stresses like noise, time constraints or shift work in the occupational environment.[34,35] Use of HRV should therefore, permit physiologists and other scientists to assess the effects of various stressors associated with scuba diving such as varying depths, exercise, different breathing gases, fast environmental changes (e.g., visibility, current), or

equipment-related problems. Normal values for HRV measures exist,[36–38] such that deviations from the standards can be detected.

Either a reduced HRV at rest or a non-physiological response during submersion could be used to identify scuba diving candidates who merit closer scrutiny to prevent events like ventricular arrhythmias,[39] or sudden cardiac death,[40] and parameters may help identify older divers at greater risk.[41] Stressful elements of training could be identified in novice divers enabling adjustment of a teaching schedule. This possibility holds for professional divers such as police, firefighters, and armed forces. HRV analyses could help identify stressing conditions for individual divers. If HRV analyses were available during accident investigation, they could contribute to identifying causes of fatal dive accidents.

We have the impression that assessment of HRV measures might support diver training, contribute to medical decisions, and foster scuba diving-related science.

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Adult attention-deficit/hyperactivity disorder prevalence among commercial divers in South Africa

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Key words
Epidemiology; Fitness to dive; Mental health; Occupational diving; Psychology

Abstract
(Van Wijk CH, Meintjes WAJ. Adult attention-deficit/hyperactivity disorder prevalence among commercial divers in South Africa. Diving and Hyperbaric Medicine. 2020 June 30;50(2):164–167. doi: 10.28920/dhm50.2.164-167. PMID: 32557419.)
Introduction: Adult attention-deficit/hyperactivity disorder (ADHD) is associated with increased chance of workplace accidents, psychiatric comorbidities, other risky behaviours and sophisticated psychopharmacological treatment. These factors all contribute to a potentially complex risk profile within the commercial diving context. In order to make informed decisions regarding ADHD and commercial diving, further description of this condition among commercial divers is required. This paper reports on a study that aimed to determine the prevalence of adult ADHD among commercial divers.
Methods: The study used a self-reporting survey-type questionnaire to determine likely diagnosis, based on *Diagnostic and Statistical Manual of Mental Disorders, 5th ed*. criteria, in a group of 245 commercial divers in South Africa.
Results: Fourteen cases (5.7% of the sample) met criteria for ADHD. The majority of the cases presented with combined type, and reflected mild forms of ADHD. Adult ADHD did not appear to occur in significantly different proportions across the biographical variables of age, education or diving qualification.
Conclusion: Based on this small survey, adult ADHD may be over-represented in commercial diving in South Africa, compared to general workplace populations. However, ADHD may not necessarily be a contra-indication to commercial diving.

Introduction

Adult attention-deficit/hyperactivity disorder (ADHD) is a condition characterised by a persistent pattern of inattention and/or hyperactivity-impulsivity that demonstrably interferes with social, academic and/or occupational functioning.[1] Established epidemiological reports for workplace samples suggest a prevalence of around 3.5% (1.2–7.3%, dependent on country) for ADHD among adults.[2,3]

Prevalence of ADHD within the commercial diving community has not yet been established. Adult ADHD is of concern in the diving medical field as it may contribute to a more complex risk profile within the commercial diving context through a number of mechanisms. Firstly, the core symptoms of ADHD reflect challenges to self-regulation, which may compromise safety performance underwater, for example, through distractibility or impulsivity. Secondly, adult ADHD has been associated with significant functional impairment in the workplace, and with an increased risk of accidents and workplace injuries, with 2.0 relative-odds of workplace accidents-injuries calculated.[2,4–7] Thirdly, comorbid disorders are common in adults with ADHD, with substance use, mood, and anxiety disorders most prominent, which may all influence a diver's risk profile. This may require careful consideration of the effect of both the co-morbid conditions, as well as potential psychopharmacological treatment, on safety performance.[1,8–13] ADHD is further associated with risky behaviours across multiple activity domains.[14]

Lastly, medical management of adult ADHD, often in the form of psychostimulants, raises concerns regarding neuropsychological performance under hyperbaric conditions. For example, hyperbaric conditions encountered during deeper diving are associated with central nervous system suppression as a result of hyperbaric nitrogen exposure, and although no direct data exist, it has been hypothesised that these conditions may render stimulant medication less effective. Very deep saturation divers may experience central nervous system stimulation (i.e., high-pressure neurological syndrome), which could also be hypothesised to change the effect of stimulant medication.[15] Further, the use of stimulants may also pose an increased risk for oxygen toxicity when a diver is exposed to elevated partial pressures of oxygen, while drugs that cause sedation may have an increased effect under hyperbaric conditions.[16]

Expert opinion guidelines for ADHD in recreational diving have recently been published, and constitute a valuable

resource when considering risk among amateur divers.[17] In order to make informed decisions to guide concerns regarding ADHD and commercial diving, further description of ADHD among commercial divers is required. This paper reports on a study that aimed to determine the prevalence of adult ADHD among commercial divers undertaking their diving medical examination in South Africa over a one-year period from May 2018.

Methods

The study was approved by the Health Research Ethics Committee of Stellenbosch University (#N18/03/039), and all participants provided written informed consent to partake in the study.

There is a legal requirement to capture all commercial diving medical examinations in South Africa within the online Southern African Undersea and Hyperbaric Medical Association (SAUHMA) database. Diving medical practitioners (DMPs) on the SAUHMA database were invited to participate and issue questionnaires to divers. Seven DMPs (of 37 active DMPs) provided questionnaires completed during annual diving medical examinations.

MEASUREMENTS

Adult ADHD was assessed using the self-report Adult ADHD Symptom Scale for DSM-5 (AASS-5). The AASS-5 consists of 21 items in question format which are formulated according to DSM-5 criteria A to D.[1,3] The scale can be used to calculate a total ADHD score, as well as determine diagnostic cases based on current DSM-5 criteria. The scale is described in detail elsewhere.[3] The nature of the questionnaire also allowed comparison to older data reported in terms of the World Health Organisation's (WHO) so-called Set-A criteria for ADHD, which uses a limited-symptom calculation to identify diagnostic cases.[3,18]

DATA ANALYSIS

Prevalence figures were calculated as cases that met current DSM-5 criteria, expressed as percentage of the total sample. The same was done for cases that met the WHO's Set-A criteria, in order to compare with older population studies. Severity of ADHD could not be directly calculated, but could be indirectly inferred from response feedback analysis, and reported here as estimates. Associations with biographical categories were calculated using Chi-Square analyses.

Results

A total of 263 surveys (from 482 examinations) were returned from seven DMP practices, giving a 55% participant uptake for these practices and representing 11% of medical examinations (namely 2,307) performed in South Africa during this period. Of the 263 surveys returned, 18 were excluded from the final dataset (13 were incomplete, and five constituted a second entry). The analysis below is based on 245 qualifying participants. The sample had a mean age of 33.1 years (SD 10.3). Twenty-nine participants were qualified as diving supervisors. Further sample composition information can be found in Table 1.

According to current DSM-5 criteria, 14 participants (5.7%; 95% CI 2.9%–8.5%) doing their diving medical examination in South Africa would qualify for a diagnosis of adult ADHD. Using the WHO Set-A criteria, five participants (2.0%; 95% CI 0.4%–3.7%) would qualify for an ADHD diagnosis. Most of the DSM-5 diagnosed cases presented with combined type (eight participants, 3.3%; 95% CI 1.2%–5.4%), with inattentive (four participants, 1.6%; 95% CI 0.1%–3.1%) and hyperactive/impulsive type (two participants, 0.8%; 95% CI 0%–1.9%) representing smaller segments. Severity estimates (according to total score interpretation) suggested that 11 participants (79%) reflected mild and two (14%) moderate forms of ADHD. One completed questionnaire may have reflected a slightly more severe expression of ADHD.

Analysis of the prevalence data showed that age had no significant effect on scores ($r = -0.016$, $P = 0.8$). Neither did ADHD appear to occur in statistically significant different proportions for language, education, or diving qualifications ($P > 0.3$ in all cases).

Discussion

For comparison, a general South African workplace sample recently reported a 3.3% prevalence rate of ADHD using DSM-5 criteria, while a South African Navy diver sample reported a 2.6% prevalence, using comparable methodologies.[3,19] The reason for the apparent higher prevalence in this sample of 245 commercial divers is unclear. From a methodological perspective, sampling biases and the self-reporting nature of the data may have contributed to an over-representation of diagnostic cases in this sample. From a workplace context perspective it could be hypothesised that individuals with ADHD may be more likely to migrate to and remain in more practical-orientated occupations, such as diving. Alternatively, there may be a bias introduced within the diving industry, where divers are controlled by the supervisor and, therefore, are able to continue to function well, whereas they may not remain in other industries. Further, the positive cases in this sample likely reflected mild ADHD, which may be easier to accommodate in this occupational field.

It is postulated that the impact of ADHD in the commercial diver population may be small, since commercial diving activities are strictly controlled and regulated by the diving supervisor. This view is supported by a preliminary report suggesting that the rate of safety incidents in the highly controlled context of naval diving is not significantly higher among divers with ADHD than among those who do not meet diagnostic criteria.[19] The safety impact may be more

Table 1

Prevalence of ADHD among commercial divers in South Africa, per biographical category, according to the World Health Organisation's Set-A, and Diagnostic and Statistical Manual of Mental Disorders (5th ed.) criteria. Class I = saturation diving; Class II = mixed gas and open bell diving; Class III = surface-supplied diving; Class IV = scuba diving. None = the examination was performed prior to a commercial diving course

Variable	Category	*n*	Set-A		DSM-5	
			n	%	*n*	%
Language	Afrikaans	142	4	2.8	8	5.6
	English	69	1	1.4	5	7.2
	Other	34	0	0	1	2.9
Education	< 12 years	75	0	0	2	2.7
	12 years	142	5	3.5	11	7.7
	> 12 years	13	0	0	1	7.7
	Unknown	15	0	0	0	0
Dive qualification	None	50	1	2.0	4	8.0
	Class I	11	1	9.1	1	9.1
	Class II	77	1	1.3	2	2.6
	Class III	99	2	2.0	6	6.1
	Class IV	8	0	0	1	12.5

pronounced in commercial diving supervisors diagnosed with ADHD, and this hypothesis could be explored in future studies. The present study was purely descriptive, and did not examine any possible impact of ADHD symptoms on diving safety performance or rates of accidents/incidents. Further work is required to explore potential associations between ADHD diagnosis and safety incidents while diving or supervising.

Adult ADHD continues to be of particular interest to DMPs in terms of assessing risk to diving safety. Theoretically at least, ADHD remains a risk factor for safe diving through mechanisms such as distractibility, impulsivity, or the effects of medical treatment. The established literature suggests a greater risk for workplace accidents associated with the diagnosis of ADHD.[5] No such data are available yet from the commercial diving field. In this regard, the higher expression of ADHD in the current sample (relative to comparable general worker samples), as well as the suggestion of no significant increased risk for adverse safety events in naval diving, both discussed above, could be hypothesised to suggest small impact on diving performance, at least in cases of mild ADHD.

The study has a number of limitations. ADHD is always a clinical diagnosis, and the symptoms were self-reported. Although the rating scale has demonstrated positive predictive validity, caution needs to be exercised when interpreting subjective scale responses. From a methodological perspective, sampling biases and the self-reporting nature of the data may have contributed to an over-representation of diagnostic cases in this sample. Therefore, the extent to which results might be confidently extrapolated to the total commercial diving industry in South Africa, let alone internationally, is unclear. Similar studies in other countries would be valuable.

Conclusions

Based on this small survey, adult ADHD may be over-represented in commercial diving in South Africa, compared to general workplace populations. However, ADHD is not necessarily a contra-indication to commercial diving. Diving medical practitioners may thus need to carefully consider the potential impact of ADHD – whether the initial condition, or its treatment – on safety performance when called to make fitness-to-dive determinations.

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Case reports

Sphenoid sinus mucocele as an unusual differential diagnosis in diving injuries

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Barotrauma; Decompression sickness; Inner ear; Hearing loss; Vertigo; Mucocele

Abstract

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Sphenoid sinus mucocele is a rare cystic lesion. It grows gradually and causes visual disturbances, ocular motility abnormalities and headache due to cavernous sinus compression. Sudden change in sinus cavity volume by a barotrauma may compress a mucocele and precipitate symptoms that may easily be confused with decompression sickness. A diver suffering from vertigo, nausea, blurry vision and hearing loss following uneventful dives is presented in this report. He underwent hyperbaric oxygen treatment for inner ear decompression sickness but later was diagnosed as sphenoid sinus mucocele. A high index of suspicion is necessary to capture rare conditions like mucocele in the differential diagnosis for divers with symptoms suggesting vestibulocochlear origin. To our knowledge, only one sphenoid sinus mucocele case presenting as a diving injury has been previously reported.

Introduction

Diagnosis of decompression sickness (DCS) depends mainly on dive history and physical examination. Vestibular symptoms and hearing loss after a dive may suggest inner ear DCS however they are not specific to it. Similar symptoms may be encountered in other dive related injuries like inner ear barotrauma (IEB) or arterial gas embolism (AGE) as well as totally nonrelated diseases like sensorineural hearing loss, so they may be misleading. Differential diagnosis through comprehensive history taking, examination and sometimes imaging methods is essential for accurate and timely treatment.

Mucocele is a rare cystic lesion that may precipitate conditions with these symptoms.[1] We present a patient who underwent hyperbaric oxygen therapy for inner ear DCS but was later diagnosed with a sphenoid sinus mucocele.

Case report

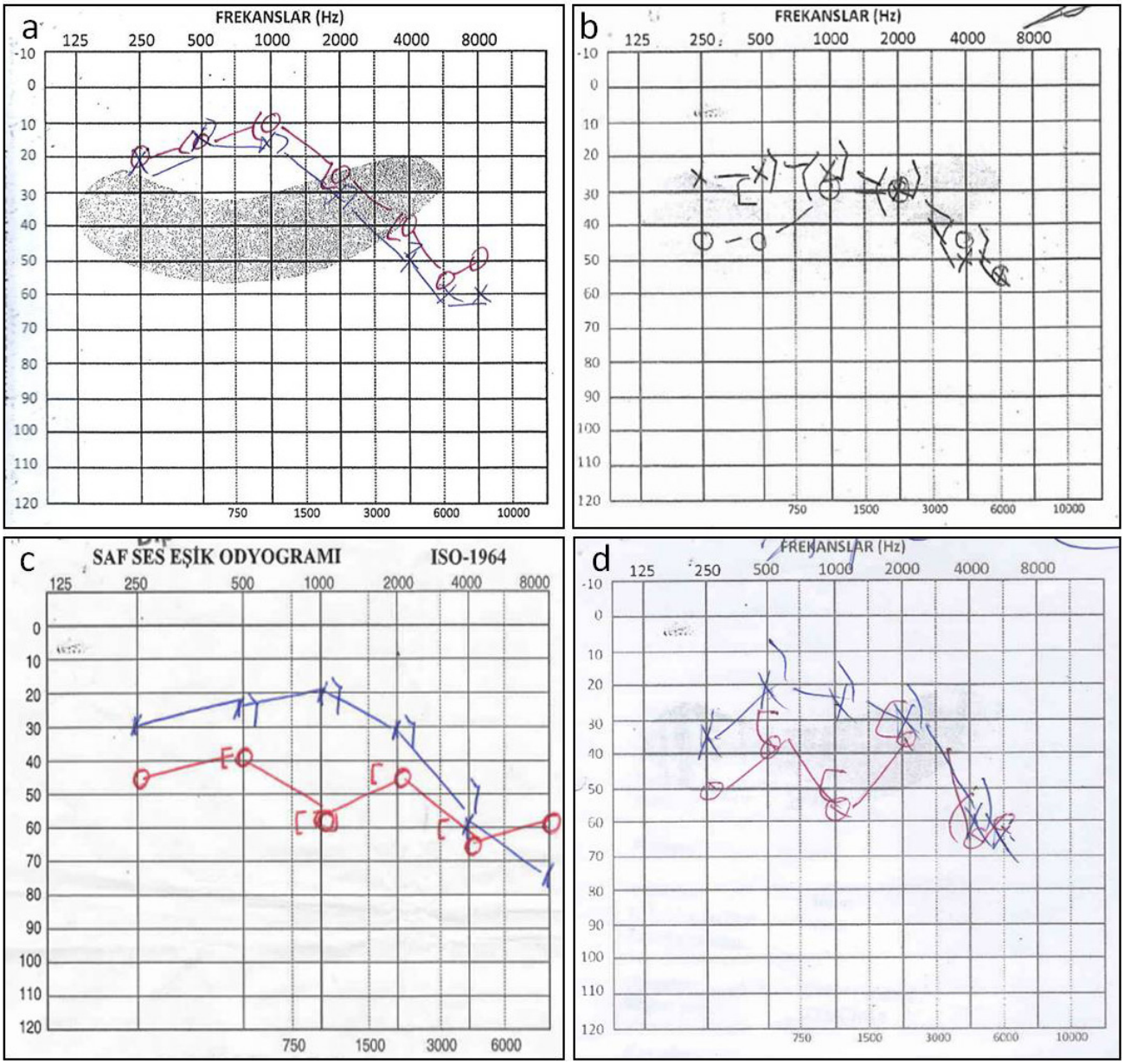
The patient was a 59 year-old male professional diver with over 2,000 dives in 20 years. He was trained to CMAS instructor level but had been working as a harvester recently. He had been assessed for fitness to dive twice previously in our department and was known to have well controlled type 2 diabetes mellitus, mild hypertension and bilateral high frequency hearing loss. (Figure 1a).

In his first incident, he presented to the emergency department of a local hospital with blurry vision, vertigo and headache followed by vomiting, hearing loss and tinnitus after a dive to 32 metres' seawater (msw) for 35 minutes with decompression stops at 12, 9, and 6 msw for 5 min, and a shallower 'safety stop'. He didn't report difficulty in equalising or physical effort during and after the dive. He was referred to a local hyperbaric oxygen treatment (HBOT) unit with a diagnosis of DCS and was treated with US Navy Treatment Table 5 (TT5) 10 hours after the incident. He reported residual visual and hearing complaints after HBOT but no further treatment was planned.

Two days later, he re-presented as his symptoms persisted. It was thought idiopathic sudden sensorineural hearing loss (ISSHL) had developed in addition to DCS so he was prescribed oral steroids and again referred for HBOT. For unclear reasons he did not present to an HBO center for a week. Upon his arrival to our department on the tenth day after the incident he had horizontal nystagmus and a positive Romberg test. The audiogram revealed a pure tone average of 50dB and 30dB for the right and left ears respectively. His tympanic membranes were intact. He was seen by the Ear, Nose and Throat (ENT) department where ISSHL was ruled out and he was administered intratympanic steroid injections for endolymphatic hydrops. Ten days later, his hearing had improved (Figure 1b) and his other symptoms had regressed. Afterwards he was lost to follow up.

Figure 1

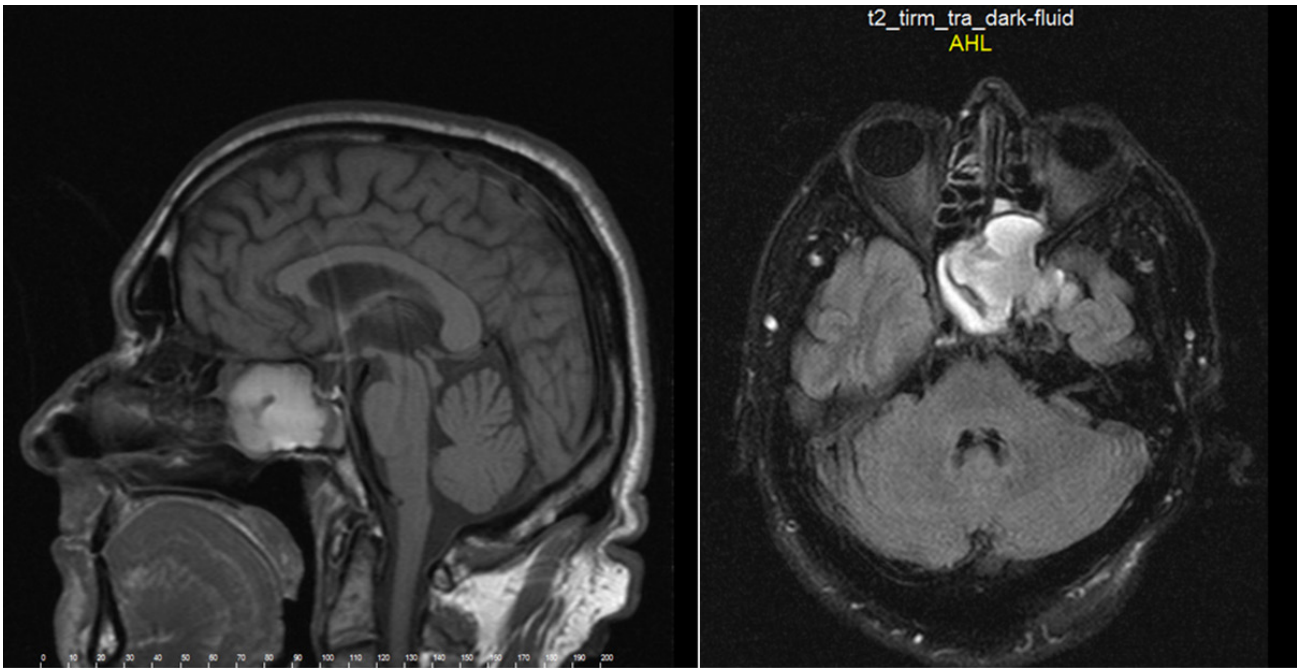
Audiograms: (**a**) during fitness to dive assessment; (**b**) after the first incident, hearing is almost back to baseline; (**c**) second incident, moderate decrease in hearing threshold (both air and bone conduction); and (**d**) when HBO was stopped, minimal change from the previous test. Red is right ear and blue is left ear. 'o' and 'x' show right and left air conduction respectively. '>' or ']' and '<' or '[' show right and left bone conduction respectively



A year later he presented to an emergency department again with blurry vision, vomiting and stomach ache and hearing loss that occurred approximately 10 minutes after surfacing from a dive. He had performed two 75-minute dives to 25 msw with a 4.5 h surface interval. Dive profiles were the same; 40 minutes of bottom time, 10-minute stops at 12, 9 and 6 msw and a shallower safety stop. He was administered 100% oxygen and IV fluids and discharged when he felt better but again symptoms did not resolve completely.

The following day he was evaluated in the ENT department where he was diagnosed with ISSHL and referred for HBOT. Again, for an unknown reason he presented to our department 10 days after the referral and exhibited vision problems, imbalance, hearing loss and dizziness. He reported that he had experienced similar symptoms four times since the first incident and only after diving. Physical examination revealed bilateral horizontal nystagmus, steady walking and dysdiadochokinesis. Otoscopic examination was normal. There was a significant deterioration in the mid-range audiometric thresholds in the right ear, compared with the audiogram recorded after the previous incident (Figure 1c). Inner ear DCS was considered the most likely diagnosis.

Figure 2
Cranial MRI showing sphenoid mucocele; sagittal (left) and axial sections



After extensive discussions about treatment choice he underwent US Navy TT6 but didn't improve. It was decided to continue HBOT with daily US Navy TT9 applications and the patient was hospitalised to investigate further for right-to-left shunting, arterial gas embolism (AGE) and possible other causes. There was no evidence of patent foramen ovale (PFO) in transoesophageal echocardiography (TOE) performed with bubble contrast and Valsalva provocation. High resolution computerised tomography (HRCT) of the chest failed to demonstrate any lesions predisposing to pulmonary barotrauma. His ophthalmological examination was unremarkable. In electronystagmography, which was offered by the neurology department, bilateral vestibular hypofunction was observed with caloric test. Finally, cranial magnetic resonance imaging (MRI) revealed a large cystic mass in the sphenoid sinus. It had eroded the temporal bone and was compressing the cavernous sinus. Paranasal CT confirmed the MRI findings but also showed that the left optic nerve was compressed (Figure 2). The lesion was identified as a sphenoid sinus mucocele (SSM) and the hearing deficit was thought to be unrelated. HBOT was stopped. At this time the patient's complaints had not changed and there was a minimal change in hearing. (Figure 1d) Endoscopic drainage was performed by the ENT department. All symptoms except hearing loss resolved after surgery.

Discussion

Mucoceles are rare cystic lesions of the sinuses that are thought to arise following obstruction of the sinus ostium.[2] Although benign, they can be very destructive due to their expansile properties.[3] Resorption and/or erosion of sinus walls are possible and further expansion may result in compression of adjacent structures. SSM, comprising only 1–2% of all mucoceles, are in close proximity to the cavernous sinus which contains the internal carotid artery, optic chiasm and the 3rd, 4th and 6th cranial nerves. An expansion in a sphenoid mucocele can therefore cause symptoms related to compression of these nerves and vessels. Sometimes inflammation of these structures may supervene.[4] Headache, visual disturbances, ocular motility abnormalities, endocrine dysfunction and nasal congestion are common symptoms.[5–8]

Generally, a mucocele grows gradually within the sinus so acute onset of symptoms is unexpected. However, barotrauma of the sinus containing the mucocele can precipitate symptoms. There may be remaining air filled spaces in the sinus until the mucocele fully fills the cavity, but drainage is blocked leaving the sinus prone to barotrauma.[9] It is probable that our patient had barotrauma. Thus, mucosal edema and bleeding or volume change in the sinus resulted in displacement and compression of the mucocele and consequently the cavernous sinus. Nystagmus and ataxia which are rarely reported in SSM were probably due to unilateral dysfunction of oculomotor nerves. Non-synchronized movements of the eyes are known to produce such symptoms.[10] Interestingly, symptoms became evident after a short lag in both incidents probably because compression developed over time. Furthermore, barotrauma explains why the patient was symptom free in his daily life but had attacks only after diving.

The patient also complained of hearing loss in his incidents however only a moderate decrease in hearing thresholds (both in air and bone conduction) was shown. In fact, a gradual decline especially in higher frequencies was already present. In addition, hearing loss was greater in the right ear during the second incident even though the mucocele was left sided. Therefore, this was considered to be an independent sensorineural hearing loss unrelated to the mucocele. Apparently, the patient did not report his baseline hearing deficit in other hospitals so was diagnosed with ISSHL in both incidents. Yet, ear MRI was not performed for this patient at the diagnosing centres although it is recommended in treatment guidelines to evaluate retrocochlear pathologies.[11] The mucocele could have been identified earlier if MRI had been performed.

Our patient presented with symptoms that suggested inner ear DCS. For many years, inner ear DCS was thought to occur primarily after deep mixed gas diving.[12] Later, however, it was seen to be possible following repetitive diving and even recreational air dives.[13,14] Recent studies have shown that right-to-left shunting which may predispose to certain forms of DCS after less provocative dives was prevalent in inner ear DCS.[15,16] Our patient had performed air dives within safe decompression limits and surface intervals, so PFO was thought to be a possible risk factor. However, TOE performed with bubble contrast and provocation manoeuvres was completely normal.

Similar symptoms may also be seen in AGE. For example, the presence of visual symptoms could be suggestive of AGE, however the patient did not report rapid ascent or breath holding during ascent. Nevertheless, pulmonary barotrauma can develop despite controlled ascent and correct exhalation especially in the presence of predisposing lung lesions.[17] In this case HRCT did not reveal any lung pathology.

IEB is another diving injury that can present with vestibulocochlear symptoms. It may occur with a forceful Valsalva or a spontaneous opening of Eustachian tube after middle ear equalisation fails.[18] IEB was unlikely in this case as the patient didn't report any difficulty in equalising during any of his dives. Additionally, in the case of an IEB, compression in the chamber could cause deterioration which was not seen. The finding of the mucocele provided a plausible explanation for all the symptoms independent of inner ear pathology, with the exception of the hearing loss.

To our knowledge, this is the second reported sinus mucocele case presenting as a diving injury. A diver presenting with acute vision loss, retrobulbar pain and headache after an uneventful dive was reported previously.[9] Contrary to our case, his symptoms were less suggestive of DCS but both divers had relatively unexpected visual symptoms in common. Sinus mucoceles may be considered if visual disturbances occurring after a dive don't associate with other symptoms or the dive profile.

Conclusion

Initial presentation of this patient suggested DCS in both incidents but SSM was found to be responsible for visual disturbances, vertigo and nausea. Hearing loss, on the other hand, was thought to be unrelated. SSM may precipitate symptoms that can easily be confused with other diving related injuries. A high index of suspicion is necessary to include it in differential diagnosis when symptoms suggesting vestibulocochlear origin are accompanied by visual disturbances.

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Bubbles in the skin microcirculation underlying *cutis marmorata* in decompression sickness: Preliminary observations

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Key words
Bubbles; Decompression illness; Decompression sickness; Skin; Venous gas embolism; Persistent foramen ovale; Pathology

Abstract
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Introduction: The cutaneous form of decompression sickness (DCS) known as *cutis marmorata* is a frequent clinical presentation. Beyond a general acceptance that bubbles formed from dissolved inert gas are the primary vector of injury, there has been debate about pathophysiology. Hypotheses include: 1) local formation of bubbles in the skin or its blood vessels; 2) arterialisation of venous bubbles across a right to left shunt (RLS) with local amplification in bubble size after reaching supersaturated skin via the arterial circulation; and 3) passage of arterialised venous bubbles to the cerebral circulation with stimulation of a sympathetically mediated vasomotor response.
Methods: Four divers exhibiting *cutis marmorata* had the underlying tissue examined with ultrasound 4–5.5 hours after appearance of the rash. All subsequently underwent transthoracic echocardiography with bubble contrast to check for a RLS.
Results: In all cases numerous small bubbles were seen moving within the skin microvasculature. No bubbles were seen in adjacent areas of normal skin. All four divers had a large RLS.
Conclusion: This is the first report of bubbles in skin affected by *cutis marmorata* after diving. The finding is most compatible with pathophysiological hypotheses one and two above. The use of ultrasound will facilitate further study of this form of DCS.

Introduction

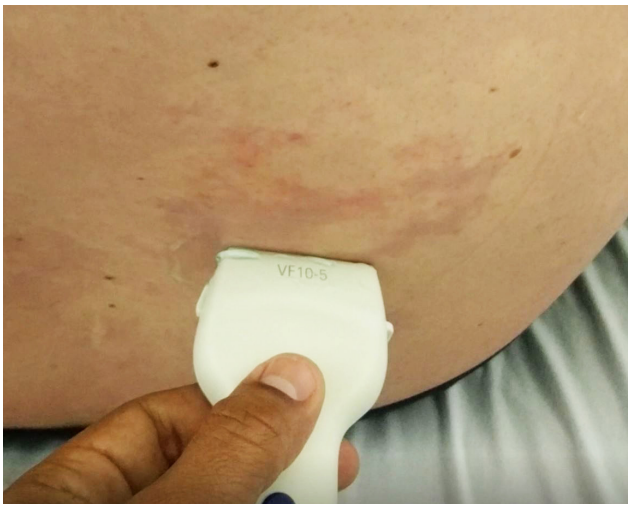
Decompression sickness (DCS) may occur in divers, aviators, astronauts or personnel decompressing from work under hyperbaric conditions. It is primarily caused by the formation of bubbles from dissolved inert gas during or after decompression.[1] These bubbles may form in tissues or in blood passing through them. The resulting venous bubbles are typically removed from circulation in the lung capillary bed and are usually asymptomatic.[1] However, they may cross to the arterial circulation via a right to left shunt (RLS) such as a persistent (patent) foramen ovale (PFO) or a pulmonary shunt, and may then enter the microcirculation of vulnerable target tissues where diffusion of supersaturated inert gas from surrounding tissue could cause them to grow.[2,3] Vascular bubbles may cause endothelial injury which can incite platelet aggregation, activation of leukocytes and fibrin deposition leading to reduced flow, greater vascular permeability and tissue oedema that exacerbates ischaemia. Cytokine release, complement activation and microparticle production are part of this inflammatory milieu and may contribute to tissue injury.[4,5] The variability in the activation of inflammatory processes could contribute to the differences between individuals in susceptibility to DCS as well as the highly variable clinical presentations which can range from a mild syndrome to a life threatening condition.[5]

CUTIS MARMORATA

One of the signs of DCS is a rash known as *cutis marmorata*. This presents with a mottled, livedoid appearance that is typically distributed 'centrally' over tissues with significant amounts of subcutaneous fat;[6] for example on the chest, back, abdomen, breasts, buttocks, upper arm and thigh areas. This may be accompanied by pruritus, pain or burning. The rash may appear in isolation or combined with other DCS manifestations, such as neurological symptoms. In the experience of the present authors, it is a common finding in patients with haemodynamic shock associated with DCS.

In the absence of co-existing serious symptoms or signs a rash is, of itself, considered a 'mild' manifestation of DCS.[7] However the 'mild' characterisation must be applied cautiously. There is a strong association between appearance

Figure 1
Cutis marmorata rash on the lower back with ultrasound probe in place



of *cutis marmorata* after diving and the presence of a large right to left shunt such as a PFO whose significance was explained above.[2] Other more serious manifestations such as cerebral, spinal and vestibulocochlear symptoms are also associated with a PFO,[8] and care must be taken to look for these problems when a diver presents with *cutis marmorata*.

PATHOPHYSIOLOGY

As with most manifestations of DCS, beyond the presumed involvement of bubbles as the primary vectors of injury, there has been uncertainty and controversy surrounding the underlying pathophysiology of *cutis marmorata*. The histopathological findings were described in a porcine model of DCS.[9] The main changes observed were an increase in skin thickness, elevation in nitric oxide, congestion, and deposits of red blood cells that clogged the dermal capillaries. Vascular dilation, haemorrhage and neutrophil infiltrates were also reported. However, that study did not elucidate the mechanisms inciting these changes. Three principle pathophysiological theories have been proposed.

Hypothesis one. Bubbles could form locally in the skin; either in the extravascular tissue, or within the subcutaneous micro-vasculature.[8] These bubbles could then produce symptoms through physical, prothrombotic and inflammatory processes alluded to above. A weakness of this theory is that bubbles have not previously been demonstrated in skin exhibiting *cutis marmorata*.

Hypothesis two. The association between a large PFO and *cutis marmorata* strongly implicates venous inert gas bubbles crossing into the arterial circulation as vectors of injury.[2] After arriving in the skin these bubbles could grow through inward diffusion of locally supersaturated inert gas; a process Wilmshurst refers to as "peripheral amplification".[8] Once again, they could then produce symptoms through physical, prothrombotic and pro-inflammatory processes. As above, a weakness of this theory is that bubbles have not previously been demonstrated in the micro-vessels underlying skin exhibiting *cutis marmorata*. Moreover, in a recent study in a porcine model of *cutis marmorata* echocardiographic monitoring of the pigs showed no arterial bubbles despite the development of florid skin lesions.[10] The latter observation could be interpreted as supportive of hypothesis one.

Hypothesis three. It has been suggested that the association of *cutis marmorata* with a large PFO may be alternatively explained by passage of arterialised venous bubbles to the cerebral circulation. These bubbles may elicit release of neuropeptides[11] or alter sympathetic outflow activity[12] which could provoke a livedoid rash peripherally through vasomotor or pro-inflammatory changes. This 'central mediation' hypothesis draws heavily on the appearance of *cutis marmorata*-like rashes in non-dived pigs subjected to cerebral arterial gas embolism,[11] but it has several crucial weaknesses.[6] In particular, the dose of cerebral arterial air employed the pig study[11] is of doubtful relevance to DCS. It resulted in the death of some pigs and would likely have produced profound neurological injury had the survivors been allowed to emerge from anaesthesia, yet divers with *cutis marmorata* frequently have no neurological manifestations. Moreover, Wilmshurst also points out that patients undergoing PFO testing with bubble contrast never develop a rash even when the test is strongly positive and large showers of small bubbles enter the arterial circulation,[6] despite the fact that cerebral symptoms are sometimes reported in this setting.[13,14]

There is no clear consensus on which of these mechanistic hypotheses is pre-eminent although the association between *cutis marmorata* and PFO is compelling. It is plausible that all could be relevant under certain circumstances. We report here an incidental clinical observation of substantial relevance to the first two hypotheses.

Clinical observations

Observations were made on four DCS cases exhibiting *cutis marmorata* and treated at Cozumel, México. All patients gave permission for relevant clinical data to be reported.

Four divers presenting with *cutis marmorata* underwent ultrasound examination directly over the involved skin using a Siemens Acuson x300 2D ultrasound machine (Siemens, Fishers, USA) coupled with a linear 4.0–11.4 MHz transducer with examination depth set at 35 mm (see Figure 1). The examination took place between 4–5.5 h after finishing diving, and between 2–4.5 h after appearance of the rash. The examination was limited to a short period (< 10 minutes) so as not to delay recompression. In all cases, moving bubbles were detected in underlying arterioles and venules as well as interconnecting vessels and

Figure 2
Two screen shots from an ultrasound video loop showing the identical location beneath a cutis marmorata rash in DCS. In A, the arrow indicates a small cylindrical bubble. In B, the arrow indicates a hypoechoic void in the micro-vessel immediately after the bubble had passed through

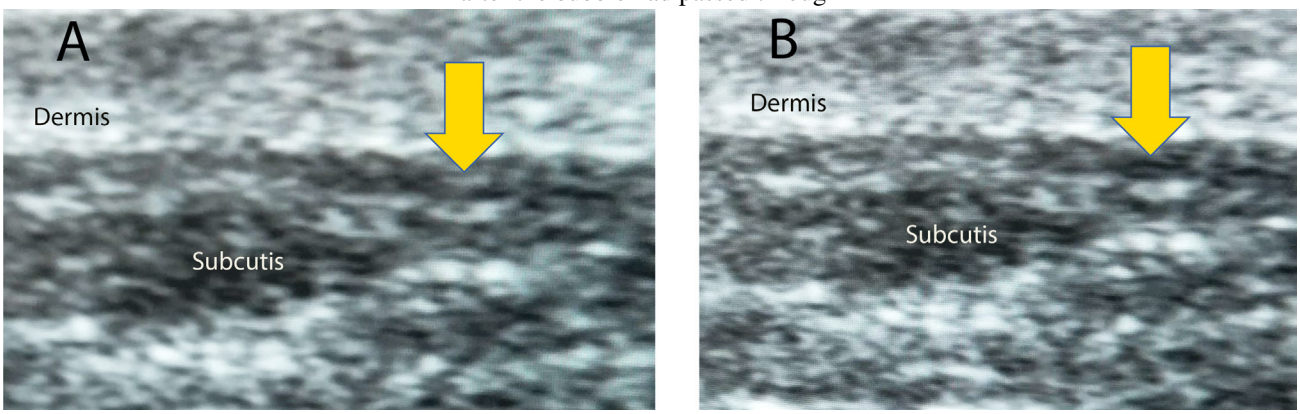


Table 1
Diving and clinical data for the four reported cases. Durations reported in the dive profile are total dive times and depths are maximum depths. All dives were compliant with the divers' dive computer recommendations. BMI = body mass index; DCS = decompression sickness; M = male; F = female; msw = metres' seawater depth; SI = surface interval

Case	Age	Sex	BMI	Dive profile	Symptoms other than *cutis marmorata*	Scan latency (hours)	Treatment
1	58	M	33	24 msw – 70 min; 50 min SI 18 msw – 78 min	Spinal DCS (decreased strength both legs, tingling, paresthesias)	4.0	1 x Table 6 6 x Table 9
2	51	M	44	27 msw – 68 min; 60 min SI 24 msw – 60 min; 90 min SI 21 msw – 56 min	Cerebral DCS (confusion, incoherence) Constitutional symptoms (fatigue)	5.0	2 x Table 6 1 x Table 5
3	42	M	40	43 msw – 43min; 60 min SI 19 msw – 60 min	Vestibular DCS	5.0	2 x Table 6 1 x Table 5
4	38	F	31	26 msw – 40 min; 60 SI 16 msw – 53 min; 60 SI 14 msw – 47 min	Vestibular DCS	5.5	2 x Table 6 1 x Table 5

capillaries. No attempt was made to quantify or measure the bubbles, but some were large enough to have formed cylindrical shapes in these micro-vessels (see Figure 2), and it can be confidently assumed that the bubbles were therefore large enough to be interacting with the vascular endothelium. In all cases there was tissue oedema apparent under the rash which can be the result of extravasation of fluids following physical or inflammatory damage to the endothelium. A short segment of video illustrating some of these observations can be found by following the link https://www.dhmjournal.com/index.php?option=com_content&view=article&id=70. Examination of normal skin adjacent to areas of *cutis marmorata* revealed no bubbles large enough to be seen using this ultrasound technology. It is germane to note that this report is not selective. No divers with *cutis marmorata* have been examined with ultrasound who did not exhibit bubbles in the subcutaneous circulation underlying the rash.

Consistent with the associations described earlier, all of these cases exhibited neurological manifestations in addition to the rash (two vestibular, one cerebral and one spinal). All were recompressed on a US Navy Treatment Table 6 as the initial intervention, and had variable numbers of follow-up treatments (maximum seven) (see Table 1). In all cases the rash resolved completely after two (two cases) or three (two cases) recompressions. It is notable that all four divers subsequently underwent transthoracic echocardiography using bubble contrast to test for a RLS; all were positive (two characterised as large, and two as small). All four divers had a body mass index (BMI) between 31 and 44.

Discussion

We believe this is the first observation of bubbles present in the subcutaneous tissues beneath a *cutis marmorata* rash in DCS. The finding must be interpreted cautiously in relation to the above mechanistic hypotheses, though it does address the criticism in relation to hypotheses one and two that local bubbles have never been demonstrated in *cutis marmorata*. The mere presence of bubbles does not prove a causative role, but it is a highly relevant observation.

We cannot be sure whether these bubbles originated through local formation or represent small venous bubbles that have crossed a RLS. The fact that all four subjects exhibited an elevated BMI and therefore had greater truncal subcutaneous fat in which nitrogen is highly soluble plausibly supports a local formation mechanism. However, the finding of a RLS in all four divers is also consistent with them having crossed a RLS from the venous circulation. These observations raise the question as to why adjacent skin unaffected by rash would not exhibit subcutaneous vascular bubbles; fat content should be similar, and any bubbles crossing a RLS should distribute widely and approximately uniformly. One possible explanation in relation to bubbles crossing a RLS holds that they might become larger and more visible in those areas of skin with relatively greater degrees of tissue gas supersaturation, but this is speculation and once again, it is not immediately obvious why this would be different in adjacent areas of skin.

This incidental finding is a preliminary observation. It is reported here in the absence of a more systematic approach to its investigation because of its potential importance to the pathophysiological hypotheses discussed above, and in the expectation that it may provide motivation for clinicians with access to the right equipment to move forward with further evaluations. Future work could include a more careful evaluation of bubbles beneath the rash versus normal surrounding tissue, an evaluation of subcutaneous bubble activity correlated against the appearance of the rash (for example, during its resolution), and examination for the presence of bubbles in a wider group of divers with *cutis marmorata* to establish whether they appear in divers without a RLS. Another important future step would be to simultaneously perform echocardiography and observation of bubble activity beneath a *cutis marmorata* rash to see if the presence of bubbles in the skin corresponds with bubbles appearing in the left heart (consistent with hypothesis two). The skin is arguably one of the most accessible of the DCS target tissues for study, and ultrasound examination would appear to be a potentially useful tool in studying pathophysiology. The presence of bubbles and the inflammatory reaction that they generate after their vascular passage might provide insight into how other vulnerable tissues such as the inner ear, spinal cord or brain behave after the insult caused by bubbles.

Conclusions

This report confirms that mobile vascular bubbles are present in the subcutaneous microcirculation in tissue underlying a *cutis marmorata* rash. A causative role cannot be confidently inferred, but the finding of bubbles strengthens the hypotheses that bubbles formed locally or distributing to the skin after crossing a RLS and undergoing peripheral amplification may be the primary agents of harm in *cutis marmorata*.

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Diaphragmatic injury a hidden issue for divers following trauma: Case report

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Key words
Diving; Trauma

Abstract

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Occult diaphragmatic injury was uncovered in a patient who returned to scuba diving after a traumatic injury. Diaphragmatic injury can be a difficult diagnosis in the setting of trauma and a significant number of injuries are missed on the initial presentation. This has potential implications to those wishing to return to diving after trauma, and diving doctors must maintain a high degree of suspicion for such injuries.

Case report

A 40 year-old female scuba diver presented to a regional emergency department (1,200 km north of the state capital and the nearest hyperbaric facility) with chest pain and vomiting following a recreational dive. The patient, an experienced diver, was returning to diving after a few years absence and was undertaking a morning shore dive. She was forced to abandon after 15 min due to left shoulder and chest pain, without any shortness of breath. At no point during the dive did she proceed deeper than 10 metres' sea water (msw) and there was no history of an uncontrolled ascent. She completed a 3 min safety stop at 5 msw with symptoms persisting on surfacing.

After exiting the water she developed nausea and vomiting, with ongoing chest pain maximal around the left shoulder, pleuritic in nature and described as 8/10 in severity. Due to the persistent nature of the pain she presented to the local emergency department that afternoon. Interestingly, on a refresher dive a few weeks prior, her first dive since 2015, she had developed a similar discomfort which had spontaneously resolved within an hour post dive.

On presentation her observations were all within normal limits, and diving first aid was commenced with high flow oxygen and intravenous fluids. On examination the pain in her left shoulder region could not be replicated on palpation, but reduced air entry in the left base was noted. Neurological examination was reported as being unremarkable. Routine blood gas and ECG were unremarkable and a chest X-ray (CXR) was performed to assess for the initial differential diagnosis of pneumothorax or pneumomediastinum. Decompression sickness (DCS) was not considered likely due to the short, shallow diving profile.

The patient's past medical history included a motor vehicle accident in 2015. This resulted in a subluxation of C1/C2, fractured pelvis and left sided rib fractures with a pneumothorax requiring an intercostal catheter. She was otherwise fit and well with a normal body mass index.

After discussion and on review of her CXR by the Fiona Stanley Hyperbaric Medicine Unit it was noted that there was a markedly distended, likely gastric bubble, in the left hemithorax (Figure 1). The treating team were advised to site a nasogastric tube (NGT), which initially drained 200 ml of air and 70 ml of fluid. The patient had persisting symptoms overnight, but was able to pass flatus. Repeat CXR the next morning showed progression of the gastric distension (Figure 2) raising concerns for an incarcerated hernia and the decision was made to transfer the patient to a tertiary centre with surgical facilities (1,200 km south) via the Royal Flying Doctor Service in a sea level cabin. A CT scan was performed on arrival which confirmed diaphragmatic rupture with hernia of the stomach and possibly the spleen. There was near complete collapse of the left lung with mild mediastinal shift to the right. (Figure 3).

An endoscopic NGT placement and decompression of the stomach was performed, due to the inability to decompress the stomach with prior blind placement of the NGT. The patient then proceeded to a laparoscopic repair of the hernia, where it was noted intraoperatively that she had a 10 x 6

Figure 1
Initial CXR showing distended probable gastric bubble, in the left hemithorax

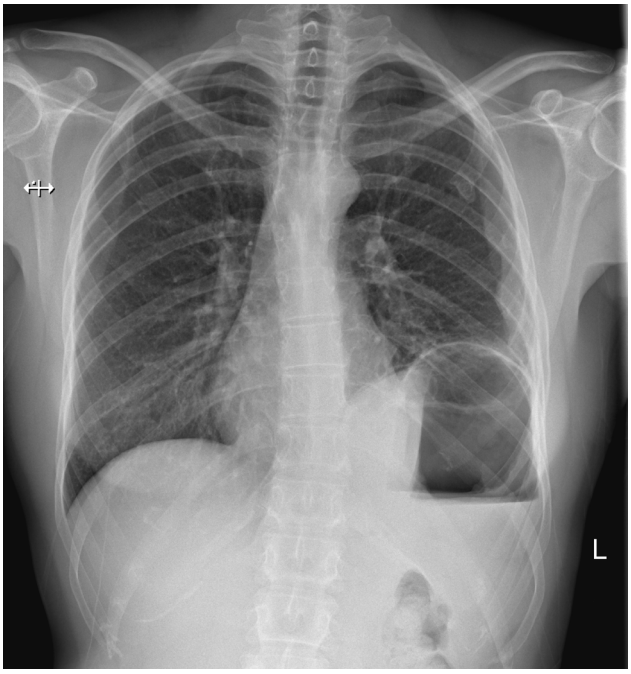


Figure 2
Follow-up CXR showing progression of the gastric distension

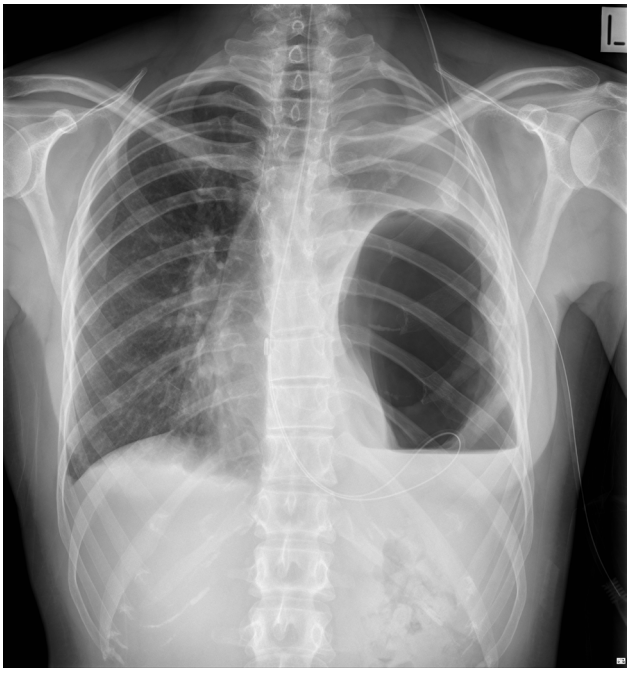
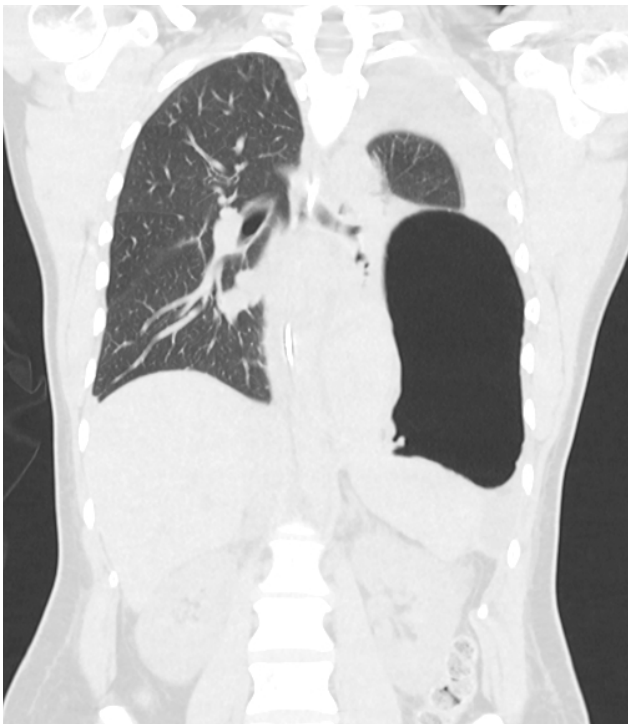


Figure 3
Coronal thoraco-abdominal CT scan showing diaphragmatic rupture, hernia of the stomach and possibly the spleen, with near complete collapse of the left lung with mild mediastinal shift to the right



cm hernia in the left diaphragm that contained stomach and the left lobe of the liver, with no sac present. The hernia was primarily closed with sutures and then reinforced with biomesh.

Discussion

This is an unusual case of an undiagnosed pathology uncovered by diving. There is one previous case of delayed diaphragmatic hernia being uncovered by diving, where a 40 year-old male had presented with thoracic and abdominal pain with associated dyspnoea following a dive to 50 msw. He was found to have transverse colon, small bowel and omentum in the diaphragmatic hernia. He had a background of chest trauma secondary to an MVA six years prior to his episode.[1]

Diaphragmatic hernia in admitted patients following blunt thoracic or abdominal trauma has been reported in up to 5% of cases.[2] Delayed recognition occurs in up to 15% of cases, due often to minimal initial symptoms and other complicating injuries.[3] In a targeted history following diagnosis, our patient had not described any reflux or digestive symptoms following her accident; just non-descript feelings of occasional shortness of breath and one episode of left shoulder spasm a year prior to her dives. Though diagnostic imaging is better with the use of helical CT, sensitivities of the diagnosis of diaphragmatic injury have still been reported between 50–87%.[4,5] Diaphragmatic hernias have also been misdiagnosed on CXR leading to the inappropriate use of chest drains.[6] A small case series reported an average age of 41 years at detection of the hernia with a mean time of 6.5 years following the traumatic event.[7] This is in keeping with the demographics of the two cases reported in divers.

The issue of missed diaphragmatic injury following trauma is particularly relevant to people returning to diving. The

ingestion of gas at depth with expansion on ascent (according to Boyle's Law) creates the potential to promote herniation through an acquired deficit. In divers presenting with chest or shoulder pain after diving the typical differential diagnoses would include pneumothorax, pneumomediastinum or musculoskeletal DCS. However as this case demonstrates, with a past history of trauma, special attention must be paid to the CXR to rule out diaphragmatic hernia as a cause of symptoms. It is also important to note the difficulty in decompressing the herniated stomach with blind NGT placements, which should warrant urgent transfer to a centre with endoscopic and surgical facilities. In terms of prevention, doctors performing diving medicals should also maintain a high degree of suspicion for the potential of undiagnosed traumatic diaphragmatic hernias in relevant patients to minimise the occurrence of such cases in the future.

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Clinical problem solving: Mental confusion and hypoxaemia after scuba diving

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Key words
Bubbles; Decompression sickness; Differential diagnosis; Hyperbaric oxygen therapy; Respiratory symptoms; Stroke

Abstract
(Blatteau J-E, Morin J, Roffi R, Druelle A, Sbardella F, Castagna O. Clinical problem solving: Mental confusion and hypoxaemia after scuba diving. Diving and Hyperbaric Medicine. 2020 June 30;50(2):181–184. doi: 10.28920/dhm50.2.181-184. PMID: 32557423.)
Introduction: We report a case of a diving accident associating both cerebral symptoms and signs of respiratory impairment after two dives. The objective is to describe the process for obtaining the diagnosis.
Case report: A 52-year-old man experienced mental confusion associated with hypoxaemia after surfacing. All decompression procedures were fully respected. The diver had a spatio-temporal disorientation accompanied by a marked tendency to fall asleep spontaneously. He had no dyspnoea and no cough, but crepitations at both lung bases were found with oxygen saturation at 80%.
Conclusions: In this clinical case, cerebral magnetic resonance imaging and chest computed tomography scan helped to exclude other pathology that would have necessitated urgent transfer rather than urgent hyperbaric treatment. The imaging is particularly useful in case of cerebral and respiratory symptoms following scuba diving.

Introduction

When clinical symptoms are observed after scuba diving, it is important to identify the type of diving accident but also to look for a differential diagnosis. The diagnosis is primarily based on the patient history and diving circumstances. But in certain situations, it may be necessary to carry out first-line complementary tests. Herein, we report a case of a diving accident associating both cerebral symptoms and signs of respiratory impairment after scuba diving. The objective is to describe the process for obtaining the diagnosis.

Case report

A 52 year-old man who experienced mental confusion and hypoxaemia following a dive, was hospitalised at the hyperbaric centre of the Sainte-Anne military hospital in Toulon (France) for seven days in September 2018.

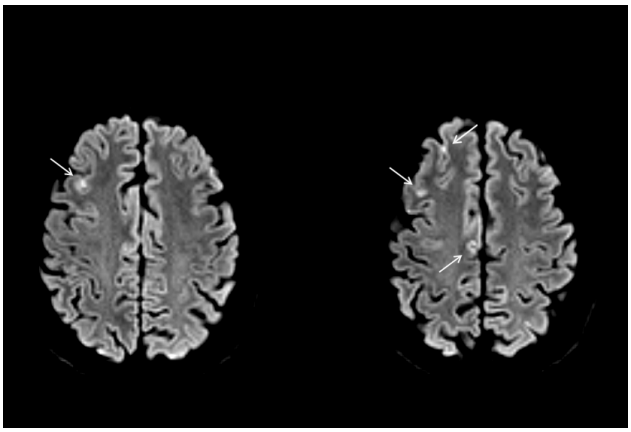
The patient was an experienced scuba diver of German nationality with several hundred dives without incident. He was in the south of France on a diving holiday. He had no significant medical past history, other than past surgical operations for inguinal hernia and nasal septoplasty. There were no known allergies and no medical treatments.

He began his vacation with a first scuba air dive at a maximum depth of 31 metres' sea water (msw) for 35 min total duration with a 3 min safety stop at 5 msw, surfacing at 10:35 am. In the afternoon, a second dive was carried out (surface interval = 4 h) using a nitrox mixture (28% oxygen, 72% nitrogen) to 26 msw for 42 min with a 3 min safety stop at 5 msw. He came out of the water at 3:30 pm without any physical problems reported. All decompression procedures were fully respected. No environmental risk factors were found.

At 4:00 pm he collapsed without loss of consciousness but exhibiting severe fatigue with nausea and pallor. He was treated immediately with oxygen (O_2) 15 L·min^{-1} and transferred by helicopter to the hyperbaric center of Toulon, France. At this point, the diver was confused with a Glasgow coma scale score of 13 but did not have any sensory-motor deficits. His temperature was 37.4°C, pulse 100 beats·min^{-1}, blood pressure 118/81 mmHg, respiratory rate 18 breaths·min^{-1} and O_2 saturation 80%. Blood glucose and the electrocardiogram were normal.

On admission to the hyperbaric centre at 5.30 pm, clinical examination revealed a spatio-temporal disorientation accompanied by a marked tendency to fall asleep

Figure 1
Cerebral MRI scan showing hyperintensities in the right frontal lobe (diffusion sequences)



spontaneously. The patient was stimulated constantly to keep him awake. The neurological examination did not show a sensory-motor deficiency, with no tendon reflex anomalies and a negative Babinski test; cerebellar and vestibular syndrome symptoms were also absent.

The diver remained haemodynamically stable but the peripheral O_2 saturation on air was 80%. He had no dyspnoea and no cough, but the pulmonary examination revealed crepitations at both lung bases. Arterial blood gases taken during air breathing showed hypoxaemia, with the $PaO_2 = 55$ mmHg (7.3 kPa). Chest and cardiac ultrasonography was immediately carried out and did not find any ultrasound lung comets or pneumothorax; however, circulating venous bubbles were detected in the right heart. The blood samples showed a leukocytosis with polynuclear neutrophils at 21,400 per µL (normal < 7700), natriuretic peptides (NT-ProBNP) at 233 ng·L^{-1} (normal < 84), positive D dimers at 1.55 mg·L^{-1} (normal < 0.5), lactate dehydrogenase (LDH) at 413 IU·L^{-1} (normal < 225), while haematocrit and albumin were normal. He had no cholestasis, no hepatic cytolysis and no inflammatory syndrome.

In view of the presenting neurological signs, possible diagnoses were stroke or cerebral decompression sickness (DCS). It was therefore decided that a cerebral MRI should be done urgently, before any recompression, to eliminate the diagnosis of ischaemic or haemorrhagic stroke that would require specific care. In addition, a computed tomography (CT) scan of the chest was performed to eliminate the diagnosis of pulmonary embolism but also to look for signs of immersion pulmonary oedema (IPO) or pulmonary barotrauma.

The cerebral MRI showed the presence of hyperintensities in the right frontal lobe, with the diffusion sequence indicating several cortical foci of ischaemia suggestive of an embolic cause; probably bubbles given the context of diving (Figure 1). No other abnormalities were observed at the cerebral level. A sphenoid-ethmoid sinusopathy with bilateral mastoiditis was also observed.

The CT chest scan did not show any changes to suggest IPO. There was no evidence of a pulmonary embolism. In addition, no pneumomediastinum and pneumothorax were found and so the diagnosis of pulmonary barotrauma was discounted. The presence of bilateral small areas of atelectasis at the pulmonary bases was noted. On the upper abdominal sections, the presence of hepatic portal venous gas was observed (Figure 2), which prompted extension of the investigation to the abdominal-pelvic region. This examination also showed the presence of venous gas in the right femoral vein (Figure 3).

Following the imaging investigations, the patient was recompressed at 7.10 pm for hyperbaric oxygen treatment (HBOT) using the treatment tables and adjunctive medical treatments (including intravenous rehydration with 2 L of normal saline over 5 h, methylprednisolone, acetylsalicylic acid, and lignocaine) currently applicable in our centre. He received an initial 5 h oxygen table (equivalent to a US Navy Treatment Table Six), two further short heliox treatments at 2.8 atmospheres absolute (atm abs) over the next 24 hours, followed by daily HBOT sessions at 2.5 atm abs for five days. Improvement across the treatment period was observed, with the neurological signs of confusion disappearing at the end of the first treatment, while the hypoxaemia gradually regressed over 24 hours.

A secondary work-up was then carried out to investigate the presence of a persistent (patent) foramen ovale (PFO), using transcranial Doppler and trans-oesophageal echocardiography. These tests revealed the presence of a massive spontaneously shunting PFO with an aneurysmal atrial septum. In the following days, a cardiac ultrasound and pulmonary functional testing were normal. A cranio-facial CT scan showed the presence of a left maxillary polyp, with a left ethmoid sinus opacity.

Discussion

Diving accidents bring together a set of varying clinical entities and separate pathophysiological mechanisms.[1,2] The most frequent diving accident see at our centre is DCS, which is thought to be caused by formation of bubbles in the body during decompression.[1]

The central nervous system and especially the spinal cord is frequently affected in DCS.[3] The brain can also be affected as a result of emboli passing from the right to left side of the heart via a PFO, which is found 80% of cerebral DCS cases.[4] The differential diagnosis of brain damage is primarily represented by ischaemic or haemorrhagic stroke that may occur in a diving context.[5]

Pulmonary lesions caused by SCUBA diving are IPO, pulmonary barotrauma, drowning or pulmonary DCS, of which the last is less common. With regard to the current

Figure 2
Abdominal CT scan (coronal reconstruction) showing portal venous gas

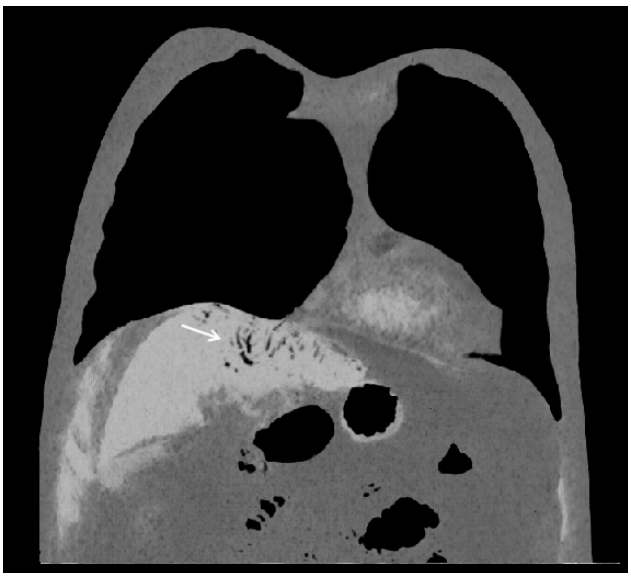


Figure 3
Pelvic CT scan (coronal reconstruction) showing gas in the right femoral vein

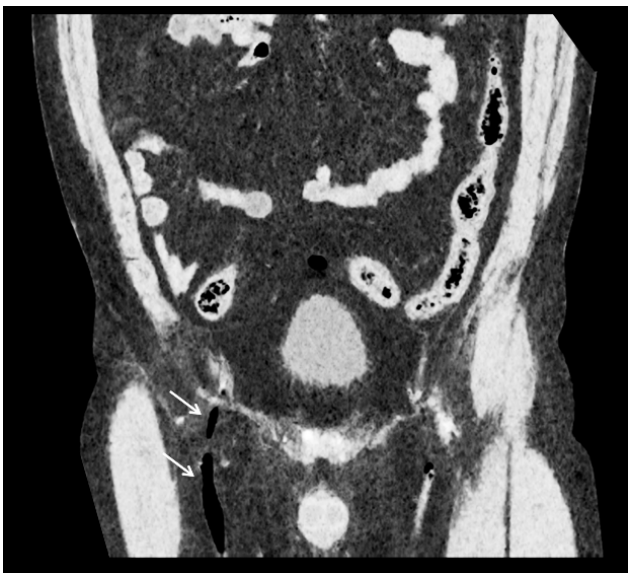
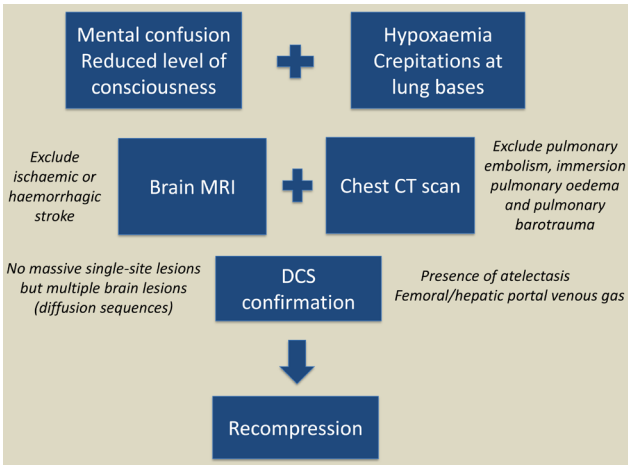


Figure 4
Decision-making process employed in managing this case



epidemiology of diving accidents, the primary aim of a clinical examination would be to investigate if IPO is present, whose origin is multifactorial.[6–8]

A pulmonary barotrauma, which is related to variations in pressure and alveolar volume, must be investigated in the case of rapid ascent and incomplete exhalation, while in the underwater environment inhalation of sea water is always possible. More rarely, a cardio-respiratory decompression sickness can also occur, when the pulmonary circulation is congested by the venous return of high numbers of circulating bubbles.[1,2]

In diving accidents with clinical signs of pulmonary damage or a high suspicion of pulmonary pathology (based on the diving history), early chest CT scanning may help clinicians exclude IPO and also to ensure the absence of barotrauma lesions such as a pneumothorax or a pneumomediastinum which could have contraindicated hyperbaric treatment.

In case of isolated respiratory symptoms, the treatment is based on normobaric oxygenation, except for the case of a cardio-respiratory decompression sickness, which requires hyperbaric recompression.[2,9]

In the presence of cerebral neurological symptoms after a dive, the problem is to determine whether the patient should be sent to a hyperbaric unit or to a stroke centre, as they are not necessarily available in the same place. Unpublished epidemiological data from our hyperbaric centre show that the differential diagnosis of stroke is more common than often thought.[5] In view of the specific care required for stroke patients, doctors practicing in hyperbaric centres should eliminate the diagnosis of stroke and perform a cerebral MRI before recompression if there is doubt.[9]

Finally, this diving accident illustrates the remarkable number of bubbles that may evolve during decompression; in this case responsible for a DCS with both cerebral and pulmonary impairment. The formation of decompression bubbles was authenticated by echocardiography and the thoraco-abdominal CT scan which showed femoral and hepatic portal venous gas. The cerebral involvement was revealed by the presence of several cortical hyperintensities observed in the MRI diffusion sequence. These diffusion sequences suggested recent ischaemic injury. Given this clinical picture, the history of diving, and considering the absence of unifocal haemorrhage or ischaemia on the MRI scan, the diagnosis of stroke was discounted.

The mechanism of cerebral embolic injury was suggested by the presence of a large spontaneously shunting PFO. The mechanism of pulmonary involvement was suggested by the elimination of other diagnoses such as IPO, pulmonary barotrauma and pulmonary embolism. The presence of atelectasis in the pulmonary bases was probably related to bubble-induced lung damage.[10] The various organ injuries found were probably related to the physical and inflammatory effects of this significant formation of bubbles.

In this clinical case the use of radiological examinations was indispensable to confirm whether hyperbaric treatment or another care plan was necessary. We were able to confirm the diagnosis of DCS with cerebral and respiratory impairment (Figure 4). The radiological examinations were carried out urgently, which did not delay recompression.

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Permission of the patient to report his experience was gratefully appreciated. Currently, he has recovered an excellent physical condition and has his PFO closed to resume diving activities.

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Sudden death after oxygen toxicity seizure during hyperbaric oxygen treatment: Case report

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Key words
Case reports; Deaths; Hyperbaric oxygen treatment; Obesity; Risk management; Side effects

Abstract

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Acute cerebral oxygen toxicity (ACOT) is a known side effect of hyperbaric oxygen treatment (HBOT), which can cause generalised seizures. Fortunately, it has a low incidence and is rarely harmful. Nevertheless, we present a case of a 37 year-old patient with morbid obesity who died unexpectedly after an oxygen toxicity seizure in the hyperbaric chamber. Considering possible causes, physiologic changes in obesity and obesity hypoventilation syndrome may increase the risk of ACOT. Obesity, especially in extreme cases, may hinder emergency procedures, both in- and outside of a hyperbaric chamber. Physicians in the hyperbaric field should be aware of the possibility of a fatal outcome after ACOT through the described mechanisms and take appropriate preventative measures. Basic airway management skills are strongly advised for involved physicians, especially when specialised personnel and equipment are not immediately available.

Introduction

Acute cerebral oxygen toxicity (ACOT) is a rare side effect of hyperbaric oxygen treatment (HBOT), during which high tissue levels of oxygen in the central nervous system (CNS) exists. Among other symptoms, generalized seizures are observed, which resolve after reducing the inspired fraction of oxygen (FiO_2). The incidence is reported as 1.2 per 10,000 HBOT sessions where air breaks are incorporated in the treatment.[1] No long term effects have been described.[2] Nevertheless, we present a case of a 37 year-old male, who died unexpectedly after an oxygen toxicity seizure.

Case report

The patient was referred for HBOT to precondition a non-healing venous leg ulcer before skin grafting. He was obese and had hypertension and obstructive sleep apnoea (OSA) syndrome. Although the medical referral mentioned use of a beta blocker for his hypertension, the patient denied this and there was no record of a prescription. Physical examination showed extreme obesity with a height of 185 centimetres and a weight of 180 kilograms (body mass index [BMI] 52.6 $kg{\cdot}m^2$), and hypertension (blood pressure 150/110 mmHg). The patient was advised to consult his general practitioner on short notice regarding the hypertension, but this was not considered to be a reason to postpone therapy since hypertension is only considered to be a mild and relative contra-indication for HBOT.

Treatment took place in a HYOT/2200/20/2/RD multiplace chamber (IHC Hytec, Raamsdonksveer, the Netherlands). The chamber was pressurised to 2.4 atmospheres absolute (atm abs; 243 kPa); and then patients breathed 100% oxygen through non-rebreathing masks in four blocks of 20 minutes, with 5-minute air breaks in between.

The first HBOT session the patient received was uneventful. Halfway through the second treatment session the patient exhibited prodromal symptoms of ACOT, immediately followed by a generalised seizure, lasting approximately 1.5 minutes. The attendant, present in the chamber, quickly removed the mask and requested the assistance of a physician. Postictally, the patient slumped down from his chair in a sitting position and had hyperpnoea. Eventually, the patient was positioned supine; the hyperpnoea persisted. Three minutes after the onset of the seizure, a physician was present inside the chamber, who also found tachycardia. Since our centre is not located in a hospital, emergency services were contacted. Due to the hyperpnoea and an absence of snoring, an unobstructed airway was assumed, and decompression was started at the usual rate of 14.9 $kPa{\cdot}min^{-1}$ (1.5 meters' seawater per minute equivalent) to minimise the risk of pulmonary barotrauma. During decompression, the patient

Table 1
Stages of obesity hypoventilation syndrome (OHS) (adapted from Randerath et al.[8])

Stage	Diagnosis	Hypercapnia status
0	At risk	None
1	Obesity-associated sleep hypoventilation	Intermittent during sleep, full recovery during sleep. Serum bicarbonate < 27 mmol·L^{-1} awake.
2	Obesity-associated sleep hypoventilation	Intermittent during sleep. Serum bicarbonate ≥ 27 mmol·L^{-1} awake.
3	Obesity hypoventilation	Sustained hypercapnia (PCO_2 > 45 mmHg or 6.4 kPa) awake
4	Obesity hypoventilation	Sustained hypercapnia awake. Cardiometabolic comorbidities.

remained hyperpnoeic and had a nosebleed, possibly from a barotrauma of the ears. In the final minute of decompression, the patient started frothing bloody sputum at the mouth and then stopped breathing. No signs of cyanosis were noted. Cardiopulmonary resuscitation (CPR) was started immediately by the physician. After decompression was completed, emergency services continued CPR inside the hyperbaric chamber. Intubation was difficult but eventually succeeded. After 30 minutes of asystole, CPR was stopped and the patient was declared dead.

Discussion

To our knowledge, this is the first published case of a fatal oxygen convulsion during regular HBOT. Although no absolute contraindications for HBOT were present in this case, several factors that may have caused or contributed to this unfortunate outcome merit discussion.

The patient's medical history contained several conditions that are considered contraindications in diving, but not in HBOT. Since HBOT takes place in a controlled environment, these conditions are far less prohibitive and not considered a contraindication. Hypertension raises the risk of both acute (cerebrovascular incidents) and chronic morbidity (end-organ failure).[3] Although HBOT causes a rise in blood pressure, the effect is small.[4] Obesity is contraindicated in diving because it causes increased nitrogen accumulation in adipose tissue, which increases the risk of decompression sickness (DCS).[5] Since patients breathe 100% oxygen during HBOT for most of a session, no nitrogen build-up takes place and there is no increased risk of DCS.

Sleep apnoea is associated with several cardiovascular comorbidities, such as hypertension, arrhythmias and pulmonary hypertension[6] but is not considered an absolute contraindication in either diving or HBOT. In this case, it is plausible that the diagnosed OSA was accompanied by an obesity hypoventilation syndrome (OHS). OHS is a sleep-disordered respiratory syndrome, defined as sustained hypercapnia in obese persons without other causes for hypoventilation present.[7] It is divided in five categories[8] (see Table 1). The estimated prevalence is 10–38% in patients with OSA,[9] and at least 14% of OSA patients present with daytime hypercapnia[10] (i.e., stage 3 or 4 OHS). The chronic respiratory changes may lead to pulmonary hypertension, cor pulmonale and peripheral oedema.[7] Like OSA, so far OHS is not been described as a contraindication for diving or HBOT. However, we hypothesise that the patient was more prone to ACOT due to OHS. While chronic hypercapnia does not influence vasodilation,[11] a small case-control study by Hollier et al.[12] showed, that even moderate amounts of supplemental oxygen induces hypoventilation and an acute increase of hypercapnia and acidaemia in OHS patients. This can cause cerebral vasodilation, counteracting the neuroprotective vasoconstriction of hyperoxaemia,[13] thereby increasing the risk of ACOT during HBOT.

After the seizure, the patient developed hyperpnoea, which is frequently seen postictally.[14] Hyperpnoea was interpreted as a sign of a patent airway. However, it is plausible that the airway was at least partly obstructed by the diminished hypopharyngeal diameter, known to occur in OSA.[15] This relative obstruction combined with hyperpnoea may have resulted in an increased negative intrathoracic pressure, which can cause pulmonary oedema, whose presence was suspected when frothy bloody sputum was observed. This condition is well-known in the field of anaesthesiology.[16]

The clinical features are reminiscent of the syndrome known as 'sudden unexpected death in epilepsy' (SUDEP), in which epilepsy patients develop similar cardiorespiratory symptoms after a generalized seizure, which can result in sudden death.[17] In autopsies performed in patients deceased after a seizure, non-cardiac pulmonary oedema is often found and regarded as a significant contributor to mortality and may be provoked by negative lower airway pressure during inspiration.[18] Pulmonary oedema can cause acute loss of the lung reservoir of oxygen. While oxygen reserves should have been adequate in other reservoirs

due to hyperoxygenation, the increased inspiration efforts causing increased respiration workload may additionally have reduced time to desaturation.[19] Moreover, while compression to 2.4 ATA may have caused an adequate tissue oxygen saturation, decompression to 1 ATA diminishes tissue oxygen saturation, similar to ascent hypoxia seen in breath holding diving.[20]

Because the airway was assumed to be unobstructed at the time of the incident, no airway management devices were used during the decompression phase. Due to the weight of the patient and the confined space of the hyperbaric chamber, attempts to reposition the patient in the recovery position failed, both during and after decompression. Intubation attempts by emergency services were hindered by these factors as well. Since airway patency is compromised in the postictal phase[18] and in overweight patients,[15] desaturation during decompression could have been prevented or ameliorated by using an oropharyngeal airway device. This is especially relevant in hyperbaric centres that do not have immediate access to specialised personnel and equipment, as is available is hospitals.

By law, no autopsy was required as the medical forensic examiner determined the cause of death to be due to a recognised side effect of the therapy. Furthermore, the family did not allow autopsy. As a result, no definitive cause of death could be determined. We can therefore only speculate on possible causes. The factors mentioned here not only increase the risk of oxygen toxicity but also mortality after the seizure has resolved. This has prompted revision of our medical screening protocol. In individuals with a BMI ≥ 35 and/or known OSA/OHS, we now perform arterial blood gas analysis. Hypercapnia (P_aCO_2 > 45 mmHg or > 6.4 kPa) at rest (OHS stage 3–4; see Table 1) is now considered a contraindication for HBOT. During the six months after this case, we screened fourteen patients based on this new criterium and excluded one. Patients weighing > 120 kilograms are counselled on the possibility that emergency procedures may be complicated due to their weight.

In conclusion, a fatal incident following an oxygen seizure during HBOT in a morbidly obese patient is described. Considering the increasing global obesity epidemic and the increasing use of HBOT, it is important to review established safety protocols for these patients. Physicians should be aware of the physiological changes in obesity that may increase the risk of ACOT. The presence of hypercapnia with OSA or OHS is an important risk factor that can easily be detected with arterial blood gas analysis. Furthermore, obesity, especially in extreme cases, may complicate emergency procedures, both in- and outside of the hyperbaric chamber and should be re-evaluated for these patients. Basic airway management skills are strongly advised for physicians working in a hyperbaric centre, especially when specialised personnel and equipment are not immediately available.

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Obituary

Richard D Vann, PhD: 1941–2020

Dick Vann grew up in Englewood, New Jersey, attended Columbia University and earned bachelor's degrees in both liberal arts and mechanical engineering. After graduation in 1965 he worked for a year at Ocean Systems as a diving engineer. He hoped to be trained as a commercial diver, but instead was sent to Union Carbide's diving physiology lab to work in a research program that was developing decompression schedules for commercial divers. He was both part of the topside crew and a 'guinea pig' for testing 40-minute dives to 650 feet. The goal was to develop decompression schedules that were faster than Ocean Systems' competition, with extensive use of oxygen to a level of pulmonary toxicity that today would be unacceptable. In the process Dick developed decompression sickness (DCS) three times and reported that after each dive his lungs took days to recover from oxygen toxicity before he could play basketball again.

In 1967 Dick joined the Navy, and after passing the grueling Underwater Demolition Team entrance test he became a platoon commander and then Diving Officer in UDT-12, serving a tour in Vietnam. He left the Navy in 1971 but stayed in the Naval Reserves for the next 26 years, serving as Commanding Officer for five Research and Special Warfare units, retiring in 1997 as Captain.

In 1970 Dick married his long-time girlfriend Sheppy von Roth, just before he began working as a Research Assistant at the Duke Center for Hyperbaric Medicine and Environmental Physiology in 1971. The same year he entered Duke graduate school in biomedical engineering. In 1976 he earned his PhD, and the same year was appointed as Assistant Research Professor of Anesthesiology, the beginning of a long career investigating cause and prevention of DCS. He focused on the mathematics of predicting the formation of inert gas bubbles and pursued numerous studies in both divers in the water and astronauts performing space suit EVAs. One particular study for which he will be most remembered, undoubtedly the largest trial in diving medicine, was a prospective study of 833 diving exposures in the Duke Hyperbaric Center followed by simulated flights to 8,000 feet altitude. The aim of this study was to develop guidelines for safe intervals between recreational divers' last dive and a commercial flight; its results were implemented as guidelines that are in use today. He also initiated Project Dive Exploration, in which, over a 13-year period, depth-time profiles of 10,358 recreational divers and 122,129 dives with clinical outcomes were monitored. The analysis was published earlier this year, providing support for the notion that for prediction of decompression sickness risk the environment (e.g., Caribbean vs. Orkney Islands) may be as important as the depth-time exposure. He also worked on flying with DCS, flying after DCS therapy, first aid oxygen for DCS and the influence of elevated inspired oxygen on carbon dioxide narcosis.

Dick worked on the importance of bubbles in other settings. He demonstrated that thrombogenicity of polytetrafluoroethylene vascular grafts can be reduced by prior denucleation by exposure to vacuum followed by saline immersion then compression to 500 psig. One of Dick's least-known studies although widely discussed in coffee rooms at the time investigated bubble formation in breast implants after hyperbaric exposure.

Dick served as Chairman of the Operations and Safety Committee and Director of Applied Research of the Duke Center for Hyperbaric Medicine and Environmental Physiology. He instituted and directed a semester course in the physiology and medicine of extreme environments at Duke Medical School. He also worked at the Divers Alert Network (DAN), establishing a summer Research Internship that trained over 60 college and graduate students in the basics of diving epidemiology. He organised international workshops on physiological basis of decompression, repetitive diving, flying after diving, management of decompression illness in remote diving locations, technical diving, diving fatalities, and rebreather diving.

Dick received numerous awards, including the Oceaneering International Award for outstanding scientific contributions to the advancement of commercial diving activities, US Navy Commendation Medal, Aerospace Medical Association Life Sciences and Biomedical Engineering Research and Development Innovation Award, UHMS Craig Hoffman Memorial award for contributions to diving safety, Diver of the Year for Science (Beneath the Sea) and DAN Asia-Pacific Contribution to Dive Safety Award.

He retired from Duke as Assistant Professor Emeritus in 2010, moving from North Carolina to Tennessee to be near his daughter and grandchildren. Despite fighting a chronic progressive illness without complaint, Dick continued to work remotely and remained active as a scientist. During a phone conversation I had with him only a few weeks before his death, he explained to me exuberantly about his idea to collect symptoms and outcomes from recreational divers using a mobile telephone app.

Dick is survived by Sheppy, his wife of 50 years, children Gene Vann, Mike Vann and Katie Vann Peck, and grandchildren Kiran and Kai Vann, Henry and Hattie Peck. He will be greatly missed by his family, friends and colleagues.

Richard Moon
Duke University Medical Center

Notices and news

SPUMS society information and news is to be found mainly on the society website: https://spums.org.au/

SPUMS President's message

David Smart

Diver health risk assessment in the setting of the Coronavirus disease (COVID-19) pandemic

As SPUMS President and a doctor who is active in the field of diving medicine, I have received multiple requests from occupational divers and other doctors for advice regarding diving health risk and medical fitness assessment in the setting of COVID-19. The rapidly evolving situation makes it very hard to produce any guidance that will remain current. Notwithstanding this, it is important that we all share and document information and support each other in challenging times. Hence my final President's report for SPUMS will focus on COVID-19. What follows are my own opinions and not those of SPUMS. I concede that the information contained in this report may well be out of date when June DHM journal is released.

Coronavirus has produced a global pandemic unprecedented in our modern era. Its manifestation predominantly as a highly infectious pneumonic disease, but also with cardiac and thromboembolic complications, has serious implications for diver health risk. As the disease spreads throughout the world, devastating clinical effects have rapidly become apparent, overwhelming even very advanced healthcare systems.

We offer our sincere support and empathy to colleagues internationally who are dealing with major outbreaks in their countries. Fortunately, Australia and New Zealand have initially been spared major population outbreaks of COVID-19 disease due to geographic isolation, prompt closing of borders and effective public health measures. Until the disease can be effectively treated, or a vaccine is available, there will always be further risk of outbreaks, because our antipodean populations remain naïve to the virus. In addition, the long-term immune status of individuals who have suffered a severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)[1] infection is not yet known.

Generating evidence-based guidelines for diver health risk assessment in the setting of COVID-19 is difficult. With only a five-month history of human spread, the long-term sequelae of COVID-19 infected individuals are still largely unknown. In this acute phase of the world pandemic, it is difficult to find studies with follow-up beyond one month. It appears that structural lung damage is possible even in individuals who have recovered from clinical illness.[2] Severity of lung damage appears to correlate with severity of disease, however even mild illness can produce significant lung pathology.[3,4] Persistent lung injury may reduce aerobic capacity and functional capability in divers. It also creates risk of pulmonary barotrauma. Cardiac effects and sequelae from the hypercoagulable state created by COVID-19 could potentially increase risk of pulmonary decompression illness, right to left shunt-related decompression illness, immersion pulmonary oedema, or even precipitate medical complications from diving.

In the wake of COVID-19 there have been bigger picture issues to consider before focusing on individual divers. Disaster triage has already occurred in many countries at community or national level. These situations have led to closure of occupational and recreational diving industries. High prevalence and huge numbers of acute cases in the community have posed far too great a risk for diving operations. Undertaking dive medicals (and many other services) in an overwhelmed health system is just not feasible.

Even if a nation's system is coping, face to face health assessments in a setting of high disease prevalence also create risk. Australia has witnessed a significant shift to telemedicine for consultations in general practice and the specialties. Consistent with this, a pragmatic approach for established occupational divers (with documented previous comprehensive dive medical assessments) has been to reassess by telephone interview. Undertaken by a diving doctor who has access to the diver's current health information, an extension may be issued for the diver's medical certification. In New Zealand where occupational diver health data is centralised, a system of on-line review of divers already operates.

A second level of triage could occur as the health system starts to function normally, still has active COVID-19 cases, but many more have recovered in the community. In this scenario, divers will be seeking to return to the water. Australia and New Zealand have currently reached this point.

Screening questionnaires and measurement of temperature are currently used in Australian hospitals as a risk control, and to identify suspected SARS-CoV-2 cases. It seems sensible to me to use a screening questionnaire, including measurement of temperature and pulse oximetry, *before* undertaking a diving medical risk assessment. To be effective, screening questionnaires must be kept up to date, and relevant to known local community risks. One size

should not fit all. My own local hospital's questionnaire has already had three updates.

Further evidence of the rapidly changing environment are the Australian Communicable Diseases Network (ACDN) guidelines which have been updated 29 times since 23 January 2020.[5] These guidelines for Covid-19 infection definitions use a combination of factors (including epidemiological screening) to classify cases.

Using ACDN definitions, six potential 'COVID' scenarios for divers could be encountered.[5]

Confirmed or probable cases:

A. Actively symptomatic with COVID-19 infection;
B. Persistent symptoms after previous COVID-19 infection;
C. Asymptomatic but have recent positive screening test (undertaken due to other risk factors – example close contact of a positive case);
D. 'Asymptomatic' after previous COVID-19 infection (Severe/moderate/mild).

Suspected cases:

E. No symptoms but demonstrate clinical and epidemiological evidence of COVID-19 risk.

No suspicion of COVID-19 illness:

F. Never had symptoms and negative for confirmed, probable or suspected case criteria defined by ACDN.

It seems sensible to me to identify and exclude from further assessment (and from diving), divers in scenarios A, B and C above, until they are free of virus and symptoms. This principle is recommended by the University of California San Diego (UCSD) group in their guidelines to assess only divers who are 'completely asymptomatic' and with normal exercise tolerance.[6]

The UCSD group have provided guidelines for diver evaluation for COVID scenarios D and F above, by defining 4 categories:[6]

Category 0: Asymptomatic diver without history of COVID-19 suspected illness.
Category 1: Asymptomatic diver who had a mild COVID-19 suspected illness.
Category 2: Asymptomatic diver who had a moderate COVID-19 suspected illness.
Category 3: Asymptomatic diver who had a severe COVID-19 suspected illness.

The UCSD group provide some pragmatic guidelines on the assessment and investigations to undertake for the various categories of previous severity of infection. Publications such as this provide a platform to guide diving physicians, and perhaps adapt to their local setting.[6] The utility of specific investigations for divers who have recovered from SARS-CoV-2 infection hopefully will become better defined in the future.

Scenarios C and E are challenging to deal with, and many questions remain unanswered. For example, how long should an asymptomatic individual with a positive test wait before commencing diving? Should they be retested to confirm they are negative? Are negative COVID-19 tests useful to confirm absence of disease and that the individual is not contagious? A cautious approach is sensible. A process for dealing with asymptomatic spread of COVID-19 in divers has been documented by Belgian Society for Diving and Hyperbaric Medicine, recommending one month before diving after testing positive for COVID-19 (scenario C).[7]

For scenario E, how long should an individual with epidemiological risk factors wait before they can resume normal activities? In Australia, this requires compulsory 14-day quarantine. COVID-19 testing is not sufficiently mature to provide answers. On 22 May 2020, the Australian Therapeutic Goods Administration stated, "*there is limited evidence available to assess the accuracy and clinical utility of available COVID-19 tests*".[8] Serology, nucleic acid testing and PCR are becoming more available, and hopefully these test results will become robust enough to guide our clinical decisions.

Even if an individual diver demonstrates a negative test, there is no guarantee that they are free of other long-term pathophysiological effects from the disease. It may be reasonable to regard all suspected cases as 'probable' until further data is available.

Our lives have been irreversibly affected by COVID-19. At the start of 2020 we could not have imagined where we are now. There will continue to be 'ripple effects' beyond our current comprehension. Globally, diving has been seriously impacted. Many occupational divers are international citizens, for whom travel is no longer available. Diving physicians are also international citizens, and we have thrived on networking via diving medicine conferences. These processes will need to be re-established in the future. Recreational dive travel to exotic destinations will take a long time to recover. Local recreational diving has already been affected. Dive boats constitute a potential source of spread and should not be exempt from broader social distancing requirements. Shared dive hire gear and communal wash bins will become a thing of the past. Viricidal disinfecting agents used in occupational diving, are likely to become standard wash solutions for recreational diving.

Whatever the future holds, we need to stay safe, support and be kind to each other. The mental health and economic effects of this world-wide COVID-19 event will have far

reaching and long-lasting impacts for years to come. We are in a marathon not a sprint. To all my colleagues in diving medicine around the world, please turn to each other and ask "*are you ok?*"

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Conflicts of interest and funding: nil

Key words
COVID-19; Diving; Health; Risk assessment; Medicals – diving; Fitness for diving; Pandemic

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 http://www.anzca.edu.au/training/diving-and-hyperbaric-medicine.

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 2020
Viva examination: 09 September 2020

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.

Carl Edmonds Memorial Scholarship

The Australasian Diving Safety Foundation is delighted to announce the release of a new Diving Medical Officers Training scholarship to honour the memory of Carl Edmonds, a Founder of SPUMS and a mentor to diving physicians throughout the world. The AUD$5,000 scholarship is to encourage doctors to attend a Royal Australian Navy Underwater Medicine Course at the School of Underwater Medicine in Sydney. One scholarship is available for each course, two of which are planned for 2020.

Application details are available at:
http://adsf.org.au/grants/scholarships/diving-medical-training

Royal Australian Navy Medical Officers' Underwater Medicine Course 2020

Venue: HMAS Penguin, Sydney

Date: 19–30 October 2020 (these dates are yet to be confirmed)

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

Cost: AUD$1,355.00 without accommodation (AUD$350.00–400.00 on Base accommodation pending approval).

For information and application forms contact:
Rajeev Karekar, for Officer in Charge
rajeev.karekar@defence.gov.au

SPUMS Facebook page

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

SPUMS Diploma in Diving and Hyperbaric Medicine

Requirements for candidates (May 2014)

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

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

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

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

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

Additional information – prospective approval of projects is required

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

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

It is expected that all research will be conducted in accordance with the joint NHMRC/AVCC statement and guidelines on research practice, available at: www.nhmrc.gov.au/_files_nhmrc/publications/attachments/r39.pdf, or the equivalent requirement of the country in which the research is conducted. All research involving humans, including case series, or animals must be accompanied by documentary evidence of approval by an appropriate research ethics committee. Human studies must comply with the Declaration of Helsinki (1975, revised 2013). Clinical trials commenced after 2011 must have been registered at a recognised trial registry site such as the Australia and New Zealand Clinical Trials Registry http://www.anzctr.org.au/ and details of the registration provided in the accompanying letter. Studies using animals must comply with National Health and Medical Research Council Guidelines or their equivalent in the country in which the work was conducted.

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

Projects will be deemed to have lapsed if:

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

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

The Academic Board reserves the right to modify any of these requirements from time to time. As of January 2016, the SPUMS Academic Board consists of:

Dr David Wilkinson, Education Officer, Adelaide
Professor Simon Mitchell, Auckland

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

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

EUBS Notices and news

EUBS notices and news and all other society information is now to be found mainly on the society's website: https://www.eubs.org/

EUBS President's message

Ole Hyldegaard

COVID-19 has changed our world – temporarily or permanently?

Hyperbaric oxygen therapy and Covid-19

The last six months have changed our world as we once knew it. Several members of our scientific community have been hit hard – some harder than others within the European region. The Covid-19 pandemic has had (and still has) a severe impact on how we perform hyperbaric oxygen treatment (HBOT), advise divers and perform diving medicals. The EUBS has endorsed several recent guideline documents on both HBOT procedures, diving medicals and return to diving from Covid-19 disease. I would like to thank all good colleagues from the EUBS ExCom, EUBS members and ECHM committee members who participated in the drafting, discussions and final publication of these guidelines. In cooperation with the ECHM, these guidelines describe on how to handle both acute and elective planned HBOT sessions when dealing with patients in a confined environment regardless of whether patients are suspected or tested positive for Covid-19 or not. In this context, many treatment facilities all over Europe have had to temporarily suspend planned activities, or on reopening doing so with reduced treatment capacity allowing greater inter patient distance within the chamber environment, and adhering to more aggressive and strict cleaning / disinfection procedures just to mention a few. Obviously, these restrictions mean that the overall treatment capacity will be reduced unless the local HBOT facility operates more hours on a daily basis.

We should all remember; the patients are still there and require our assistance – restrictions caused by Covid-19 or not. The different guidelines on HBOT and Covid-19 are published on the EUBS website and translated into several European languages. We recommend that anyone involved in HBOT adhere to these guidelines and have them adjusted and used in agreement with local, national regulations with respect to Covid-19. Nonetheless, as the pandemic wanes over time and our societies slowly but surely recover and return to more normal activities, so will (and should) the capacity and treatment procedures of HBOT centres. In this respect, it's important that the recommendations and our procedures regarding the handling of HBOT patients will be adjusted in parallel to the opening of other hospital procedures and ambulatory visits to the greatest extent possible so we align with general recommendations and avoid unnecessary (or specifically restrictive) procedures that could otherwise result in an unjustified and undocumented overall reduction in HBOT treatment activity.

Fitness to dive, diving medical guidelines and Covid-19

Similarly, guidelines on the return to diving after possible or confirmed Covid-19 infection have been published. Divers should be aware that Covid-19 infections and especially those with previous symptoms and more intensive treatment requirements may require extended examination and pause from diving activities. There are still lessons to be learned, and experiences from practical diving procedures and possible Covid-19 sequelae to be collected. The current recommendations are therefore obviously dynamic documents. We will keep members of the EUBS posted through our website and e-mail newsflashes when changes in recommendations and guidelines may occur. Return to more normal activities is slowly emerging but different nations are at different levels and timing within their epidemic – thereby also causing different travel restrictions on a global scale that will likely persist for several months to come.

Randomised trials studying HBOT in treating Covid-19

It is encouraging to see the different initiatives taken globally to perform different scientific studies on the effects of HBOT in the possible treatment of Covid-19 patients. Randomised controlled trials are highly recommended and endorsed by the EUBS, and if HBOT could prove a useful adjuvant therapy in the increasing armamentaria of drugs fighting the virus this could have important impact for patients and our community in general. So far only anecdotal report suggest possible effects for HBOT but hopefully some of the planned studies will generate sufficient information and power to support future recommendations. There may be light at the end of the tunnel after all.

On behalf of the EUBS Excom we wish you all good health and stay safe.

Ole Hyldegaard
EUBS President

EUBS Facebook page

Follow us for updates at:
https://www.facebook.com/European-Underwater-and-Baromedical-Society-283981285037017

Postponed: EUBS2020 Scientific Meeting on Diving and Hyperbaric Medicine

Due to the current COVID-19 pandemic and the ongoing uncertainty concerning the possibility of holding a 'physical' meeting in September, the EUBS ExCom has decided to postpone the 46th EUBS Annual Scientific Meeting to next year. This decision has not been made lightly, as we are fully aware of the importance, both scientifically and socially, of an 'in-person' gathering of our members. However, as the time of our meeting comes closer, we needed to consider increasing cancellation costs and a possibly very low attendance.

Our 46th Annual Scientific Meeting will thus take place in Prague, Czech Republic, in September 2021. Those who have already registered will be contacted by the organising committee and their registration will be automatically valid for next year. All submitted abstracts should be considered 'released'.

EUBS ExCom is reviewing different solutions for the annual General Assembly, which should be held every year. We will communicate about this shortly. Also, the possibility to have one or several 'webinar' sessions on specific topics is under investigation, even though we realise that these can in no way replace a scientific meeting.

EUBS Elections, Member-at-Large

Around the time of publication of this issue of DHM, the election process for the 2020 ExCom members (Member-at-Large) of EUBS will have been started.

Member-at-Large: we will be saying goodbye to Dr med. Rodrigue Pignel as Member-at-Large 2017. The ExCom extends their thanks to Rod for the work he did in ExCom, and hopes to be able to continue counting on his support and help.

Candidates for the position of Member-at-Large 2020 will be presenting themselves on the EUBS website with a picture and short CV and you will receive soon, by email, an internet ballot where you can cast your vote.

If you have not received such an email by the end of June, please notify us at secretary@eubs.org, and we will work with you to find out the reasons why. As the system works via e-mail, it is possible the message ended up in your spam folder. There may be other reasons but usually, we are able to solve them.

COVID-19 Pandemic: EUBS – ECHM Position Statements

As the COVID-19 pandemic affects not only the general healthcare system but also diving and hyperbaric medicine, a number of position statements have been published by EUBS, ECHM and other scientific organisations in our field.

These position papers are now all available on the EUBS website, in the 'Endorsed Docs & Guidelines' section. They are free to download and circulate.

For now, four official EUBS–ECHM Statements are available:

- ECHM position on HBOT and COVID-19 (16 March 2020).
- EUBS–ECHM position on diving and COVID-19 (26 March 2020).
- ECHM–EUBS position on the use of HBOT in the treatment of COVID-19 patients (01 May 2020).
- ECHM–EUBS position on resuming recreational and professional diving after the Coronavirus disease (COVID-19) outbreak.

The EUBS endorses other statements made by affiliate organisations, and these can also be found on the EUBS site. There are statements regarding the medical surveillance of divers after COVID-19, on the restarting of HBOT activities, and others. The page will be updated as new Statements become available. http://www.eubs.org/?page_id=227.

You are free to distribute these documents, or any of the translated versions downloaded from the EUBS website.

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, logo's 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

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.

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

Hyperbaric Oxygen, Karolinska

Welcome to: http://www.hyperbaricoxygen.se/

This site, supported by the Karolinska University Hospital, Stockholm, Sweden, offers publications and high-quality lectures from leading investigators in hyperbaric medicine. Please register to obtain a password via email. Once registered, watch on line, or download to your iPhone, iPad or computer for later viewing.

For further information contact via email:
folke.lind@karolinska.se

The EUBS website is at
https://www.eubs.org/

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

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

Courses and meetings

Scott Haldane Foundation

As an institute dedicated to education in diving medicine, the Scott Haldane Foundation has organized more than 295 courses all over the world, over the past 26 years. SHF is targeting more and more on an international audience with courses world wide. Due to the COVID-19 pandemic some courses are re-scheduled. Fortunately we were able to find new dates for all courses in 2020. Below the upcoming SHF-courses in 2020.

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).

2020	
04–05 September	Medical Examiner of Divers part 1 Zeist, NL
10–12 September	Medical Examiner of Divers part 2 Amsterdam Univ. Med. Centre, NL
16–17 October	In-depth course Decompression, Recompression and HBOt (Level 2d) Mil. Hospital, Brussels, Belgium
28–29 October	Internship different types of diving (2d) Den Helder NL
07–14 November	Medical Examiner of Divers part 1, TBD
14–21 November	28th In-depth course diving and mental health, TBD
21–28 November	28th In-depth course diving and mental health, TBD
2021	
10–07 April	Medical Examiner of Divers part 2 Bonaire, Dutch Caribbean
On request	Internship HBOT (level 2d certification) NL/Belgium

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

Please also check the COVID-19 news update on this website for the latest schedule changes.

Capita Selecta Diving Medicine

The Capita Selecta Diving Medicine of the University of Amsterdam annually offers symposia presented by internationally renowned speakers to a multinational audience of diving physicians, paramedics and highly educated instructors. The level is advanced (1 and 2d) and often beyond that. All lectures are in English.

Due to the COVID-19 pandemic this is postponed until 2021. Dates are to be confirmed.

Visit: http://www.duikresearch.org/

For more information: n.a.schellart@amsterdamumc.nl

German Society for Diving and Hyperbaric Medicine (GTÜM)

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

Baltic International Symposium on Diving and Hyperbaric Medicine – new dates

The 2nd Baltic International Symposium on Diving and Hyperbaric Medicine (BIS_on_DHM) will now take place in Gdynia, Poland, (**new dates**) from 10–12 December 2020. There will also be two satellite Masterclasses; one on Advanced Diving Medicine and the other one on Complications in HBOT with a possibility to participate in the fire drills inside the hyperbaric chamber, to get wet under pressure!

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

DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA

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Website: www.classicdiver.org

Diving and Hyperbaric Medicine: Instructions for authors

Full verison (updated May 2020)

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

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

Types of articles

DHM welcomes contributions of the following types:

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

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

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

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

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

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

Formatting of manuscripts

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

Title page: Irrespective of article type, it must have a Title page which lists the title of the paper, all authors' names in full and their affiliations and provide full contact details for the first (and corresponding, if different) author(s).

Key words: The title page must also list a maximum of seven key words best describing the paper. These should be chosen from the list on the journal website DHM Key words 2019 or on the Manuscript Manager website. New key words, complementary with the US National Library of Medicine NML MeSH, https://www.nlm.nih.gov/mesh/meshhome.html/ may be used but are at the discretion of the Editor. Do not use key word terms that already appear in the title of your article.

Text format: The preferred format is Microsoft Office

Word or rich text format (RTF), with 1.5 line spacing, using both upper and lower case throughout. The preferred font is Times New Roman, font size 11 or 12. Please avoid using auto formatting tools such as automatic spaces before and after paragraphs. Lines **must** be numbered **continuously** throughout the manuscript to facilitate the review process.

Section headings should conform to the current format in DHM
This is:
Section heading (for Introduction, Methods, etc)
SUBSECTION HEADING 1
Subsection heading 2

Numbering: All pages must be numbered, but no other text should appear in the header and footer space of the document. Do not use underlining. No running title is required.

English spelling will be in accordance with the Concise Oxford Dictionary, 11th edition revised (or later). Oxford: Oxford University Press; 2006.

Measurements will be in SI units (mmHg are acceptable for blood pressure measurements) and normal ranges should be included where appropriate. Authors are referred to the online BIPM brochure, International Bureau of Weights and Measures (2006), The International System of Units (SI), 8th ed, available as a pdf at https://www.bipm.org/en/publications/si-brochure/. Atmospheric and gas partial pressures and blood gas values should be presented in kPa (atmospheres absolute [abbreviated as atm abs]/bar/mmHg may be provided in parenthesis on the first occasion). The ambient pressure should always be given in absolute not gauge values unless there is a particular reason to use gauge pressure and the distinction is made clear. Water depths should be presented in metres of sea (or fresh) water (msw or mfw). Cylinder pressures may be presented as 'bar'.

Abbreviations may be used once they have been shown in parenthesis after the complete expression. For example, decompression illness (DCI) can thereafter be referred to as DCI. This applies separately to the abstract and main text. Use generally accepted abbreviations that readers are likely to be familiar with rather than neologisms of your own invention. The overuse of abbreviations is strongly discouraged.

References: References should be numbered consecutively in the order in which they are first mentioned in the text, tables or figures where they should appear as superscript numbers, either following the statement referenced,[1] or at the end of the sentence, **after the full stop**.[1,2] Do not use references in the Abstract. References appearing in tables or figures or their legends should continue the sequence of reference numbering in the main text of the article in accordance with the position of first citing the table/figure in the text. Use MEDLINE abbreviations for journal names. Journals not indexed in MEDLINE should have the journal name written in full.

The Journal reference style is based exactly on that of the International Committee of Medical Journal Editors (ICMJE) *Recommendations for the Conduct, Reporting, Editing and Publication of Scholarly Work in Medical Journals: Sample References* (updated April 2018) https://www.nlm.nih.gov/bsd/uniform_requirements.html. Examples of the formats for different types of references (journal articles, books, monographs, electronic material, etc.) are given in detail on this website. Authors MUST consult this in preparing their reference list.

An example of a journal reference in the ICMJE format is:

Wilson CM, Sayer MDJ. Transportation of divers with decompression illness on the west coast of Scotland. Diving Hyperb Med. 2011 June;41(2):64–69.

If a journal uses continuous pagination throughout a volume (as many do) then the month and issue number should be omitted and the pagination reduced. Therefore, the shortened ICMJE version used in DHM is:

Wilson CM, Sayer MDJ. Transportation of divers with decompression illness on the west coast of Scotland. Diving Hyperb Med. 2011;41:64–9.

If an article has a unique identifier for the citation (e.g., PubMed PMID, PubMed Central PMCID or DOI number) then this must be included at the end of the reference. The format and order for this is:
doi: number. PMID: number. PMCID: number. For example:

Doolette DJ, Mitchell SJ. In-water recompression. Diving Hyperb Med. 2018;48:84–95. doi: 10.28920/dhm48.2.84-95. PMID: 29888380. PMCID: PMC6156824.

An example book reference is:

Kindwall EP, Whelan HT, editors. Hyperbaric medicine practice, 3rd ed. Flagstaff (AZ): Best Publishing Company; 2008.

Examples of many other types of references are to be found on the National Library of Medicine site (see link above)

When citing workshop/conference proceedings or technical reports, authors are requested to investigate their availability on-line, and provide an on-line source for the reference if available. The date that the reference was cited (year/month/day) from the source should be noted. For example:

Goodman MW, Workman RD. Minimal-recompression, oxygen-breathing approach to treatment of decompression sickness in divers and aviators. Research Report NEDU TR 5-65. Washington (DC): Navy Experimental Diving Unit;

1965. Available from: http://archive.rubicon-foundation.org/3342. [cited 2019 Sep 12].

Many of the proceedings and technical report documents commonly cited in diving and hyperbaric medical manuscripts can be found on The Rubicon Research Repository website http://archive.rubicon-foundation.org/xmlui/.

Additional notes regarding referencing in DHM are:

If using **EndNote** to prepare the references in the document see EndNote website for advice. Once accepted, the final version of the submitted text should have all EndNote field codes removed.

Verifying the accuracy of references against the original documents is the responsibility of authors.

Personal communications should appear as such in the text and not be included in the reference list (e.g., Smith AN, personal communication, year).

Abstracts from meeting proceedings should not be used as references unless absolutely essential, as these are generally not peer-reviewed material.

Please avoid using auto-formatting functions like numbering, indentations, and spaces before and after paragraphs in compiling your reference list.

Tables must **not be** embedded in the main manuscript document. They are to be uploaded as separate Word documents (one document per table) in Manuscript Manager (use the 'other' category when asked to select a description of the document being uploaded). Name the document with the first author's name and table number as appropriate. Tables need to be labelled at the top of the page with **first** author name and the Table number.

Tables should be presented using MS Word table format with frames shown, auto-formatted to fit content. Please avoid complicated, large tables whenever possible. Very large tables (full page or more) may not be incorporated into the final article but, rather, displayed in the journal website as additional material at the Editor's discretion.

The title of the table and caption are not to be included in the table. These appear in the 'legends and captions' section at the end of the manuscript document. Legends should generally contain fewer than 40 words and should be thorough enough to be understood independently of the main text.

The table must be mentioned within the text of the article, e.g., "*Differences in rates of decompression illness were not significant (Table 1)*", etc. The approximate positions of tables/figures should also be identified in the manuscript text.

Figures (including photos, graphs, diagrams, illustrations and radiographs) must **not be** embedded in the main manuscript document. They are to be uploaded as separate electronic files in high resolution TIFF or JPEG format in Manuscript Manager. Name the document with the first author's name and figure number as appropriate. Figures should be uploaded to Manuscript Manager in their numbered order, which results in them being compiled in the review document in correct order.

The title of the figure and caption are not to be included in the figure. These appear in the 'legends and captions' section at the end of the manuscript document. Legends should generally contain fewer than 40 words and should be thorough enough to be understood independently of the main text. Magnification should be indicated in the captions for photomicrographs, and consideration given to the positioning of labels on diagnostic material as this can greatly influence the size of reproduction that can be achieved in the published article.

Graphs may be submitted either in colour or grey-scale, with no unnecessary shading, grid lines or box lines. Please choose the simplest graphical format that displays the data effectively. 3-D graphs are discouraged unless they are necessary to display 3-D data. Both markers and lines should be unique to facilitate easy discrimination of the data being presented. Special attention should be given to ensuring that font sizes within a diagram are sufficiently large to be legible should the diagram be sized for single-column presentation. The preferred font in diagrams and graphs is Times New Roman. Graph symbol keys should appear within the white space of the figure (not outside the axes) if possible or be included in the legend. Please ensure that axes are labelled using sentence case and the same data formatting conventions presented below.

Any graphs or histograms created in Excel should be sent within their original Excel file, including the data table(s) from which they were produced. This allows the journal office to edit figures for maximum legibility when printed. Upload the spreadsheet to Manuscript Manager with the other manuscript documents and select the designator 'other' and the option 'hide from reviewers' so that the spreadsheet is not incorporated in the review document.

Any photograph or radiograph of a patient must be de-identified. Patient details must be removed and photographs made unrecognizable. Colour photos are acceptable.

If any figures, images or tables are to be reproduced from previous publications, it is the responsibility of the author(s) to obtain the necessary permissions. This permission should be acknowledged in the figure caption using the format "*Reproduced with permission of .....*" or, if necessary, another format specified by the copyright holder granting permission.

Miscellaneous data formatting conventions: Please follow the following recommendations when presenting data in text, figures or graphs.

Standard deviations and standard errors should be expressed as mean (SD), not mean ± SD.

Composite units of measurement should be expressed as (for example) $g{\cdot}L^{-1}$ or $mL{\cdot}kg^{-1}{\cdot}min^{1}$, not g/L or mL/kg/min.

Please use a space between symbols like <, >, ≤, ≥. Thus (for example) > 25, not >25.

Please use decimal points and not commas in decimals. For example: 2.5, not 2,5.

Numbers greater than 999 should contain commas. For example: 1,000 or 25,300,000.

Please leave a space between a number and unit of measurement. For example: 25 msw.

Please italicize *n* when used to indicate number and *P* when used to indicate *P*-values.

Please leave spaces in expressions like $n = 25$ or $P < 0.05$ (not n=25 or P<0.05).

For number ranges please use an em dash without spaces. For example: 17–420. This also applies to page ranges when citing references.

Percent signs should immediately follow a number without a space. For example: 51% not 51 %.

Other manuscript requirements and guidelines

DHM follows as much as possible the ***Recommendations for the conduct, reporting, editing and publication of scholarly work in medical journals***. International Committee of Medical Journal Editors; December 2015.

Available from: http://www.icmje.org/recommendations/. Authors are strongly encouraged to read this and other documents on the ICMJE website in preparing their submission. Authors should also consult guidelines for specific types of study (e.g., the CONSORT guidelines for the reporting of randomized controlled trials); see http://www.equator-network.org/.

Trial design, analysis and presentation. Before preparing their manuscript, authors must read the summary advice on the journal website on the reporting of trial design, sample size calculation, statistical methods and results. http://www.dhmjournal.com/images/Docs/Trial-design-analysis-and-presentation.pdf.

Consent and ethical approval. Studies on human subjects must comply with the Helsinki Declaration of 1975, revised October 2013 (see https://www.dhmjournal.com/index.php/author-instructions for a copy).

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. It is insufficient to refer to previous publications for details of animal welfare and procedural care. The Physiological Society provides detailed advice regarding animal experimentation and its reporting in research publications and this link is provided with their kind permission: https://physoc.onlinelibrary.wiley.com/doi/full/10.1113/jphysiol.2010.192278.

A statement affirming Ethics Committee (Institutional Review Board) approval (and the approval number) should be included in the text at the beginning of the methods section. A copy of that approval should be uploaded with the submission. Similarly, a statement affirming the securing of written informed consent from subjects should be included in the methods where this was part of the methodology.

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/ or EudraCT in Europe https://eudract.ema.europa.eu/. Details of the registration must be provided in the accompanying MSF, and should also be mentioned in the methods section.

For individual case reports, evidence of informed patient consent to anonymous publication of their clinical details and/or images, etc. must be provided. Case series, where only limited anonymous summary data are reported do not require patient consent, but must have been assessed by an ethics committee and, if indicated, have ethics approval. Consult your local ethics committee if you are unsure.

Authorship: Authors must have contributed significantly to the study (see guideline to authorship at: https://www.dhmjournal.com/images/Docs/Guideline_to_authorship_in_DHM_journal-2015.pdf.
Inclusion of more than six authors in any one manuscript requires strong justification. Other contributors may be listed in the Acknowledgements section.

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Authors should consult the WAME website http://www.wame.org/about/conflict-of-interest-in-peer-reviewed-medical if they need further clarification.

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If the submission requires revision and resubmission before it can be accepted for publication (and the majority of papers do), then the revised files must be submitted by logging on again at http://www.manuscriptmanager.net/dhm with the same user name and password created for the original submission, then the article can be **resubmitted** by clicking the **resubmit** link NOT the new submission link. Do NOT create a new account.

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English as a second language. Adequate English usage and grammar are prerequisites for acceptance of a paper. However, some editorial assistance may be provided to authors for whom English is not their native language. English language services can be accessed through the European Association of Science Editors (EASE) website http://www.ease.org.uk/. Alternatively, the journal office may be able to put you in touch with a commercial scientific ghost writer.

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PDFs. Following publication, two electronic PDFs of articles will be forwarded to the corresponding author. One of these, watermarked "*restricted use*" may be placed on the author's institutional website during the one-year embargo following publication. Thereafter, the non-watermarked pdf may be used ad lib.

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and on the web platform. They are also available on the EUBS and SPUMS websites.

Summary of files to be uploaded in Manuscript Manager when submitting an article

Mandatory submission form.
Ethics approval letter where relevant, and/or signed patient consent.
Manuscript document.
Tables where relevant (each table as a separate Word document).
Figures where relevant, uploaded in the order in which they should appear in the manuscript (each Figure as a separate high resolution TIFF or JPEG file).
Excel spreadsheet with data and graphs if graphs have been generated in Excel.
Submission letter; authors can use this to communicate any particular considerations or issues they wish the editor to be aware of in relation to their manuscript. The letter should state that the paper is being submitted exclusively to DHM.

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 (this document)
DHM Key words 2019
DHM Mandatory Submission Form 2018
Trial design analysis and presentation
English as a second language
Guideline to authorship in DHM 2015
Helsinki Declaration revised 2013
Is ethics approval needed?

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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: http://adsf.org.au/grants/scholarships/diving-medical-training and send it by email to John Lippmann at johnl@adsf.org.au.

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