Diving and Hyperbaric Medicine

Providing oxygen for diving emergencies

The safety record of a dive charter business

Fatal shark attacks on divers are a rarity

Not all dive computers work the same way

Pharmacological reduction of oxygen toxicity

HBOT patients need nutritional assessment

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Editorials

Malnutrition screening in outpatients receiving hyperbaric oxygen therapy: an opportunity for improvement?

Outpatients who receive hyperbaric oxygen treatment (HBOT) may represent a group at significant risk of malnutrition owing to the underlying conditions that are often treated with HBOT (e.g., non-healing diabetic wounds and radiation-induced skin injury). In this issue, See and colleagues provide new, preliminary evidence of the prevalence of malnutrition in a small group of HBOT outpatients treated in an Australian hospital, reporting that approximately one-third of patients receiving HBOT were at risk of malnutrition.[1]

To our knowledge, routine malnutrition screening is not available in HBOT centres providing outpatient treatment, which may be a key gap in the nutrition care of these patients. Malnutrition screening was developed to identify those at risk of malnutrition across the healthcare continuum.[2] In the outpatient setting, it is recommended that patients are screened at their first clinic appointment and that screening is repeated when there is clinical concern.[2] Malnutrition screening tools are designed to be quick and simple to complete by trained healthcare staff and include questions relating to appetite, oral intake and recent weight loss.[2,3] The early identification of patients at risk of malnutrition using validated screening tools enables the appropriate and timely referral of patients to dietetic services for assessment and treatment.[2,3]

Why might malnutrition screening in HBOT services be important? It is well documented that the consequences of malnutrition are systemic, with increased morbidity and mortality attributed to malnutrition.[4] Beyond the detrimental impact of malnutrition to the individual, malnutrition also has significant economic ramifications, with medical costs significantly higher in severely malnourished compared to well-nourished patients.[5] Of particular relevance, malnutrition is associated with impaired and prolonged wound healing.[6] This may influence the effectiveness and success of HBOT treatment, although studies in the area of HBOT and concurrent nutrition therapy are lacking.

Furthermore, there are no reliable markers of nutrition status that are easily obtainable in the healthcare setting. In the past, prealbumin (transthyretin) and albumin have been used as surrogate markers of nutritional status.[4] However, these serum proteins are acute-phase proteins and, therefore, are reduced during acute inflammation and infection, making them unreliable indicators of nutrition status.[4,6] Transferrin, retinol binding protein and C-reactive protein are similarly not recommended as markers of nutrition status and malnutrition.[4,6] Therefore, the implementation of malnutrition screening may be the most practical and validated method of identifying patients who would benefit from a comprehensive assessment of their nutrition status and provision of nutrition support in the HBOT setting.

The assessment of nutrition status involves the collective evaluation of anthropometric data, biochemical markers, clinical symptoms impacting on nutrition (e.g., nausea) and oral intake. Tools such as the subjective global assessment have been developed and validated to assess nutrition status and diagnose malnutrition by trained staff.[4] In contrast to other outpatient services, HBOT presents a unique opportunity to complete both malnutrition screening and engage a relevant dietetic service for nutrition assessment early in the course of treatment. The frequent contact with outpatients would also lend itself well to group nutrition education sessions to address important nutrition information related to wound healing.

Although there is a paucity of data to support the use of malnutrition screening and dietetic assessments in HBOT, current best practice guidelines recommend these services in outpatient settings.[2] The implementation of routine malnutrition screening and referral processes to dietetic services warrants consideration in the HBOT outpatient setting. If going down this path, careful consideration of available resources, how referral systems can be incorporated into current procedures as well as partnership with dietetic departments is integral. In the interim, the referral of patients to dietetic departments who are suspected to be at risk of poor wound healing due to nutrition factors and those failing treatment should be considered by treating hyperbaric physicians. Although further research is required to assess the effectiveness of malnutrition screening and nutrition intervention in the HBOT outpatient population, the data by See and colleagues provides an important starting point in unpacking malnutrition risk in this population.

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The Editor's offering

This is my final Editor's offering, as I retire as Editor at the end of this year. As such, I thought it worthwhile to tell members of EUBS and SPUMS something about the development of *Diving and Hyperbaric Medicine* (DHM) and how I have seen its role for the societies. In the inaugural John J Bonica lecture at the University of Washington, Seattle, in the early 1970s, Tom Hornbein, famously known for his ascent of the South Face of Everest without oxygen, described the three pillars upon which the department of anesthesiology, indeed the university hospital as a whole, was founded – Service, Education and Research. The same may be said of SPUMS and EUBS and of this, your Journal. Many members provide clinical services to their communities in hospitals, hyperbaric medicine units, the wider community and to the recreational, occupational and military diving communities. DHM obviously serves alongside the societies' annual scientific meetings to educate and to provide a forum for the publication of clinical and applied scientific research, thus supporting all three pillars of our medical endeavours.

In the first joint SPUMS/EUBS issue of DHM in March 2008, I quoted Richard Smith, a past Editor of the *British Medical Journal*, who wrote that journals were for readers first and foremost and what they do best "*is what the rest of the media do best: stir up, prompt debate, upset, probe, legitimise and set agendas. They are good at telling readers what to think about but not what to think* …".[1] This has continued to be my 'lodestar' (Judge Kavanaugh before the US Senate hearing) as Editor of DHM.

Nevertheless, the quality of the articles submitted and published remains the keystone to the success of DHM. Without good support from authors, no minor specialty journal can survive; so, thank you everyone who has submitted their work over the years! To achieve as high a standard as possible, a strong peer review process has been put in place. Some may regard this as a barrier to publication, but my philosophy has always been to try to facilitate publication – the majority of submissions have a kernel of worth to them and one must strive to tease this out from the verbiage. I am convinced that there has never been a single paper published in DHM that has not benefitted from this process and been a better read as a result. Most authors have little or no education or guidance in how to write a scientific paper and I know from my own experience that one can only improve with practice. Your new Editor, Simon Mitchell, is a case in point, having had the good fortune myself to watch his career closely over several decades. Members can rest assured that DHM will be in excellent hands.

During my nearly 17 years as Editor, there have been four key moments in the development of DHM.
•   The first was the amalgamation in 2008 of the SPUMS Journal (by that time indexed on Scopus/Embase and newly named Diving and Hyperbaric Medicine) with EUBS's non-indexed and struggling European Journal of Underwater and Hyperbaric Medicine to become a specialty journal for our two societies. The three-way relationship between the two societies and myself as Editor has not always been an easy one. Nevertheless, this pooling of resources has proved extremely successful and I firmly believe it benefits both societies. I feel that EUBS as a whole has had less of a sense of 'ownership' than has SPUMS. This is understandable since DHM is registered and published in Australia. However, from an editorial standpoint this is a very real partnership, evidenced by our 16-person, international Editorial Board from 12 countries, including eight from Europe.

- The second was achieving MedLine citation in 2011 – a hard won recognition that involved our successfully appealing an initial rejection by the National Library of Medicine (NLM).
- The third was the adoption in 2015 of web-based management of submissions to the journal, via the web platform Manuscript Manager. This has greatly enhanced the efficiency of the DHM office and allows easy communication between the editors, the editorial assistant, reviewers and authors. Even after three years we are still learning how to best take advantage of its capabilities.
- The fourth was this year, with DHM as an e-publication and becoming archived on PubMed Central (PMC).

Moving to e-publication changed our status with MedLine in that we were now required to deposit articles from DHM with PMC or one of the few commercial archiving services recognised by the National Library of Medicine if we were going to retain MedLine Abstract citation. We chose to apply for PMC archiving for many reasons, some of which are set out in the following quotations from PMC Overview.[2]

"*PubMed Central® (PMC) is a free archive of biomedical and life sciences journal literature at the U.S. National Institutes of Health's National Library of Medicine (NIH/NLM). Five million articles are archived in PMC.*

As an archive, PMC is designed to provide permanent access to all of its content, even as technology evolves and current digital literature formats potentially become obsolete. NLM believes that the best way to ensure the accessibility and viability of digital material over time is through consistent and active use of the archive. For this reason, free access to all of its journal literature is a core principle of PMC.

In addition to its role as an archive, the value of PMC lies in its capacity to store and cross-reference data from diverse sources using a common format within a single repository. With PMC, a user can quickly search the entire collection of full-text articles and locate all relevant material. PMC also allows for the integration of its literature with a variety of other information resources that can enhance the research and knowledge fields of scientists, clinicians and others."

This proved to be a far more challenging task than we anticipated, adding an enormous amount of work for myself and, more particularly, our Editorial Assistant, Nicky Telles. Nicky has done an outstanding job for you in navigating the challenges of creating the necessary XML files for archiving. As a result, all articles in DHM from the March 2017 issue will be searchable on PMC in perpetuity (that is, until climate change destroys modern society, which at the current rate seems likely!). We still have some catching up to do, but the March, June and September 2017 issues are now accessible and the December one will be ready early in the New Year. PMC supports our one-year embargo policy, so further issues will appear as they come out of their one-year

embargo period, except for articles for which authors have paid a fee for immediate release, which will appear earlier.

As part of this process, DHM is expected to comply fully with the recommendations of the International Committee of Medical Journal Editors (ICMJE), the World Association of Medical Editors (WAME) and the ethical standards promulgated by the Committee on Publication Ethics (COPE). These also impact authors submitting their work. For this reason, various support documents to help with preparing manuscripts are to be found on the DHM website (http://www.dhmjournal.com/index.php/author-instructions), with more to come. These increased standards are reflected in a rejection/author withdrawal rate that is a disappointing 29% over the past four years. I am particularly indebted to our large cadre of volunteer reviewers for their unflinching support (see p. 270 of this issue).

Articles come from all over the world, with Australia (20%), the United Kingdom (9%) and Turkey (7.5%) being the leading submitting countries. There are some notable gaps and over half of all European publications in the fields of diving and hyperbaric medicine occur in other journals. Both Simon Mitchell and I encourage a change to this attitude – DHM is the leading journal devoted entirely to diving and hyperbaric medicine and physiology, with a steadily improving five-year Impact Factor (IF, see p. 275 of this issue). Since DHM became indexed on PubMed, its contents have been increasingly cited and retaining an IF of around 1.2 is a satisfactory situation for a small specialty journal such as this. With all articles from early 2017 onwards now to be permanently deposited in PubMed Central and all having DOI links, this will further enhance the visibility of our authors' work. There is every reason, in most though not all instances, to regard DHM as your first choice for publication. It is disappointing to see potentially good but poorly written articles published elsewhere.

I wish everyone the very best for the future – good health, happiness and success in all your endeavours!

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Michael Davis

Front page photo: A mast on the wreck of the Hoki at Truk Lagoon, taken by the incoming editor, Simon Mitchell. A riot of colour to wish all our readers a healthy and successful 2019.

Original articles

Comparison of tissue oxygenation achieved breathing oxygen from a demand valve with four different mask configurations

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Abstract

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Introduction: High concentration normobaric oxygen (O_2) is a priority in treating divers with suspected decompression illness. The effect of different O_2 mask configurations on tissue oxygenation when breathing with a demand valve was evaluated.

Methods: Sixteen divers had tissue oxygen partial pressure ($P_{tc}O_2$) measured at six limb sites. Participants breathed O_2 from a demand valve using: an intraoral mask (IOM®) with and without a nose clip (NC), a pocket face mask and an oronasal mask. In-line inspired O_2 (F_IO_2) and nasopharyngeal F_IO_2 were measured. Participants provided subjective ratings of mask comfort, ease of breathing and holding in position.

Results: $P_{tc}O_2$ values and nasopharyngeal F_IO_2 (median & range) were greatest using the IOM® with NC and similar with the IOM® without NC. O_2 measurements were lowest with the oronasal mask which also was rated as the most difficult to breathe from and to hold in position. The pocket face mask was reported as the most comfortable to wear. The NC was widely described as uncomfortable. The IOM® and pocket face mask were rated best for ease of breathing. The IOM® was rated as the easiest to hold in position.

Conclusion: Of the commonly available O_2 masks for use with a demand valve, the IOM® with NC achieved the highest $P_{tc}O_2$ values. $P_{tc}O_2$ and nasopharyngeal F_IO_2 values were similar between the IOM® with and without NC. Given the reported discomfort of the NC, the IOM® without NC may be the best option.

Introduction

Arterial gas embolism (AGE) and decompression sickness (DCS), collectively termed decompression illness (DCI), are risks for scuba divers. AGE results from pulmonary barotrauma introducing gas directly into the vascular system. DCS is caused by the formation of bubbles primarily from dissolved inert gas.[1] During a dive, an increase in the ambient pressure leads to a higher breathing gas pressure being delivered to the lungs, and more inert gas (nitrogen in air diving) dissolving in the blood and body tissues. This process is called on-gassing. At the end of a dive as the diver ascends, both the ambient pressure and the pressure of the inspired inert gas decrease. The on-gassing process is reversed as tissue inert gas diffuses into the blood for carriage back to the lungs. However, if the pressure of dissolved gas in a tissue significantly exceeds ambient pressure (a condition referred to as '*supersaturation*') the gas may form bubbles either in the extravascular space or within tissue capillaries. These bubbles are considered to be the primary cause of injury leading to the symptoms of DCS.

It follows from the above that arresting the growth of bubbles and encouraging their involution is a primary goal of treatment in DCS. Breathing a high concentration of oxygen (O_2) and therefore a lower partial pressure of inert

gas, creates a larger diffusion gradient between the blood and body tissues and blood and alveoli such that more inert gas moves from the tissue into the blood, and is then transported to the lungs to be exhaled.[2] Bubbles formed in decompressed divers without DCS have been demonstrated to resolve more quickly during O_2 breathing.[3] Therefore, it is recommended that O_2 be given early to a diver with signs and symptoms of DCI, DCS and AGE, enhancing inert gas elimination from the body and supplying O_2 to hypoxic tissues.[2,4–6]

The current pre-hospital care recommendation for divers with symptoms and signs of DCI is for O_2 delivery at the highest possible concentration (close to 100%).[7] Divers Alert Network (DAN) has designed a variety of portable O_2 delivery units to provide divers with pre-hospital O_2.[8,9] These units have two common components: (1) a constant flow capability for use with a non-rebreather mask (NRB) or other constant flow delivery device; and (2) a pressure-activated demand valve. Previous research comparing tissue oxygenation found that the NRB performed better than the demand valve with an oronasal mask.[10] This research has been questioned as experts believe that the demand valve should be able to provide near 100% O_2 and therefore better tissue oxygenation than the NRB.

The present study used transcutaneous oximetry measurement (TCOM) to determine tissue oxygenation at multiple standardised sites in participants breathing O_2 from a demand valve using four different mask configurations. TCOM is a non-invasive technique that uses heated electrodes on the skin to measure the partial pressure of tissue oxygen $(P_{tc}O_2)$.[11] The null hypothesis was that there would be no difference in the $P_{tc}O_2$ achieved after 10 minutes (min) of breathing O_2 with any of four different mask configurations.

Methods

Ethics approval was granted from the Townsville Health Service District Human Research Ethics Committee (HREC16/QTHS/196). The volunteers for this study were healthy non-smoking certified scuba divers of both sexes. Participants were recruited from James Cook University and the diving community in Townsville, Queensland, Australia. All participants were older than 18 years of age and had performed at least one dive within the previous year. Exclusion criteria included facial hair or anatomical abnormality that might impair mask seal, any medical condition or medication that may affect tissue oxygenation, or an allergy to the topical anaesthetic. All participants received a study information sheet and gave their written informed consent.

Participants were asked to refrain from consuming food or caffeine or performing heavy exercise for six hours prior to participating in the study. Age and sex were reported; height, weight and waist and hip circumferences were measured upon arrival for the study day. The participants were then placed in a supine position on a hospital stretcher with their head slightly raised on one pillow and remained in this position for the duration of the study. The room temperature was maintained between 22.4 and 23.7°C; participants were covered with a blanket for comfort and to limit any vasoconstrictive effects of being cold.

Resting baseline measures included heart rate, respiratory rate, O_2 saturation and blood pressure. Topical lignocaine (5%) and phenylephrine (0.5%) (Co-Phenylcaine™ forte spray, ENT Technologies Pty Ltd., Hawthorne East, Australia) was sprayed into the right nares and an 8 French paediatric feeding tube (ConvaTec Ltd., Deeside, UK) was inserted. Position was verified visually with the tip of the tube placed just proximal to the soft palate and the tube then secured in place. The tube was attached to the E-sCO-OO module of a bedside monitor (GE Carescape Monitor B650, GE Healthcare Finland OY, Helsinki, Finland) allowing for both inspired O_2 (F_IO_2, paramagnetic) and end-tidal carbon dioxide ($ETCO_2$, infrared) measurements via a water trap (D-fend Pro+ Water Trap™, GE Healthcare Finland OY, Helsinki, Finland). The gas module was calibrated against room air before each mask configuration was used. The gas sampling rate was 120 ml·min⁻¹.

Tissue oxygenation was measured using the TCM400 Transcutaneous (tc) PO_2 Monitoring System (Radiometer, Copenhagen, Denmark) with tc Sensor E5250. Zero current calibration of the $P_{tc}O_2$ electrode using CAL2 gas (10% CO_2 with N_2 as balance) was performed prior to commencement of the study and calibration with atmospheric air occurred prior to each monitoring period. A 'humidity correction factor' was entered into the machine prior to each monitoring period. All assessments were performed by the same technician (AB). The TCM400 displayed $P_{tc}O_2$ values in units of mmHg.

Six sensors were used: three on the left arm and three on the left leg.[10] One sensor was placed on the lateral aspect of the upper arm, mid-way between the acromial process and the olecranon process, one sensor 5 cm distal to the brachial crease on the lateral aspect of the lower arm and one sensor over the thenar eminence (palm hand). One leg sensor was placed 10 cm distal to the lateral femoral epicondyle (lateral leg); one 5 cm proximal to the lateral malleolus (lateral ankle) and one on the dorsum of the foot between the first and second metatarsal heads, attempting to avoid large superficial vessels. Participants rested quietly while the sensors were placed. They were requested to minimise talking during the study, but were not allowed to sleep. Initial normobaric, room air readings from all sensors were recorded after a minimum 20-min equilibration period that allowed all sensors to stabilize.

The participants were then asked to breathe O_2 for 10 min from a demand valve (L324-020, Life Support Products (LSP), Allied Healthcare Products, St. Louis, MO, USA)

Figure 1
Demand valve, spacer with side port, one-way valve configuration and intraoral mask

Figure 2
Study configuration with demand valve, spacer with side port and oronasal mask

Figure 3
Study configuration with demand valve, spacer with side port, in-line and intra-mask gas sampling lines, nasopharyngeal catheter and pocket face mask (with subject's permission)

using each of four different mask configurations in randomized order:

- Intraoral mask (IOM®) with nose clip (NC) (NuMask®, Inc., Woodlands Hills, CA, USA) (Figure 1);
- IOM® without NC;
- Adult soft silicone oronasal mask (Tru-Fit mask, Allied Healthcare Products Inc., St. Louis, MO, USA) (Figure 2);
- Pocket face mask with air cushion (Sturdy Industrial CO., Ltd., Wugu Shiang, Taipei County, Taiwan) (Figure 3).

The oronasal, pocket face mask and demand inhalator valve which are provided in portable DAN O_2 units were used for this study. A flexible high-pressure O_2 hose was used to connect the demand valve to the hospital wall medical grade O_2 outlet (415 kPa). The demand valve was attached to a spacer with a side port allowing pressure and gas measurements. To measure the delivered O_2, a one-way valve was attached to the spacer and a T-piece with a one-way exhaust valve on the side (Figure 1). The side port was connected to the bedside monitor and a delivered O_2 percentage of 99% was obtained. The one-way valves and T-piece were removed and the single spacer with side port (in-line F_IO_2 measurement) was attached to each O_2 mask in turn during the study (Figure 2). A pressure line

was attached to the side port and then to the bedside monitor via a BD DTXPlus™ pressure transducer (Argon Medical Devices Inc., Frisco, TX, USA). The monitor was configured to settings used for central venous pressure monitoring to give a high sensitivity in the lower range, and zeroed before each participant.

A single, new demand valve was used and the inspiratory opening pressure required to trigger the valve and the expiratory resistance pressure were verified prior to studying each new participant. The cracking pressure of the demand valve is 0 to -2 cm H_2O and exhalation pressure is 1.5 to 6.4 cm H_2O dependent on flow (LSP demand inhalator valve product insert). The pocket face mask has a nipple for the attachment of supplemental O_2, through which a gas sampling line (Microstream®, Oridion Medical Ltd., Jerusalem, Israel) was inserted and secured near the central opening of the mask to measure intra-mask F_IO_2 levels (Figure 3). Mask dead space was determined by measuring the amount of water required to fill each device. Fill levels were estimated by placing the masks on a mannequin's face and visual inspection of the intrusion of the facial features into the mask.

The order of the four O_2 mask configurations was randomised using the random number generator in Excel (Microsoft® Corporation, Redmond Washington, USA). The participants were asked to position and hold each mask for comfort and to ensure a tight seal to avoid leakage and to breathe deeply enough to trigger the demand valve as outlined in DAN educational material.[8,9] In-line F_IO_2, nasopharyngeal F_IO_2, $P_{tc}O_2$ and other respiratory measures were recorded at the end of the 10-min breathing period. Nasopharyngeal gas sampling was intermittent and frequent throughout the

Table 1
Demographic and baseline measurements for the 16 participants; IQR - inter-quartile range

Characteristic	Median (IQR)	Range
Age (years)	27 (23, 30)	20–57
Body mass index (BMI) (kg·m^{-2})	23 (22, 25)	17–29
Underweight (BMI < 18.5) (n)	1	
Normal (BMI 18.5–24.9) (n)	11	
Overweight (BMI 25.0–29.9) (n)	4	
Obese (BMI ≥ 30.0) (n)	0	
Waist-to-hip ratio		
Males (optimal < 0.82)	0.86 (0.84, 0.92)	0.84–0.92
Females (optimal < 0.71)	0.76 (0.74, 0.79)	0.69–0.88
Heart rate (beats·min^{-1})	66 (60, 72)	52–85
Systolic BP (mmHg)	109 (100, 116)	96–137
Diastolic BP (mmHg)	66 (58, 67)	52–87
Respiratory rate (breaths·min^{-1})	16 (12, 19)	12–22
Oxygen saturation (%)	97 (96, 98)	95–100
End-tidal CO_2 (mmHg)	36 (34, 39)	32–47

study to prevent clogging of the catheter and in an attempt to capture peak values. Consistent repeated values were seen during each oxygen breathing session. After each 10-min O_2 breathing period, participants breathed room air for 10 min, allowing all $P_{tc}O_2$ levels to return to baseline before the next mask was trialled.[12] At the end of the data collection period all participants used a five-point Likert scale to rate each mask configuration on comfort, ease of breathing, and ease of holding the device in place. A final open-ended question asked about any adverse effects while breathing O_2.

ANALYSIS

Collected data were de-identified and entered into a preformatted Excel worksheet, then exported into Statistical Package for the Social Sciences version 23.0.0 (SPSS®, IBM® Corporation, Armonk, New York, USA) for analysis.

Based on recent research, we expected mean $P_{tc}O_2$ values between 310 mmHg (forearm) and 421 mmHg (upper arm), with a sample standard deviation of 75 mmHg, when subjects breathed 100% oxygen.[13] Using the mid-point of that range (365 mmHg), assuming a difference of 75 mmHg (smallest increase in $P_{tc}O_2$ at one sensor site breathing 100% oxygen with a hood[13]) would be clinically significant, and allowing for substantial correlation (r = 0.90) between the repeated

measures, we estimated that a sample size of 16 subjects would provide a power of 80% (with α = 0.05) to detect significant changes in tissue oxygenation.

Using G*Power (version 3.1.9.2)[14] with medium effect size and the plan for a 2-way ANOVA, a sample size of 16 was calculated to give a power of 90% (α = 0.05) to detect a significant change in inspired O_2 values (notionally defined as 5%). We estimated this by using a 4 x 2 (4 masks x 2 O_2 values, in-line and nasopharyngeal F_1O_2) within and between factor analysis plan as no set values from previous research were available.

The Shapiro-Wilk test was used to evaluate normality of data distribution. None of the data were normally distributed. Thus, differences between median $P_{tc}O_2$, ETCO$_2$, in-line, and nasopharyngeal F_1O_2 readings whilst breathing O_2 using the various masks and mask configuration ratings were analysed using non-parametric tests. Initial analysis was completed using the Friedman Test with *post hoc* paired analyses completed using the Wilcoxon Sign Rank test with Bonferroni correction. For the *post hoc* tests, a corrected P-value of 0.008 (0.05/6) was considered significant.

The primary outcome measure was a comparison of the median $P_{tc}O_2$ measurements recorded across the six

Table 2
Transcutaneous oxygen partial pressures (median and inter-quartile range shown in mmHg) while breathing oxygen using the demand valve with four different mask configurations; * P-values based on the Friedman test

Anatomical site	Baseline (room air)	Intraoral mask with nose clip	Intraoral mask without nose clip	Pocket face mask	Oronasal mask	P-value*
Upper arm	69 (59, 75)	454 (437, 488)	427 (396, 474)	376 (347, 415)	250 (160, 326)	< 0.001
Lower arm	65 (59, 82)	393 (332, 437)	357 (324, 427)	312 (256, 383)	187 (135, 263)	< 0.001
Palm hand	77 (67, 81)	283 (229, 323)	263 (208, 285)	248 (160, 277)	157 (104, 217)	< 0.001
Lateral leg	61 (50, 75)	358 (283, 398)	345 (248, 394)	278 (243, 333)	156 (95, 294)	< 0.001
Lateral ankle	59 (51, 63)	310 (255, 329)	281 (238, 329)	252 (205, 286)	137 (110, 184)	< 0.001
Dorsum foot	64 (55, 78)	199 (175, 237)	188 (124, 228)	156 (132, 219)	123 (67, 137)	< 0.001

Figure 4
Transcutaneous oxygen partial pressures (median in mmHg) while breathing oxygen using the demand valve with four different mask configurations
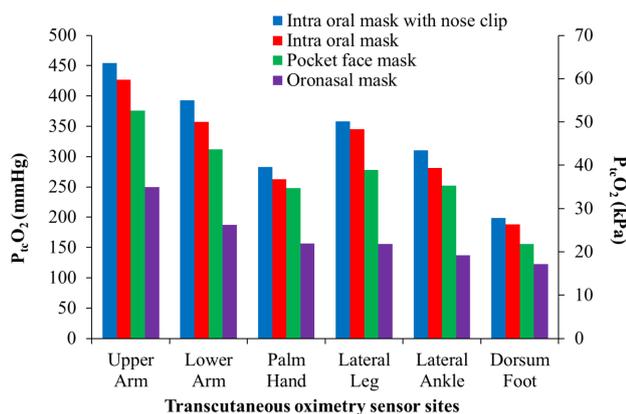
Table 3
Statistically significant differences in transcutaneous oxygen partial pressures *post hoc* comparisons using Wilcoxon Sign Rank test with Bonferroni correction; $P < 0.008$ (0.05/6) considered significant; IOM® – intraoral mask

Post hoc analysis:
IOM® with nose clip ≈ IOM without nose clip
   (all P-values > 0.01)
IOM with nose clip > Pocket face mask
IOM with nose clip > Oronasal mask
IOM without nose clip > Oronasal mask
Pocket face mask > Oronasal mask
   (for all sensor sites)
IOM without nose clip > Pocket face mask
   (for 3 of the 6 sensor sites)
Upper arm $P = 0.003$
Lower arm $P = 0.001$
Lateral leg $P = 0.005$

sensor sites after breathing O_2 for 10 min using each mask configuration. Secondary outcome measures included in-line and nasopharyngeal F_IO_2, $ETCO_2$, mask comfort, ease of breathing and holding of each device.

Results

Sixteen healthy volunteers, 13 female and three male, met all inclusion criteria and completed the study protocol. Their demographic and baseline measures are shown in Table 1.

Figure 4 displays the median $P_{tc}O_2$ readings across all sensor sites and mask configurations. $P_{tc}O_2$ values were statistically different across each mask configuration for each sensor site (Table 2). The IOM® with NC delivered statistically better tissue oxygenation than the pocket face mask and the oronasal mask at all sensor sites and was similar to the oxygenation achieved with the IOM® without NC. Table 3 summarizes the *post hoc* comparison results.

Nasopharyngeal F_IO_2 was highest using the IOM® and similar results achieved with and without the NC. Nasopharyngeal F_IO_2 was lowest when breathing with the oronasal mask (Table 4). One participant's nasopharyngeal F_IO_2 values were lost due to ongoing catheter clogging. $ETCO_2$ was statistically lower when participants breathed O_2 using the oronasal mask (Wilcoxon Signed Rank Test $P < 0.008$), but this almost certainly represents an artefactual reading due to an imperfect mask seal. In-line F_IO_2 did not exceed 97% with any of the mask configurations and was lowest using the oronasal mask (Table 4). Estimated mask assembly dead space is presented in Table 4. Actual individual pocket face and oronasal mask volumes would vary slightly depending on each participant's facial features.

The pocket face mask was rated as most comfortable (Table 5). Ease of breathing rating for each mask is listed in Table 6. Participant ratings for the holding of each mask configuration are presented in Table 7. On *post hoc* analysis no statistical

Table 4
Inspired oxygen and respiratory measurements while breathing oxygen using the demand valve with four different mask configurations and estimated mask assembly dead space (median and inter-quartile range); n/a – not applicable; $ETCO_2$ – end-tidal carbon dioxide; * P-values based on the Friedman test

Parameter	Intraoral mask with nose clip	Intraoral mask without nose clip	Pocket face mask	Oronasal mask	*P-value
In-line O_2	95 (94, 96)	96 (94, 97)	94 (93, 95)	93 (89, 95)	< 0.001
Nasopharyngeal O_2 ($n = 15$)	95 (95, 96)	96 (95, 96)	84 (75, 88)	56 (37, 70)	< 0.001
Intra-mask O_2	n/a	n/a	84 (72, 88)	n/a	
$ETCO_2$ (mmHg)	34 (30, 38)	32 (30, 36)	35 (28, 38)	29 (23, 36)	0.001
Respiratory rate (breaths·min^{-1})	9 (8, 12)	10 (9, 10)	10 (8, 11)	10 (8, 12)	0.945
Mask assembly dead space (ml)	14	14	119	195	n/a

Table 5
Mask comfort rating for each mask configuration (n (%)); * P-value = 0.000, Friedman test

Comfort assessment	Intraoral mask with nose clip	Intraoral mask without nose clip	Pocket face mask	Oronasal mask
Very uncomfortable	2 (12.5)	0	0	3 (18.8)
Uncomfortable	5 (31.3)	3 (18.8)	1 (6.3)	2 (12.5)
Neither	5 (31.3)	4 (25.0)	0	5 (31.3)
Comfortable	4 (25.0)	8 (50.0)	7 (43.8)	5 (31.3)
Very comfortable	0	1 (6.3)	8 (50.0)	1 (6.3)
Median (IQR)*	3.0 (2.0–3.8)	4.0 (3.0–4.0)	4.5 (4.0–5.0)	3.0 (2.0–4.0)

Table 6
Ease of breathing rating for each mask configuration (n(%)); * P-value = 0.020, Friedman test

Breathing assessment	Intraoral mask with nose clip	Intraoral mask without nose clip	Pocket face mask	Oronasal mask
Very difficult	0	0	0	2 (12.5)
Difficult	0	0	1 (6.3)	6 (37.5)
Neither	8 (31.3)	4 (25.0)	3 (18.8)	2 (12.5)
Easy	2 (12.5)	5 (31.3)	5 (31.3)	4 (25.0)
Very easy	6 (37.5)	7 (43.8)	7 (43.8)	2 (12.5)
Median (IQR)*	3.5 (3.0–5.0)	4.0 (3.3–5.0)	4 (3.3–5.0)	2.5 (2.0–4.0)

Table 7
Ease of holding rating for each mask configuration (n (%)); * P-value = 0.015, Friedman test

	Intraoral mask with nose clip	Intraoral mask without nose clip	Pocket face mask	Oronasal mask
Very difficult	0	0	0	1 (6.3)
Difficult	0	0	1 (6.3)	3 (18.8)
Neither	3 (18.8)	3 (18.8)	3 (18.8)	4 (25.0)
Easy	8 (31.3)	7 (43.8)	8 (31.3)	6 (37.5)
Very easy	5 (31.3)	6 (37.5)	4 (25.0)	2 (12.5)
Median (IQR)*	4.0 (4.0–5.0)	4.0 (4.0–5.0)	4 (3.25–4.75)	3.5 (2.25–4.00)

difference was found between each mask configuration for ease of breathing and holding of each mask configuration. The NC was frequently described as uncomfortable. The IOM® was described as easiest to use as it rested in the mouth whereas constant pressure was required to maintain a seal against the face with the two other masks.

Discussion

Oxygen is the primary first-aid treatment for divers suspected of having DCI.[1,2,7] Oxygen has been shown in retrospective reviews to improve symptoms and decrease the subsequent number of hyperbaric treatments required.[4] Of the commercially available O_2 masks for use with a demand valve delivery system designed for diver first aid, our study has shown that the IOM® with NC is the configuration that achieves the highest level of tissue oxygenation and nasopharyngeal F_IO_2.

DAN portable O_2 delivery units can provide a constant flow capability or operate as a pressure-triggered demand valve. The demand valve only delivers O_2 when the diver breathes in and, therefore, allows for conservation of O_2, dependent on the respiratory minute volume of the user. The ease of use, familiarity for divers, potential to deliver high inspired O_2 concentrations,[15] as well as the potential for O_2 supply conservation, has led to the recommendation of the demand valve as the O_2 delivery method of choice in the pre-hospital treatment of DCI.[2]

However, previous research unexpectedly showed that the demand valve with oronasal mask provided less tissue O_2 than a constant flow NRB.[10] $P_{tc}O_2$ readings whilst breathing O_2 via the demand valve with oronasal mask were anticipated to exceed those achieved with NRB at 15 L·min^{-1}.[10] The previous contradictory findings[10] were almost certainly explained by poor fit of the oronasal mask and subsequent entrainment of air. This assumption is supported by the current findings. Not only are the $P_{tc}O_2$ values lowest whilst

breathing O_2 using the oronasal mask, but the in-line and nasopharyngeal F_IO_2 values are also consistent with dilution of the O_2 with entrained air. The demand valve has been promoted to provide near 100% O_2;[2] however, this study highlights the need to assess the complete O_2 delivery system as different mask configurations provide different levels of O_2. It is important to remember that the one-way valve and filter provided for use with the pocket face mask and IOM® must be removed prior to use with the demand valve as leaving them in place leads to entrainment of air.

Oxygen therapy devices have traditionally been referred to as fixed or variable performance devices.[16] When used appropriately, fixed performance devices deliver a constant fraction of O_2 to the patient's airway whereas the fraction delivered by variable performance devices can be affected by factors such as O_2 supply flow rate and the patient's respiratory minute volume. Demand regulators have traditionally been regarded as fixed performance devices based on the assumption that the composition of gas delivered to the patient's airway matches that delivered to the demand valve itself.[16] However, our results show that even a demand regulator can, in fact, behave like a variable device depending on the interface between the valve and the patient. Use of an oronasal mask introduces variability to the behaviour of a demand valve depending on the adequacy of the seal on the patient's face, whereas when used with an IOM and NC the demand regulator likely behaves as a true fixed delivery device.

The IOM® with NC obtained the best $P_{tc}O_2$ results. Previous research comparing demand systems using both an oronasal mask and a mouthpiece with NC found no difference in inspired O_2 and nitrogen washout between the masks.[17] We verified F_IO_2 of the demand system using one-way valves (99%), but these valves were removed for the study. In-line F_IO_2 measured during the study did not reach 99% and was significantly lower when breathing with the oronasal mask (Table 4). These lower in-line F_IO_2 levels are reflective

of gas contamination with exhaled air and possible air entrainment from mask leakage. The differences between the in-line F_IO_2 using the different masks in our study are subtle (Table 4). It is only when examining the delivery of the O_2 to the participants ($P_{tc}O_2$ and nasopharyngeal F_IO_2) that the differences between the masks become obvious. The IOM® out-performs both oronasal masks.

Divers are accustomed to breathing from a demand valve with a mouthpiece. This is reflected in their rating of the different mask configurations. The IOM® was commonly rated as easy to breathe from and easy to hold (Tables 6 and 7). The NC was reported as uncomfortable. The oronasal mask was ranked as the most difficult in terms of breathing ease and holding the device. Two subjects were noted to have difficulty breathing using the oronasal mask. When questioned, they stated that they had to hold the mask tightly and use larger breaths to trigger the demand valve. It is likely that poor mask fit and the large mask dead space (Table 4) contributed to a larger breath being required to adequately trigger the demand valve.

$ETCO_2$ was significantly lower when breathing with the oronasal mask though there were no differences in the respiratory rates while breathing with the different masks (Table 4). Entrained air from a sub-optimally fitting mask diluting the $ETCO_2$ is the most likely explanation and is onsistent with the observed lower in-line F_IO_2 measured with this device.

There was a low number of male participants in this study due to a predominance of facial hair (an exclusion factor) as it was thought it could contribute to mask leak.[18] Previous research showed no significant difference in $P_{tc}O_2$ by sex,[13] but facial size may be a factor in mask fit.

Other investigators have explored closed-circuit O_2 delivery devices, other than demand valves, for the delivery of O_2 in DCI.[19,20] None of these closed-circuit devices, however, are commonly used by recreational divers, partly due to increased complexity and operational requirements.[2,21] Future research should compare continuous flow devices with demand valve and closed-circuit O_2 delivery systems.

LIMITATIONS

P_aO_2 was not measured but, rather, TCOM was used as a non-invasive method of measuring tissue oxygenation.[22] Some DCS symptoms are presumably caused by tissue inert gas bubbles; therefore, a measurement estimating tissue oxygenation seems relevant to a study of first aid O_2 delivery. It could be argued that a higher $P_{tc}O_2$ must inevitably indicate higher values at all levels of the pre-tissue O_2 cascade, and that this, in turn, likely indicates greater drive for tissue inert gas elimination, bubble resolution and oxygenation of hypoxic tissues.[2,5,6] However, although obvious, it is acknowledged that this is speculative as this study did not address the clinical efficacy of these devices in treating DCS.

The nasopharyngeal catheter provided valuable information on the oxygenation provided by each delivery system but may have compromised the seal of both the pocket face and oronasal masks. The catheter was secured to the nares and laid against the face, passing under the edge of the masks. The pocket face mask has an air-filled cushion which can easily mould around irregular facial features. The oronasal mask has a soft but more rigid edge and may have been more affected by the position of the catheter. However, participants who had difficulty sealing the oronasal mask felt air leaks at the apex of the mask at the bridge of the nose, not at the site of the catheter. Indeed, poor mask fit to facial contours and the large dead space likely contribute more to the poor performance of the oronasal mask than any small leak near the catheter.

Nasopharyngeal gas sampling was intermittent but frequent throughout the study with only the peak values recorded. Plotting any variable F_IO_2/$ETCO_2$ values obtained during the 10-minute oxygen breathing period was not possible.[16]

Conclusion

Of the commonly available O_2 delivery systems for use with the demand valve, the IOM® with NC is the device that achieved the highest $P_{tc}O_2$ values at the measured sites. We do not dispute previous findings that an oronasal mask could perform as well as a mouthpiece and noseclip,[17] but this would require that great care be taken to ensure a perfect seal. Our results suggest that this would be very unlikely in field use of oronasal masks by divers. The IOM® is likely the most effective option of those tested. $P_{tc}O_2$ and nasopharyngeal F_IO_2 values were similar between the IOM® with and without NC. Given the reported discomfort of this, the IOM® without NC may be the best option.

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Conflicts of interest

Professors Mitchell and Pollock are members of the Editorial Board of Diving and Hyperbaric Medicine but had no involvement in the peer review or publication decision-making process for this article.

Decompression illness and other injuries in a recreational dive charter operation

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Abstract

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Introduction: Health and safety within the recreational diving industry are poorly described. We aimed to obtain the true prevalence of decompression illness (DCI) and other diving and non-diving injuries, including occupational injuries, in a large recreational diving charter operation.

Methods: A New Zealand recreational diving operator keeps detailed records of diving activity and event/incident reports. We extracted passenger and crew numbers, dive numbers and incident statistics from all boat trips and associated work-related injuries between 01 January 2008 and 31 December 2014. The records of divers referred to the regional hyperbaric unit for suspected DCI were reviewed retrospectively. Using these data the prevalence of DCI and non-diving injuries were calculated.

Results: There were 65,536 person-trips to sea and 57,072 divers undertook 97,144 dives. Fifty-five injury events were documented over seven years, 31 in customers and 24 in staff. Four divers (including one staff member) diagnosed with DCI underwent recompression therapy, giving a prevalence of 0.41 cases requiring recompression per 10,000 dives, or one case per 24,386 dives, whilst five other divers were assessed as not having DCI. There was one cardiac-related fatality. Thirty-five non-diving injuries (mainly lacerations and minor musculoskeletal injuries) were documented in 30 people resulting in 10 consulting a general practitioner and seven presenting to the local regional hospital emergency department.

Conclusions: DCI requiring recompression was relatively rare in this supervised recreational diving operation. Minor non-diving injuries were the most common adverse event. Compared to other adventure sports, the prevalence of injury in recreational scuba diving is low.

Introduction

There are few reliable estimates of the prevalence of decompression illness (DCI) and little attention has been given to other mishaps or incidents that may occur in a typical recreational diving operation. There are a number of explanations for this, but chief among them is the difficulty in simultaneously acquiring an accurate numerator (number of incidents) that can be confidently matched to an accurate denominator (number of participants or dives). Studies reporting prospectively acquired numerators and denominators pertaining to dive injuries seem rare, particularly in datasets large enough to allow calculation of injury prevalence.

One exception in our jurisdiction is maintained by Dive! Tutukaka (D!T); a recreational dive charter operator that primarily runs day-boat trips from Tutukaka (Northland, New Zealand) to the Poor Knights Islands which lie 20 km offshore. This site is an internationally renowned temperate water diving destination (water temperatures range from 14−17°C during winter to 18−25°C during summer). Access to the islands, which are a National Park, is strictly limited to authorised personnel and informal landings are prohibited. The surrounding waters are a 'no-take' Marine Park. Recreational diving activities are conducted by a number of commercial diving companies and many private boats visit the islands. D!T is the busiest recreational diving operator in New Zealand. Diving injuries sustained during its commercial activities have been reported briefly previously.[1] In the present study, a broader spectrum of health and safety issues within the company, including injuries sustained by employees in the course of their duties, is examined.

Methods

This study was approved by the University of Auckland Human Participation Ethics Committee (Reference number

013608). D!T is required by law to maintain detailed records of customer numbers, dive numbers, staff deployment to sea and any incidents. Much of this is achieved through skipper's vessel reports for each period at sea which include records of dive operations and any injury incidents (diving and non-diving) during each day. Every diver is logged in and out of the water by a Dive Master on board the vessel and maximum depth and total dive time are recorded for every dive, as are any incidents during the dive.

For the period 01 January 2008 to 31 December 2014, data were extracted from two Excel-format databases kept by the company:
1. Diver, snorkeller and non-diving passenger and crew numbers, and dive numbers to provide denominators for calculation of the prevalence of injuries;
2. All individual injury or incident reports pertaining both to diving and non-diving-related events involving both customers and staff, to provide numerators for calculation of injury prevalence.

Dive-related incidents/injuries were those occurring during diving (such as barotrauma) or as a direct consequence of diving but manifesting after surfacing (which includes DCI). Non-diving-related incidents/injuries were other events (such as lacerations and musculoskeletal injuries) occurring at any point in the trip or working day (including those shore-based processes of preparing for and arriving back from each trip).

Each individual incident was de-identified and given a case ID number. The month and year were recorded, whilst the only demographic data were the gender and age of the victim. The incident and any resulting injury/injuries were classified as either diving-related or non-diving-related. The specific injuries sustained were recorded as was the nature of any treatment required by the patient. Any free-text description was extracted from the vessel log or company incident report. If the incident was diving-related, then the maximum depths and total times for that person's dive were taken from the Dive Master's dive log for that diving day. Case records of divers referred to the Slark Hyperbaric Unit in Auckland for suspected DCI were reviewed for the nature and number of recompression treatments provided, and the clinical outcomes were noted.

The primary outcome of the study was the prevalence of DCI calculated both as the number of DCI cases per 10,000 dives and as the number of dives per case of DCI. A principal secondary outcome was calculation of the number of non-diving events resulting in injury per 10,000 trip participants (a combined total of customers and staff).

Results

The diving database contained records of 65,536 person-trips (passengers and crew) to the Poor Knights Islands (or to two wreck sites along the adjacent Tutukaka Coast). There were 97,144 scuba dives undertaken by 57,072 divers. Although not formally recorded, the vast bulk of the diving was on open-circuit (OC) scuba air with a small number of OC nitrox provided by a limited number of customers for their own use. D!T did not provide nitrox diving at that time. There would have been only a small handful of mixed-gas OC and rebreather divers.

Fifty-five injury incidents were documented in the injury database over the study period, 31 in customers and 24 in D!T staff. Twenty were diving-related, nine being suspected DCI, and 35 non-diving related. One diver died after surfacing.

All the nine cases of suspected DCI occurred in OC air divers. Four cases of DCI underwent recompression therapy (Table 1) giving a prevalence of 0.41 cases requiring recompression per 10,000 dives, or one case per 24,386 dives. The other five divers were referred for medical evaluation with symptoms thought possibly caused by DCI (Table 1) but did not undergo recompression because their symptoms were attributed to alternative diagnoses.

There were 11 non-DCI diving-related incidents recorded. Apart from the fatality, these included four barotraumas (two middle ear, one inner ear, one sinus); three panic attacks causing termination of the dive; one uncontrolled inverted ascent (caused by accumulation of air in dry-suit legs); one jellyfish sting and one event in which an unfit diver became short of breath and had to terminate the dive.

The fatality involved a 55-year-old diver who was a foreign tourist travelling with a group. He had a history of two myocardial infarctions, coronary stents, was on cardiovascular medication and wore a Medic Alert bracelet, none of which were declared on his waiver form or to the Dive Master of the day. He was obese, requiring an XXL-sized wetsuit. He was a certified diver who claimed to have done over 50 dives (though only about 25 could be verified). He dived with his own buddy but they separated during the dive as the victim signalled he was low on air 20 minutes into the dive. He was seen by his buddy to surface in no apparent distress and start swimming towards the boat but soon after reaching the boat the victim became unconscious. Basic life support was instituted without success. His computer showed that he had reached a maximum depth of 35 metres' sea water and his air cylinder was empty. The cause of death was given by the Coroner as "*a cardiac event while diving*".

There were 35 non-diving related injuries in 30 people, being 17 lacerations; 12 non-fracture musculoskeletal injuries; three fractures; two electrocutions (both in D!T staff) and one traumatic eye injury. Seventeen of these occurred in D!T staff (who were far outnumbered by passengers). As a result of these injuries, 10 people were known to consult a general practitioner, and seven presented to the local hospital

Table 1
Profiles (depth(s) and total dive time(s)), principal symptoms, recompression treatment and outcome for four cases of decompression illness (DCI) and principal symptoms for five divers referred for evaluation but not recompressed; msw – metres' seawater; USN – US Navy; RNZN1A is a 4 atm abs (405 kPa) heliox treatment table

DCI confirmed, recompressed				
	Dive profiles	**Symptoms and signs**	**Treatment**	**Outcome**
Case 1	23 msw / 52 min + 22 msw / 53 min	Rash + headache	USN Table 6	Full recovery
Case 2	9 msw / 37 min + 10 msw / 37 min	Paraesthesias + joint pain	USN Table 6	Full recovery
Case 3	21 msw / 30 min	Numbness / paraesthesias one side face and body	USN Table 6	Full recovery
Case 4	20 msw / 55 min + 19 msw / 55 min	Rash, shoulder & back pain	RNZN1A + 2 follow up recompressions	Full recovery
Not DCI, not recompressed				
Case 5		Severe fatigue		
Case 6		Chest tightness		
Case 7		Nausea and paraesthesiae		
Case 8		Panic attack, paraesthesiae, cramps, hyperventilation		
Case 9		Fatigue, vertigo		

emergency department. These 35 events in 65,536 person-trips represents a prevalence of 5.3 per 10,000 person-trips, or one event per 1,872 person-trips.

Discussion

The prevalence of DCI in recreational scuba diving reported here represents one of very few estimates based on numerator and denominator data collected prospectively in the field. The same D!T database was previously interrogated for a three-year period between 2005 and 2008 (and data from that evaluation overlap the present data by six months).[1] This revealed seven DCI cases in 70,600 dives (a prevalence of one case per 10,000 dives). Another study which also obtained numerator and denominator data from a database prospectively maintained by a single dive operation reported a strikingly similar prevalence of approximately one case per 10,000 dives.[2] A third study reported data prospectively collected as part of the Divers Alert Network Project Dive Exploration initative,[3] giving a prevalence of 3.11 cases per 10,000 dives.

Other approaches have used less precise measures of the denominator. In one study, regional treated DCI cases

provided a numerator, and scuba tank fill numbers over the corresponding region and period served as a denominator.[4] This revealed a notional DCI rate of one case per 10,000 dives (where one tank fill is assumed to equal one dive). Although subject to a number of potential selection, recall and reporting biases another strategy is to use voluntary diver surveys. One such Japanese study estimated a rate of 0.53 cases per 10,000 dives.[5] A survey of Divers Alert Network members reported a similar DCI prevalence of 0.63 per 10,000 dives,[6] and a small survey of mainly experienced divers at several diving symposia reported a rate of 1.83 cases per 10,000 dives.[7] Other studies have derived denominators from various sources with increasing reliance on estimations and assumptions.[8,9] The key elements of the above studies are summarised in Table 2.

It is difficult to reliably interpret any differences in the DCI rates reported between these studies. For example, in the present study we report a rate that is lower than that arising from our earlier analysis,[1] and lower than the rate calculated using similar methodology from another discrete dive operation.[2] It is tempting to conclude that this represents a true improvement or difference in diving safety. D!T is a conservative organisation whose trips are closely supervised

Table 2
Key elements of studies that calculate a prevalence of decompression illness (DCI) in large cohorts of recreational divers

Study	Numerator descriptor	Denominator descriptor	Numerator (DCI cases)	Denominator (dives)	Prevalence (DCI cases per 10,000 dives)
Present study	Prospective field data	Prospective field data	4	97,144	0.41
Davis[1]	Prospective field data	Prospective field data	7	70,600	0.99
Gilliam[2]	Prospective field data	Prospective field data	7	77,680	0.90
Buzzacott[3]	Prospective field data	Prospective field data	38	122,129	3.11
Ladd[4]	Regional clinical data	Regional tank fills	14	146,291	0.96
Nakayama[5]	Retrospective self-report	Retrospective self-report	60	1,140,653	0.53
Ranapurwala[6]	Retrospective self-report	Retrospective self-report	11	174,912	0.63
Klingmann[7]	Retrospective self-report	Retrospective self-report	52	284,067	1.83
Lippmann[8]	Regional clinical data	Informed estimate	188	1,750,000	1.07
Harris[9]	Regional clinical data	Informed estimate	16	57,000	2.81

and incorporate thorough briefings, guided dives and matching of dive sites to diver capabilities. There is evidence from a database of similarly disciplined American scientific dives that attention to safety results in a low prevalence of DCI (33 DCI cases in 1,019,159 dives; approximately 0.3 cases per 10,000 dives).[10]

Both scientific diving in the USA[10] and diving at D!T in NZ are regulated by a wide range of legislation and codes of practice in their respective countries. Whilst the legislative environment in NZ for adventure sports was complex and not well policed during the study period, all employed divers at D!T were required to practice under Department of Labour Health and Safety regulations (ASNZS2299.1:2007[11]). The New Zealand Maritime Authority requires dive vessels to be in survey and skippers to have the appropriate certification levels. Direct observation of D!T by three of the authors (MH, FMD and SJM) suggests a strong 'safety first' work ethic throughout the company. This could explain the similar low prevalence of treated DCI amongst USA science and D!T divers. Also, because of 'no fault' legislation in New Zealand (Accident Compensation Act 2001 No. 49 and the Health and Safety at Work Act 2015), it is likely that non-diving trauma resulting in injury both to customers

and staff was documented accurately and has a relatively low prevalence.

However, there are other factors that may have influenced our measured outcomes. For example, the reference period of the present study corresponds to that over which the findings of the remote DCI workshop in relation to recompression for mild DCI became influential.[12] The workshop's endorsement of treatment without recompression in some cases that met a strict definition of "*mild DCI*" might have influenced decisions by physicians to ascribe alternative diagnoses to the five equivocal cases that were not recompressed (Table 1). Nevertheless, even if these are included in the present analysis as DCI cases, then the calculated rate in our study would be 1:10,000; very similar to several of the other studies. Methodological differences can also account for different results between studies (see above).

What is clear from these various data is that DCI seems relatively uncommon in mainstream recreational scuba diving. This observation segues into consideration of the use to which data of this nature can be put. We would suggest that accurate estimates of the DCI rate derived from large cohorts of divers conducting activity typical of the vast majority of

recreational scuba diving are important for purposes such as actuarial evaluations, healthcare resource planning, informing choices of prospective divers and characterizing the safety of the sport in relation to other activities. For example, in 2018, the Mountain Safety Council of New Zealand reported 5,504 tramping injuries in more than 1.5 million trampers; that is, one in 279 trampers needed medical care.[13] Recreational scuba diving in northern New Zealand, by comparison, would appear to be a relatively injury-free adventure activity. The requirement for operators like D!T to maintain accurate recording of diving activity and related incidents that is enshrined in health and safety legislation and adventure sports standards seems justified for this purpose alone.

It needs to be clearly understood, however, that the risk of DCI for an individual diver or dive is extremely context-sensitive and may not conform to 'population estimates'. Even if it is assumed that there are no untoward events on a dive, there are other factors that may significantly alter risk such as the presence of a large persistent foramen ovale (PFO).[14] Water temperature may have a profound influence on DCI risk. For example, in the Project Dive Exploration study, a separate series of 6,527 dives in cold water resulted in a reported DCI rate of 28 per 10,000 dives.[15] The explicit collection and reporting of such dives in Project Dive Exploration will partly explain the increased overall prevalence of DCI reported from that database (Table 2).[3] Some sub-types of 'recreational' scuba diving, such as deeper decompression dives conducted by 'technical' divers may carry a substantially higher risk.

Diving incidents other than DCI and non-diving incidents/injuries were both more common in the present study than DCI. It is much more difficult to benchmark the prevalence of relatively minor non-diving injuries because of a lack of comparable studies. However, it is clear that such injuries accrued in the operation of boats are recognised as important and qualitatively similar to those reported here.[16]

LIMITATIONS

Our data likely under-estimate the true prevalence of mild DCI due to under-reporting. It is well recognised that minor symptoms of DCI are often unappreciated or even concealed by divers. Nevertheless, the number of divers recompressed for DCI is a hard numerator, and any cases not reported (or recompressed) were almost certainly mild. Therefore, our DCI data can be considered a valid indicator of the rate of clinically significant DCI.

In relation to our other injury data, it is widely recognised in medical research that the published prevalence of such events is often strongly influenced by how those outcomes are defined and measured. For example, only two symptomatic middle ear barotraumas were recorded over the seven-year period of the present study, yet a recent study involving prospective examination by expert observers demonstrated middle ear barotrauma in 48 of 67 open-water course trainees.[17] Middle ear barotrauma likely occurred much more often than recorded in our study; in fact, it is sufficiently common that both divers and skippers might not consider it worthy of reporting, unless severe. In a similar vein, the more common (and minor) an event, the less likely it is to be meticulously recorded even if reported to the crew. Therefore, incomplete recording is another potential cause for underestimation of the rate of common minor events. Nevertheless, because of the no-fault injury compensation system in NZ, the recording of non-diving injury requiring either first aid or further medical attention is likely to be fairly reliable.

We must acknowledge the fact that some non-DCI diving-related injuries or complications may not have become apparent for up to days after diving. For example, a recent study that included evaluation of post-diving presentations to American emergency departments suggested that otitis and other infections that could be expected to develop slowly accounted for approximately 16% of consultations.[18] These will not be accounted for in our data.

Finally, this is a single-centre study and the extent to which the DCI prevalence estimate can be generalised across the dive charter industry or recreational diving in general is uncertain. However, as discussed above there is reasonable agreement with estimates from other settings, which is encouraging.

Conclusions

Recreational diving in this temperate water, off-shore environment had a remarkably good safety record given that all levels of diving experience were being catered for. Diving-related injuries were generally minor and uncommon. Staff members appear to have been at more risk of injury than customers. Care in a marine environment needs to be stressed at all times.

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Conflicts of interest

Kate Malcolm is the General Manager of Dive!Tutukaka. Simon Mitchell and Michael Davis are editors of this journal but the peer review process and decision regarding publication were managed entirely by the European Editor, Lesley Blogg.

Fatal shark attacks on divers in Australia, 1960–2017

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Abstract

(Lippmann J. Fatal shark attacks on divers in Australia, 1960–2017. Diving and Hyperbaric Medicine. 2018 December 24;48(4):224–228. doi: 10.28920/dhm48.4.224-228. PMID: 30517954.)
Aim: The aim of this study was to identify the number, location and characteristics associated with fatal shark attacks on divers in Australian waters from 1960 to 2017, inclusive.
Methods: Searches were made of the Divers Alert Network Asia-Pacific Diving Mortality Database; the International Shark Attack File website; the Australian Shark Attack File and the Global Shark Attack File to identify cases of fatal shark attacks on divers in Australia. In addition, a systematic search of published medical and sporting literature was conducted to identify relevant reports. The data collected were scrutinised for relevance and duplication.
Results: There were 187 recorded attacks on divers, comprising 112 snorkellers, 62 scuba divers and 13 on divers using surface-supplied breathing apparatus. These included 28 verified deaths: 13 involving snorkellers, eight involving scuba divers, and seven divers using surface-supply. The victims' ages ranged from 13–50 years (mean 31 years). All but three were males. The vast majority of attacks were by *Carcharodon carcharias* (Great White Shark).
Conclusion: Spearfishing and other seafood collection, as well as diving near fishing activities and/or seals, were identified as major risk factors. However, shark attacks on divers are relatively rare and represent only 3% of diving-related fatalities in Australia over this 57-year period.

Introduction

When humans partake in activities in or on the sea, it is inevitable that some of these result in interactions with sharks. However, there is a huge imbalance in comparative mortality from these shark-human interactions. It has been estimated that between 63 and 273 million sharks are killed by humans each year.[1] On the other hand, according to the International Shark Attack file (ISAF), there was an average of 77 reported shark attacks on humans annually from 2007 to 2016. These included 61 fatalities, an average of six deaths per year worldwide.[2]

The ISAF defines unprovoked attacks as incidents where an attack on a live human occurs in the shark's natural habitat with no human provocation of the shark. Provoked attacks occur when a human initiates contact with a shark and include situations where a diver is bitten after grabbing a shark, feeding a shark, attacks while spearfishing, unhooking a shark from a line or removing one from a net. The ISAF data indicate that there were 238 unprovoked attacks on divers (i.e., snorkellers, scuba divers and divers using surface supplied breathing apparatus) worldwide from 1960 to 2015. It does not publish the number of provoked attacks.[2]

The aim of this study was to determine the number, location and characteristics of fatal shark attacks (provoked or unprovoked) on divers in Australia from 1960 to 2017 inclusive.

Methodology

Searches were made of a variety of databases which were likely to include relevant data on Australian shark attacks. These were:
- the Divers Alert Network Asia-Pacific (DAN AP) Diving Mortality Database which incorporates data from Project Stickybeak from 1965 to 2000, and subsequent data collected by DAN AP;[3]
- the International Shark Attack file (ISAF) website;[2]
- the Australian Shark Attack File (ASAF)[4] (additional data was also provided by the ASAF);
- the Global Shark Attack File (GSAF).[5]

In addition, a systematic search of published literature for all dates up to and including July 2018 was conducted using Medline Complete, CINAHL Complete, Health Source (Nursing/Academic edition) and SPORTDiscus with Full Text. The search terms were: scuba or "*compressed air*" or "*compressed gas*" or snorkel* or div* AND shark AND death* or fatalit* or mortalit* AND Australia. The data collected were scrutinised for relevance and duplication and compiled into a single list of diving-related fatalities from shark attacks.

Figure 1

Time of day of 28 fatal shark attacks on divers compared with 138 general shark attacks incidents, expressed as percentages[5]
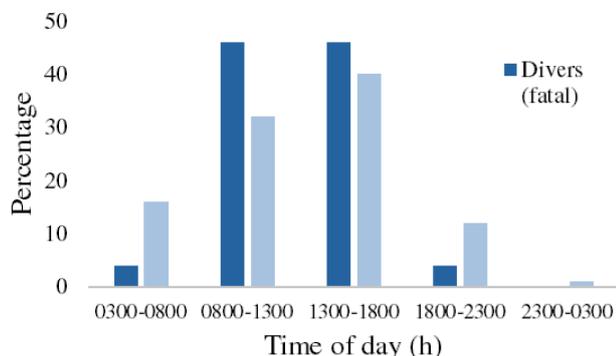
Figure 2

Frequency of fatal shark attacks on divers by decade, 1960–2017 (final number is for only eight years)
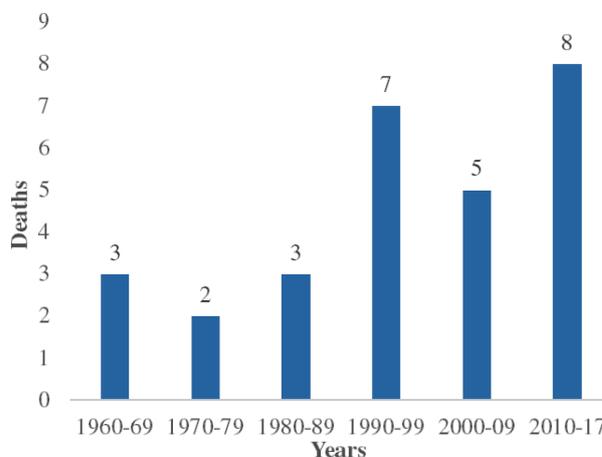
Results

The data revealed a total of 562 recorded shark attacks in Australia from 1960 to 2017, inclusive. One hundred and eighty-seven of these were attacks on divers, comprising 112 snorkellers, 62 scuba divers and 13 on divers using surface-supplied breathing apparatus (SSBA). These included 28 verified deaths: 13 involving snorkellers, eight involving scuba divers, and seven divers using SSBA (Table 1). The number of reported shark attacks on both divers and non-divers in Australia increased from 6.5 per year in 1990–2000, to 15 per year from 2001–10, with an average of 11 deaths per decade over this 20-year period.[6] As shown in Figure 1, shark attack-related deaths in divers have risen from three per decade in the 1960s, to eight over the last seven years (Figure 1). The mean (SD) age of the fatality victims was 31 (11) years (range 13–50 y). Only three were females, two being scuba divers. Nineteen of these deaths occurred in South Australia (10) and Western Australia (nine), with the remaining nine spread across Tasmania (four), Queensland (three), New South Wales (two) and none in the Northern Territory and Victoria.

The attacking sharks were sighted or otherwise positively identified in 21 (75%) of the fatal incidents. The species identified were *Carcharodon carcharias* (Great White Shark, *n* = 18)*, Galeocerdo cuvier* (Tiger Shark, *n* = 2) and Carcharhinus leucas (Bull Shark, *n* = 1). A Great White Shark was implicated as the very probable attacker in another three incidents on the basis of location, size and/or bite marks.

Twenty-one of the 28 divers killed were involved in seafood collection; predominantly spearfishing (*n* = 9), abalone collection (*n* = 5) and scallop collection (*n* = 4). Six attacks occurred near seal colonies, and at least three others occurred in areas with nearby fishing.

Although shark attacks can occur at any time,[6] all of the fatal attacks on divers occurred in daylight with most incidents in early to mid-afternoon (Figure 2). There was no obvious pattern in the month of the year, or sea and weather conditions. There were spikes in June and December but the numbers are too small for meaningful interpretation; for each of the four quarters of the year there were eight, six, seven and seven deaths, respectively. The bodies of 11 of the victims were never found, although in all of these, the attack was witnessed.

Of the 187 recorded attacks on divers, most of the 28 deaths involved males who were spearing fish or otherwise collecting seafood. The vast majority of the fatal attacks occurred in temperate waters, and between 0800 h and 1800 h, reflective of when most diving is conducted. In the vast majority of incidents, the attacking shark was identified as a *C. carcharias*.

Discussion

SPEAR FISHING AND SEAFOOD COLLECTION

In the Australian shark attack database, a shark attack on a diver while spearfishing and other seafood collecting while diving is categorised as a 'provoked attack', as the diver is impacting the shark's behaviour. There is no doubt that such activities increase the likelihood of a diver being attacked, evidenced by the fact that over three quarters of the victims were involved in such activities. Diving near to where fishing is being conducted increases the risk, especially if burley is being used, as this will attract and excite nearby sharks. One spearfisherman was wearing a bait pouch attached to his weight belt, a practice that is obviously highly provocative and strongly discouraged.

DIVING NEAR SEALS

Seals are a regular food source for large sharks, especially when seal pups are present, so diving near seal colonies is

Table 1
Characteristics of 28 fatal shark attacks on divers in Australia; * distant; NR – not reported; US – unsighted; UK – unknown; GWS – Great White Shark;
US-GWS – unsighted but probably GWS; SC – scuba; SN – snorkel; SS – surface supply

Year	Mode	Month	Time	State	Gender	Age	Activity	Buddy	Shark	Contributing factors?	Conditions
1960	SN	Jan	1530	NSW	M	13	Spearfishing	NR	Bull	Spearing	Calm, poor visibility
1962	SN	Dec	1430	SA	M	16	Spearfishing	NR	GWS	Spearing	Poor visibility (4 m)
1967	SN	Aug	1100	WA	M	24	Spearfishing	Yes	GWS	Spearing; seal colony	NR
1974	SS	Jan	pm	SA	M	26	Abalone	No	GWS	Seals nearby; seafood	NR
1975	SS	Jul	1300	TAS	M	37	Abalone	No	US-GWS	Seals nearby; seafood	Calm, dark water
1982	SN	Feb	NR	TAS	M	32	Spearfishing	No	GWS	Spearing; cleaning fish	Calm
1985	SN	Mar	1230	SA	F	33	Scallops	Yes	GWS	Scallops; offal in area	Calm, clear
1987	SC	Sep	am	SA	M	47	Scallops	No	US-GWS	Fish, dolphins; scallops	Moderate smooth sea
1990	SN	Apr	NR	QLD	M	37	Trocus	No	US	Unknown	NR
1991	SC	Sep	1500	SA	M	19	Sightseeing	Yes	GWS	Unknown	Poor visibility (3 m)
1993	SC	Jun	0930	NSW	M	31	Sightseeing	Yes	GWS	Unknown; fish running	Calm, clear (10 m)
1993	SC	Jun	1055	TAS	F	34	Sightseeing	Yes	GWS	Seal colony; abalone	Calm, clear
1993	SS	Nov	1515	WA	M	28	Pearl farm	Yes*	Tiger	Pearls	Calm, no current, poor visibility
1995	SS	Sep	1500	WA	M	29	Abalone	No	GWS	Abalone; seals; whale calves	Rough, poor visibility
1998	SN	Jun	1400	SA	M	26	Abalone	Yes	GWS	Seal colony; abalone	NR
2002	SS	Apr	1240	SA	M	23	Scallops	Yes	GWS	Scallops	Clear
2004	SN	Dec	1300	QLD	M	38	Spearfishing	No	US	Spearing; wearing bait pouch	NR
2005	SN	Mar	1400	WA	M	26	Sightseeing	Yes	GWS	Fishing nearby?	NR
2005	SC	Aug	1610	SA	M	23	Cuttlefish eggs	Yes	GWS	Cuttlefish eggs; burley nearby	NR

Table 1 continued

Year	Mode	Month	Time	State	Gender	Age	Activity	Buddy	Shark	Contributing factors?	Conditions
2008	SN	Dec	0700	WA	M	51	Crabs	Yes	GWS	Crab collecting?	Calm, clear
2011	SS	Feb	1820	SA	M	50	Abalone	No	NR	Abalone	NR
2011	SC	Oct	1325	WA	M	32	Spearfishing	No	GWS	Spearing	NR
2012	SC	Mar	0930	WA	M	33	Sightseeing	Yes	GWS	Nearby fishing?	Choppy, poor visibility
2014	SN	Feb	1200	SA	M	28	Spearfishing	Yes	GWS	Spearing	Choppy, clear
2014	SN	Dec	am	QLD	M	18	Spearfishing	No	US	Spearing	Calm, clear
2014	SN	Dec	1130	WA	M	17	Spearfishing	Yes	Tiger	Spearing, baiting	NR
2015	SS	Jul	1000	TAS	M	46	Scallops	No	GWS	Scalloping, fishing nearby	NR
2016	SC	Jun	1130	WA	F	50	Sightseeing	Yes	GWS	Fishing nearby?	NR

more risky (There is an old adage that "*a diver is the slowest seal*"). The Australian unprovoked shark attack data indicate that "*swimming, surfing or diving near seals or seal colonies has the highest rate of severe injury and fatalities*".[6] The provocation of hunting seafood near seals would add to this inherent risk.

TEMPORAL AND LOCATIONAL PATTERNS AND SHARK SPECIES

The fatal attacks in temperate waters were associated with *C. carcharias*. Attacks by Great White Sharks in more tropical waters are rare, and most fatal attacks in warmer waters involve *G. cuvier*.[6] Although a broad variety of shark species are known to attack humans, from 1990 to 2010, only three species of sharks were identified as responsible for fatalities during general aquatic activities in Australia – *C. carcharias*, *G. cuvier* and *C. leucas*.[6] This is consistent with these data on diving-related fatalities.

BUDDY SITUATION

The presence or absence of a buddy did not appear to influence the likelihood or the ultimate outcome of these severe and fatal attacks. As shown in Table 1, more than half of the fatality victims were with a buddy at the time of the attack. However, it is very likely that the presence of a buddy has enhanced the rescue of some victims of non-fatal attacks. All divers are encouraged to undergo training in first aid, including the management of severe bleeding. The use of a tourniquet in the event of amputation has recently been re-introduced in first aid guidelines.[7]

RISK

The main reason for the increase in fatal shark attacks in recent decades appears to be the rise in population and greater participation in aquatic activities in general, including in more isolated locations.[6] It undoubtedly also reflects the increase in diving-related activity since 1960, and a more recent resurgence in spearfishing as a recreation. However, it has also been argued that the rise in attacks by *C. carcharias* along some Australian coastlines may be associated with the whale migration season and an increase in the numbers of migrating whales.[8,9]

It is almost impossible to determine the absolute risk of being attacked by a shark while diving owing to the difficulty of determining an accurate denominator for the number of dives. One report for Western Australia estimated the risk of a fatal shark attack while diving using scuba or SSBA to be less than one in three million dives.[10] Australian divers have been estimated to have conducted an average of 1.55 million scuba dives each year from 2001–2010, inclusive and there was only one fatal shark attack on a scuba diver during this period.[11] Whatever the actual risk may be, it is extremely small and far lower than what many in the community

perceive it to be. Current DAN AP diving fatality data reveal that death from shark attack represents only approximately 3% (28/926) of recorded Australian diving-related fatalities from 1960 to 2017 inclusive.[3] In many places, divers seek out and enjoy their interactions with a variety of sharks, the vast majority of which are uneventful. However, even small sharks can sometimes be dangerous when provoked and divers should never be complacent about a shark's presence.

LIMITATIONS

It is possible that some shark attack-related fatalities in divers were not recorded in the available databases and so not included here. In addition, two cases that were recorded in one of the databases as diving-related were excluded due to insufficient available evidence to confirm that the victims were in fact diving.

Conclusion

Shark attacks on divers are relatively rare, representing approximately 3% of diving-related fatalities in Australia over more than half a century. Most of the victims were relatively young males who were attacked by Great White Sharks. Spearfishing and other seafood collection, as well as diving near fishing activities and/or seals, were identified as major risk factors. Therefore, to reduce risk, divers should avoid collecting seafood in areas known to be frequented by large sharks, avoid diving near seal colonies or where there is fishing activity, especially if burley is likely being used to attract fish.

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Assessment of hyperbaric patients at risk of malnutrition using the Malnutrition Screening Tool – a pilot study

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Abstract

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Background: Nutritional assessment and support is essential for wound management. The hyperbaric oxygen clinic is a unique outpatient service where chronically unwell patients present daily for hyperbaric oxygen treatment (HBOT) over several weeks, allowing time for effective nutritional intervention. This is the first study to examine the prevalence of those at risk of malnutrition in a cohort of hyperbaric medical patients.

Methods: A prospective study was undertaken over six months. Following consent, 39 enrolled patients had the Malnutrition Screening Tool and Baseline Characteristic Collection Form completed. Those at risk of malnutrition were given an option to be assessed by a dietitian to complete a Subjective Global Assessment (SGA). At the completion of treatment, the patients completed a questionnaire.

Results: Twelve of the 39 patients screened were at risk of malnutrition using our screening process. Of these, all the patients with available SGA results were diagnosed with moderate to severe malnutrition. Patients receiving HBOT for non-healing wounds and osteoradionecrosis were most at risk of malnutrition.

Conclusion: The prevalence of patients being at risk of malnutrition in our hyperbaric medical service was about one in three. Malnutrition screening should be part of routine patient assessment in order to ensure patients receive timely nutritional intervention. This may improve wound healing.

Introduction

Good nutrition plays a major role in wound prevention and healing.[1,2] International advisory bodies incorporate nutritional assessment and support as standard for wound care prevention and management.[1,3–5] The National Institute for Health and Care Excellence (NICE) recommend malnutrition screening for all hospital inpatients on admission, and all outpatients at their first clinic appointment.[1] In the acute care setting, routine malnutrition screening is supported by level II evidence, with a National Health and Medical Research Council (NHMRC) Grade B recommendation.[6] Most Australian hospitals have implemented malnutrition screening as part of routine nursing workflow for inpatients on admission. This same standard is not applied in the outpatient setting.

There is level I evidence for a high prevalence of malnutrition in the community, which is under-recognised and associated with adverse clinical outcome and cost.[6] Presently, there is only level IV evidence supporting malnutrition screening in the community.[6] This may reflect the fact that outpatient visits are relatively infrequent encounters. Hyperbaric oxygen treatment (HBOT) requires patients to present daily for treatment over a typical period of four to eight weeks. This period provides hyperbaric physicians with an opportunity to identify malnourished patients and provide appropriate nutritional intervention in tandem with HBOT for wound management.

Many hyperbaric patients have chronic non-healing wounds, such as diabetic foot ulcers (DFU), osteoradionecrosis (ORN) or are at risk of ORN following radiotherapy for head and neck cancers.[7] ORN patients often have significant dental pain, reduced oral intake, weight loss and low body mass index (BMI) – all risk factors for malnutrition.[1,6,8] Malnourished patients have a two to three times increased risk of postoperative complications including infections, prolonged hospital stay, delayed recovery and increased mortality.[1,9–12] Because nutritional support should start seven to 10 days prior to surgery,[9] a preoperative course of HBOT presents an opportunity to establish this.

Figure 1
Study profile
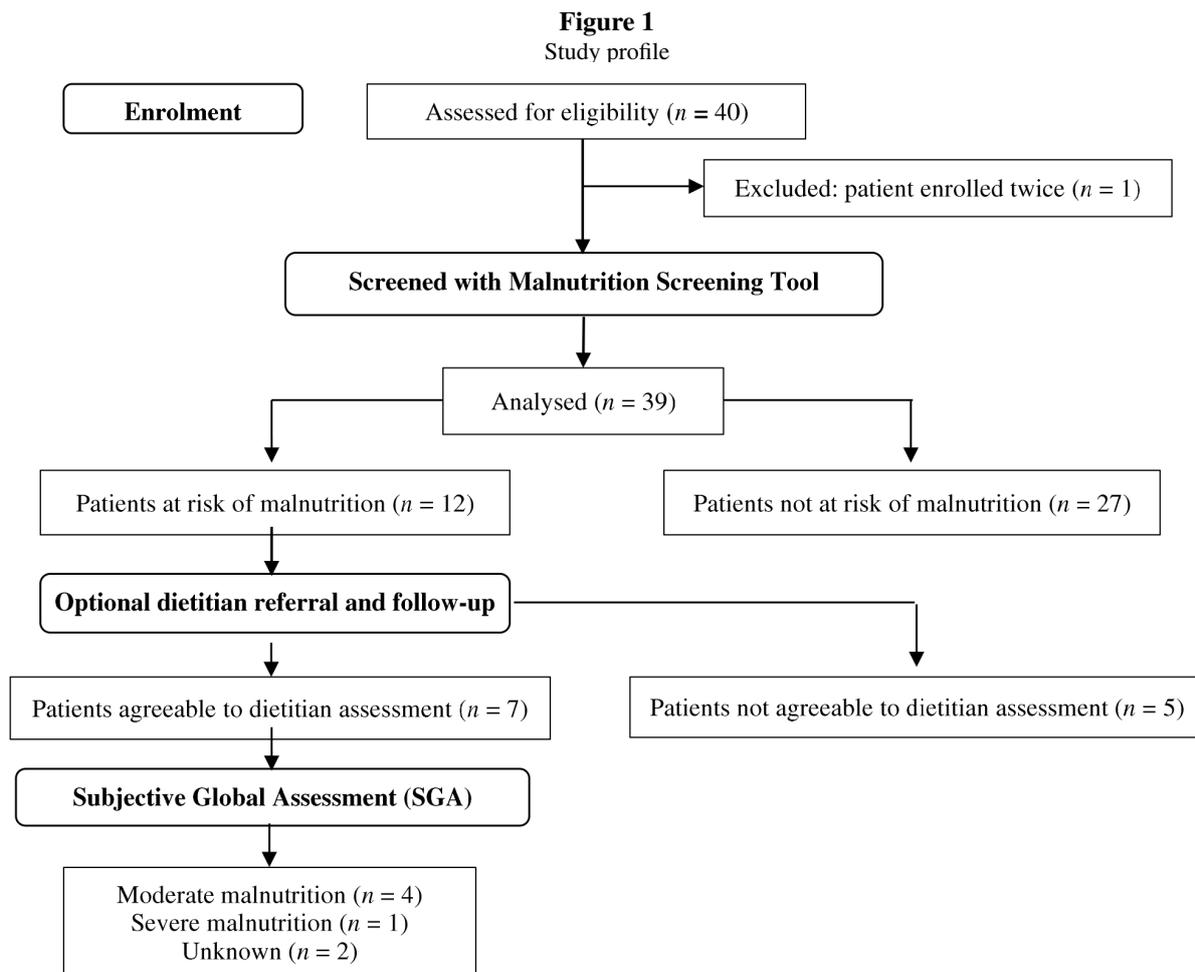
The primary objective of this study was to determine the prevalence of patients at risk of malnutrition in a cohort of patients treated at the Department of Diving and Hyperbaric Medicine (DDHM) at the Prince of Wales Hospital (POWH), Sydney, Australia. The secondary objective was to identify the category of hyperbaric patients most at risk of malnutrition by comparing the primary indication and baseline characteristics of all recruited patients to those in the at-risk-of-malnutrition group.

Methods

Following local ethics approval (HREC Ref no: 15/286 LNR/15/POWH/564), we conducted a prospective, single centre cohort study over the six months from 07 December 2015 to 31 May 2016. Patients of all ages who received more than five treatment sessions of either wound care and/or HBOT were included. The requirement for five sessions was an arbitrary threshold to ensure the recruitment of patients committed to attend the DDHM for treatment. This represents the hyperbaric patient population amenable to continuous nutritional intervention. Patients were excluded if the primary indication for HBOT was diving-related decompression illness, an acute medical emergency such

as necrotising fasciitis, gas gangrene or carbon monoxide poisoning or HBOT starting in the immediate post-operative period following limb or flap revascularisation. Typical treatment courses for these patients are much shorter (about one week).

Eligible participants were identified on arrival for assessment by the DDHM staff and written informed consent was obtained before enrolment. The investigator then completed the Malnutrition Screening Tool (MST) (Appendix A) and the Baseline Characteristic Collection Form (Appendix B). Patients with an MST score of two or more were deemed at risk of malnutrition and were given the option to undergo formal dietitian review within the following week. Patients were rated A to C using Subjective Global Assessment (SGA).[13] Malnourished patients (SGA-B and C) received continuous nutritional intervention. On completion of treatment, we conducted a follow-up questionnaire (Appendix C).

In the absence of existing data to perform sample size calculations, we planned to recruit a convenience sample of 30 patients. We planned to perform a descriptive analysis and to use a chi-squared analysis to compare the prevalence

Table 1
The primary indication for hyperbaric oxygen treatment and the baseline characteristics of the at-risk-of-malnutrition group compared to all recruited patients

Baseline characteristic	All subjects	At risk of malnutrition
Total number of patients	39	12
Female/male ratio	15:24	7:5
Mean age, years (standard deviation)	66.5 (13.7)	72.5 (9.3)
Indication for HBOT		
Non-healing wound without HBOT	10	1
Non-healing wound with HBOT	7	4
Prevention/treatment of osteoradionecrosis	14	5
Other soft tissue radiation injury	6	1
Sudden sensorineural hearing loss	1	0
Other (e.g., sternum osteoradionecrosis)	1	1
Body mass index (BMI) kg·m^{-2}		
Underweight < 18.5 kg·m^{-2}	4	4
Normal weight 18.5–24.9 kg·m^{-2}	13	5
Overweight 25–29.9 kg·m^{-2}	14	2
Obese > 30 kg·m^{-2}	8	1
Diabetes	5	1
Chronic renal impairment	2	0
Chronic gastrointestinal disease	1	0
Smoker or quit less than 3 months	3	1
Alcohol > 2 standard drinks/day	9	5
Pain associated with eating	9	5
Dysphagia	9	4
Pre-existing nausea and vomiting	3	1
Malabsorption suspected	3	1
Current dietitian supported	8	2
Prescribed nutritional supplement	6	3

of high malnutrition scores between diagnostic groups. All analyses were made using the StatsDirect analytical package (StatsDirect Ltd, Cambridge).

Results

The study profile is presented in Figure 1. Thirty-nine patients were enrolled during the study period, of whom 12 were identified to be at risk of malnutrition. Seven of these 12 patients opted for referral to a dietitian. Four patients were diagnosed with moderate malnutrition (SGA-B) and one patient had severe malnutrition (SGA-C). The other

two patients' SGA records could not be obtained. Overall therefore, all five patients with available SGA results were diagnosed with moderate to severe malnutrition. Of the other five who declined referral to a dietitian, one was already seeing a dietitian, one was taking a nutritional supplement and one subsequently saw an external dietitian. Two patients declined without giving any reason.

The primary indication for HBOT and the baseline characteristics of the at-risk-of-malnutrition group compared to all recruited patients are presented in Table 1. Four of seven patients receiving HBOT for a non-healing wound and

five of 14 with/or at risk of ORN were assessed as at risk of malnutrition. Compared to the remainder of the cohort, these two groups of patients were at increased risk, but the difference was not statistically significant (9 of 21 versus 3 of 18; risk difference 26%, Chi^2 = 3.1, P = 0.08). No further meaningful statistical analysis was possible due to the small numbers for comparison.

Five of nine patients who reported having more than two standard drinks per day or had pain associated with eating and four of nine patients with dysphagia were identified to be at risk of malnutrition. Patients who were already consulting a dietitian (two of eight individuals) or taking prescribed nutritional supplements (three of six individuals) were still assessed to be at risk of malnutrition. All four patients who were underweight according to body mass index were found to be at risk of malnutrition. In the follow-up questionnaire, 17 of 39 thought the option of seeing a dietitian during their course of HBOT would be beneficial.

Discussion

HBOT facilities deal with an outpatient population that presents daily to the centre for up to eight weeks. We found over a six-month period that almost one third of the recruited patients were at risk of malnutrition using an accurate and validated tool, the MST.[6,8,12–15] All patients with available SGA results were diagnosed as having moderate to severe malnutrition. This presents an opportunity to provide meaningful nutritional interventions together with HBOT to promote wound healing and reduce post-operative complications.

The overall prevalence of being at risk of malnutrition in our study was higher than the estimated prevalence in the outpatient clinics and community setting.[11,16,17] Data from various outpatient clinics suggest an estimated prevalence of 16 to 21% of patients at risk of malnutrition,[16] while one Australian study involving 1,145 individuals requiring care at home and using the same tools as in our study, showed 15% to be at risk of malnutrition.[17] However, as well as being only a small sample, ours was a highly selected population many of whom had chronic non-healing wounds. The inclusion of only those who had already received five or more treatment sessions into our study might also have skewed our findings.

Mild and/or moderate malnourishment may be hard to identify by untrained healthcare staff. The MST is a widely utilized and validated screening questionnaire for patients at risk for malnutrition[8,13] and has high reliability with 93% sensitivity and 93% specificity in both inpatient and outpatient settings.[6,13–15] It is easily scored by both health care workers and patients with a high inter-rater reliability (93–97%).[15] Any patient who scores two or more on the MST is deemed to be at risk of malnutrition. Using this tool, a patient who lost between 0.5 to 5 kg unintentionally and

ate poorly due to loss of appetite would achieve a score of 2 (Appendix A).

Trained dietitian are a limited resource, and the diagnosis of malnutrition requires lengthy and detailed nutritional assessment.[6] The MST is used to minimise unnecessary dietitian referrals. Our study suggests the patients undergoing HBOT most at risk are those with non-healing wounds or with/or at risk of ORN, those who are underweight, have more than two standards drinks of alcohol regularly and swallowing with difficulty and/or pain.

Our study has a number of limitations. Five of the 12 patients at risk of malnutrition declined further nutritional assessment and two patients' SGA results could not be obtained, indicating the prevalence of malnutrition might be higher than estimated. Further, we were unable to secure routine dietitian referral in the 'at risk' group and could only offer this support on specific request. The high proportion of those at risk who declined this opportunity was of interest to us and deserves further investigation. Any future investigation will need to be formally planned in conjunction with our dietitian service.

The MST was easily administered and the cost of such screening is negligible. The failure to look for malnutrition when it is so simple to do so is not, in our view, best practice.[18] Any influence on actual clinical outcome remains to be evaluated in future studies.

Conclusion

This pilot study suggests that among patients presenting to a hyperbaric facility, the prevalence of being at risk of malnutrition is high and justifies the screening of these patients in order to identify those who require intervention. Further investigation is required urgently to better define the potential positive impact of screening and nutritional intervention on outcomes from HBOT.

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Footnote: Appendices A, B and C are presented on the next page.

Appendix A
Malnutrition Screening Tool (MST)

Name:
Date of Birth:
Study assigned number:
Please circle scores and add for a total score

A. Has the patient lost weight recently without trying?
　　Yes　Go to question B
　　No　Go to question C
　　Unsure　　　Score 2 and go to question C
B. How much weight has the patient lost?
　　0.5–5.0 kg　　Score 1
　　5.0–10.0 kg　　Score 2
　　10.0–15.0 kg　Score 3
　　> 15.0 kg　　Score 4
　　Unsure　　Score 2
C. Has the patient been eating poorly because of a decreased appetite?
　　No　Score 0
　　Yes　Score 1
　　Nutritional score:

If the patient's score is 2 or more please refer them to the Dietitian
Date referred:
Signature:
Referrer's name:

Appendix C
Follow-up questionnaire

Please circle or tick the relevant answer
Study assigned number:
Date of data collection:

1. Are you aware that nutrition is important for wound prevention and/or healing before the study?
　　　Yes / No
2. If you were referred to a dietitian, did you go for consult?
　　　Yes / No
If Yes, did you find it beneficial?:
　　　Yes / No
If No, state reason:
3. Is the option of seeing a dietitian during your treatment beneficial?
　　　Yes / No
If No, state reason:

Appendix B
Baseline characteristic collection form; HBOT – hyperbaric oxygen treatment; MST – malnutrition screening tool; NHW – non-healing wound; PORN – prevention of osteoradionecrosis; ORN – treatment of osteoradionecrosis; PEG – percutaneous endoscopic gastrostomy tube; STRI – soft-tissue radiation injury

Please circle or tick the relevant answer
Study assigned number:
MST score:
Date of data collection:

Primary indication for HBOT
　　NHW without HBOT
　　NHW with HBOT
　　Head and neck STRI (including PORN/ORN)
　　STRI
　　　　Radiation enteritis
　　　　Radiation cystitis and proctitis
　　　　Radiation cystitis only
　　　　Other STRI
　　　　Refractory osteomyelitis / intracranial abscesses
　　　　Sudden sensorineural hearing loss
　　　　Other:
Age:
Sex:　　　Male / Female
Diabetic:　　　　　　　　　　Yes / No
Chronic renal impairment:　　　　Yes / No / on dialysis
Chronic gastrointestinal disease:　Yes / No
(excluding gastric reflux)
Smoker or quit less than 3 months: Yes / No
Alcohol standard drinks per day:　< 2 / > 2
Pre-existing PEG tube:　　　　Yes / No
Pain associated with eating:　　　Yes / No
Difficulty in swallowing:　　　　Yes / No
Pre-existing nausea and vomiting:　Yes / No
Malabsorption suspected:　　　　Yes / No
Current dietitian support:　　　　Yes / No
Prescribed nutritional supplement:　Yes / No
Body mass index (kg·m^{-2}):
　　　　Underweight (< 18.5)
　　　　Normal Weight (18.5 – 25)
　　　　Overweight (25 – 30)
　　　　Obese (> 30)

Preventive effects of ketone ester BD-AcAc$_2$ on central nervous system oxygen toxicity and concomitant acute lung injury

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Key words

Hyperbaric oxygen; Hyperoxia; Injuries; Respiratory; Animal model; Pharmacology

Abstract

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Background: Recent studies indicated that ketone ester R,S-1,3-butanediol acetoacetate diester (BD-AcAc$_2$) may be effective in preventing central nervous system oxygen toxicity (CNS-OT) and concomitant acute lung injury, a serious medical problem to be faced when breathing hyperbaric oxygen (HBO). This study aimed to further investigate the protective effects of BD-AcAc$_2$ against CNS-OT and concomitant acute lung injury (ALI) in mice.

Methods: Mice were treated with BD-AcAc$_2$ in peanut oil vehicle (2.5, 5.0 or 10.0 g·kg^{-1} body weight) by gavage 20 minutes before 600 kPa HBO exposure. Control mice received the vehicle only. Seizure latency was recorded. Malondialdehyde content in brain and lung tissues, total protein level in bronchoalveolar lavage fluid (BLF) and lung water content were measured 60 minutes after the hyperbaric exposure. Histopathology of lung tissue was undertaken.

Results: Compared with the vehicle alone, BD-AcAc$_2$ prolonged seizure latency in a dose-dependent manner ($P < 0.01$). The HBO-induced increase in brain malondialdehyde, BLF protein and lung water were significantly reduced by BD-AcAc$_2$ ($P < 0.01$).

Conclusion: Oral administration of the ketone ester BD-AcAc$_2$ significantly protected against CNS-OT and concomitant ALI. Alleviation of oxidative stress may be one underlying mechanism providing this effect.

Introduction

Hyperbaric oxygen (HBO) treatment is widely used in the treatment of a variety of medical conditions and diving related illnesses.[1] It has been demonstrated that HBO alleviates tissue hypoxia and stimulates endogenous protection mechanisms, including expression of cytoprotective proteins thus enhancing cellular tolerance against harmful stimuli.[2,3] To a certain extent, the therapeutic effect of HBO is positively correlated to dose (a function of pressure and duration of exposure). However, HBO may also lead to oxygen toxicity (OT), whose likelihood is also positively correlated to inspired oxygen pressure and duration. OT is one of the most concerning complications of HBOT and the dose of HBO is therefore limited in clinical practice.

OT results from the harmful effects of breathing oxygen (O$_2$) at elevated partial pressures.[4] Depending on the inspired O$_2$ pressure and duration, different types of OT may occur. Long-term exposure to elevated O$_2$ levels, even at normal atmospheric pressure, may affect the lungs, eyes and other organs. Short-term exposure to a partial pressure of O$_2$ greater than at normal atmospheric pressure may lead to central nervous system OT (CNS-OT), which is of great

concern in both diving operations and during HBOT. With little or no warning, CNS-OT can result in a grand mal convulsion and, if occurring during diving, in drowning and death.[5,6] Development of strategies to prevent CNS-OT could improve the safety of HBOT and diving operations.

Previous studies have reported that fasting and a ketogenic diet (KD) can inhibit refractory seizures, an effect which might be associated with increased ketone in the blood.[7-9] Administering the KD has been reported to completely abolish seizures in up to 13% of patients and reduce seizure frequency by > 50% in approximately 67% patients with refractory epilepsy.[10] The KD is now well established as a preventative measure for seizures. Convulsions resulting from CNS-OT share many common pathophysiological mechanisms with other clinical seizures. Some reports have shown that KD can delay the onset of CNS-OT.[11,12] However, in order to be effective, the diet needs to have been established over a long period and needs strict dietary compliance that can impede its successful use.

A recent study reported that oral administration of ketone ester BD-AcAc$_2$ could mimic the effects of a KD and induce ketosis.[13] It could also inhibit seizures and delay CNS-OT

Figure 1
Structure of BD-AcAc$_2$
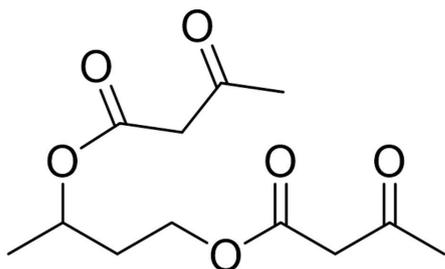
onset in rats.[14] In the present study, the protective effects of BD-AcAc$_2$ on CNS-OT, including concomitant acute lung injury (ALI) were evaluated, as were any possible antioxidative mechanisms underlying any protective effects.

Methods

The experimental procedure was approved by the Institutional Animal Care and Use Committee of the Naval Medical University, Shanghai (number 201711270007). All efforts were made to minimize suffering to the animals.

ANIMALS AND COMPOUND BD-ACAC$_2$

Male mice (C57BL/6) (20 ± 2 g) were provided by the animal centre of the University and housed in a temperature (24 ± 1°C) and humidity (54 ± 2%) controlled environment with a 12/12 h light/dark cycle and free access to food and water. Compound BD-AcAc$_2$ (Figure 1) was synthesized by transesterification and verified by proton nuclear magnetic resonance (1HNMR) and mass-spectrometry.

GROUPS AND TREATMENT

After being fasted for 18 h, mice were randomly divided into six groups ($n = 8$): Normal group, Null group, Vehicle group and treatment groups receiving three different doses of BD-AcAc$_2$. BD-AcAc$_2$-treated mice received BD-AcAc$_2$ in 0.4 ml of a peanut oil vehicle by gavage in a dose of 2.5, 5.0, or 10.0 g·kg^{-1} body weight, and the Vehicle group mice received 0.4 ml peanut oil alone. The Normal group did not receive any treatment and was used as control. The Null group was exposed to HBO but received neither the vehicle nor BD-AcAc$_2$. The Null, Vehicle and three Treatment groups were exposed to HBO (see below). In the Vehicle and Treatment groups this occurred 20 minutes (min) after gavage with the vehicle or vehicle + BD-AcAc$_2$ respectively. All mice were anaesthetized 60 min following HBO exposure with 1% pentobarbital sodium (50 mg·kg^{-1} body weight) intraperitoneally before sampling.

HBO EXPOSURE

The mice were placed singly in an animal compression chamber (RDC150-300-6, Naval Medical University,

Shanghai, China). The chamber was flushed with pure oxygen for 5 min before being compressed to 600 kPa. The compression and decompression were both carried out at a rate of 100 kPa·min^{-1}. The previously used exposure duration of 30 min was adopted.[15,16] However, if the convulsion latency was longer than 30 min in the treated mice, the exposure was be continued until the onset of a convulsion. A continuous oxygen flow of 0.5 L·min^{-1} was maintained during the exposure and soda lime was placed in the bottom of the chamber to prevent the accumulation of CO_2.

CNS-OT LATENCY

During the HBO exposure the behaviour of the mice was observed. The convulsion latency was the elapsed time from achievement of a chamber pressure of 600 kPa to the occurrence of tetanic contraction of whole body and persistent spasm.[17]

BRONCHOALVEOLAR LAVAGE PROTEIN

After midline laparotomy, the abdominal aorta was cut and the mouse was exsanguinated. After isolation of the right lung, the left lung was lavaged with 0.3 ml precooled phosphate buffered saline (PBS) three times. The lavage fluid was then collected and centrifuged at 1500 g for 10 min; the supernatant was stored at -80℃. The protein in the supernatant was measured using a bicinchoninic acid (BCA) kit (Beyotime, Haimen, China).

LUNG WATER CONTENT

The superior and inferior lobes of the right lung were collected. The water on the lung surface was dried with blotting paper and the lung was weighed after drying in a 60℃ oven for 72 h. Lung water content (%) was calculated as:
Wet weight – Dry weight / Wet weight ×100.   (1)

MALONDIALDEHYDE (MDA) IN BRAIN AND LUNG

The brain and middle lobe of the right lung were washed with precooled saline and stored at -80℃. Fifty milligrams of the tissue was lysed with western and cell lysis buffer (Beyotime, Haimen, China). Homogeneous lysates were centrifuged at 1600 g for 10 min and the supernatant was measured using a BCA kit. An MDA Kit (Beyotime, Haimen, China) was then used to assay MDA as an index of oxidative injury to the brain and lung.

HAEMOTOXYLIN AND EOSIN (H&E) STAINING

The post-caval lobe of the right lung was fixed with 4% paraformaldehyde followed by paraffin embedding. The tissue was then sectioned followed by H&E staining. The sections were examined by plain microscopy to look for tissue inflammation, oedema or other damage.

Figure 2

Latency of CNS-OT in BD-AcAc$_2$ treated mice; data are expressed as mean ± SD, $n = 8$; ** $P < 0.01$ vs. Vehicle group
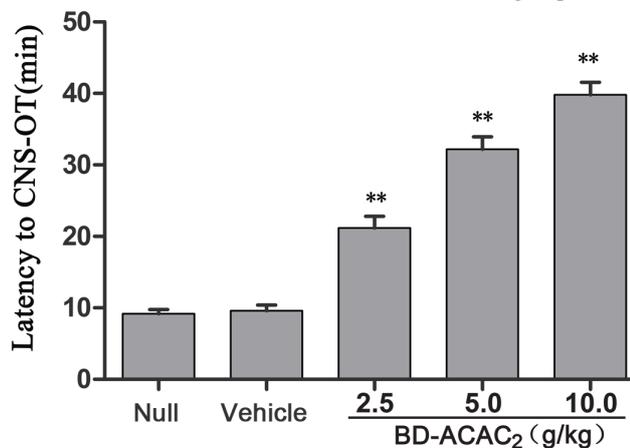
Figure 3

Total protein in bronchoalveolar lavage fluid after HBO exposure; data are expressed as mean ± SD, $n = 8$; ** $P < 0.01$ vs. normal group; ## $P < 0.01$ vs. Vehicle group
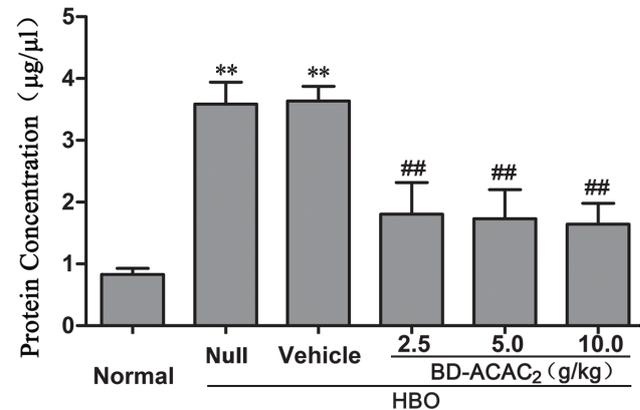
Figure 4

Water content (expressed as a percentage of total weight) in lung tissue after HBO exposure; data are expressed as mean ± SD, $n = 8$; ** $P < 0.01$ vs. Normal group; ## $P < 0.01$ vs. Vehicle group
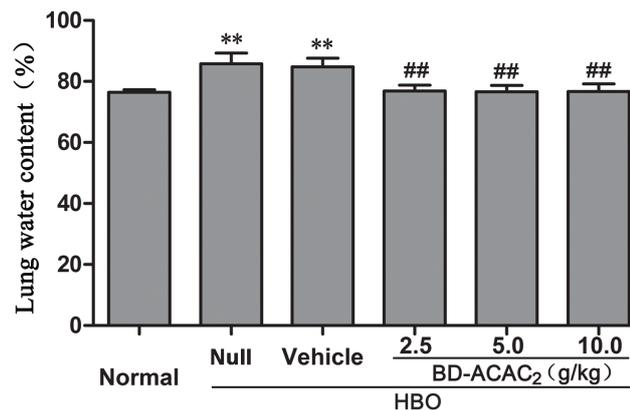
Figure 5

MDA content in cerebral cortex and lung tissues after HBO exposure; data are expressed as mean ± SD, n = 8. ** $P < 0.01$ vs. Normal group, ## $P < 0.01$ vs. Vehicle group
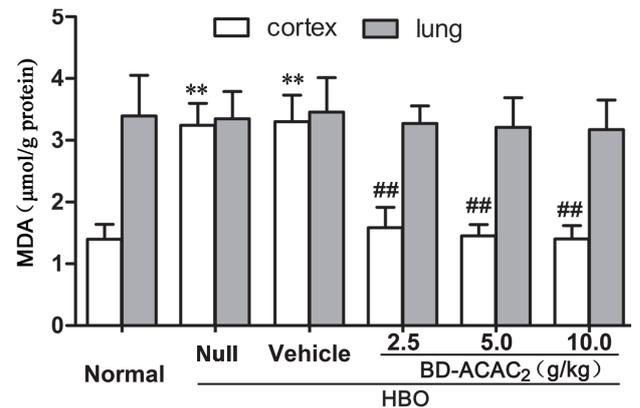
STATISTICAL ANALYSIS

The data were analyzed with SPSS21.0 software. All data were presented as mean ± standard deviation (SD), and the differences between groups were compared with one-way ANOVA. $P < 0.05$ was taken to indicate statistical significance.

Results

Compared with the vehicle group, BD-AcAc$_2$ significantly prolonged the seizure latency of CNS-OT in a dose dependent manner ($P < 0.01$). There was no difference between the Vehicle and the Null group (Figure 2) suggesting that the vehicle had no role in the beneficial effect of BD-AcAc$_2$.

Total protein levels in the bronchoalveolar lavage fluid were significantly increased by HBO exposure in the Null and Vehicle groups ($P < 0.01$) whereas pretreatment with BD-AcAc$_2$ significantly reversed this effect ($P < 0.01$) (Figure 3). Similarly, HBO exposure significantly elevated the water content in lung tissues ($P < 0.01$), whereas pretreatment with BD-AcAc$_2$ significantly decreased this effect ($P < 0.01$) (Figure 4).

MDA content in brain tissue was significantly increased after HBO exposure ($P < 0.01$) and this was reversed by BD-AcAc$_2$ at all tested doses ($P < 0.01$). The HBO exposure did not affect lung MDA content (Figure 5).

Staining and microscopy revealed significant lung damage in the Vehicle group, which comprised congestion in the bronchial wall and alveolar tissue, inflammatory cell infiltration and lung tissue structure distortion. This was substantially alleviated in groups receiving BD-AcAc$_2$, with the only change noted being pulmonary vasodilation (Figure 6).

Figure 6
Examples of lung histology (haemotoxylin and eosin staining) after HBO exposure; A – Normal group (No intervention); B – Null group (HBO only); C – Vehicle group HBO and drug vehicle (see test); D – BD-AcAc$_2$ 2.5 g·kg^{-1}; E – BD-AcAc$_2$2 5.0 g·kg^{-1}; F – BD-AcAc$_2$ 10.0 g·kg^{-1}; magnification x 40
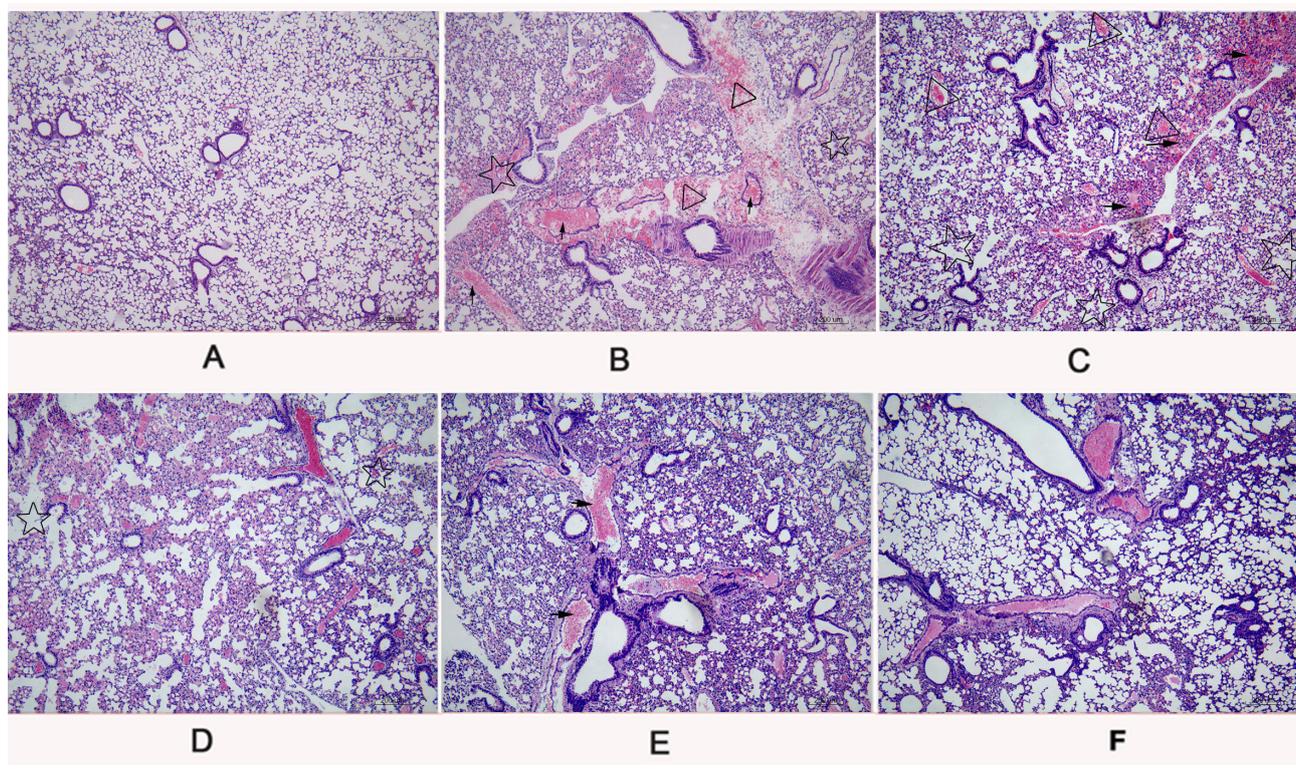
Discussion

A previous study showed that fasting delayed the latency of CNS-OT; an effect which appears to be related to changes of brain energy metabolism induced by blood ketones.[18] The KD mimics the metabolic state of fasting (i.e., therapeutic ketosis) and has proved to be a potential preventive measure for convulsions.[19] However, as the KD requires strict compliance and long-term preparation, its usefulness for this purpose in practice is greatly restricted. Recently, some preliminary studies suggested that oral administration of the ketone ester BD-AcAc$_2$ could mimic the KD and delay the onset of CNS-OT in rats.[13,14] In the present study, these findings were confirmed in a mouse model and the underlying mechanisms were further explored from the perspective of protecting against oxidative injury and the effects of BD-AcAc$_2$ on concomitant ALI were assessed.

The ketone ester BD-AcAc$_2$ and its resulting metabolic compounds might contribute to a range of mechanisms that could delay the onset of OT. Oral administration of BD-AcAc$_2$ would generate acetoacetate (AcAc) and β-hydroxybutyrate (βHB); then part of the AcAc would be decarboxylated to become acetone.[14] All three of these resulting compounds could then easily pass through blood brain barrier via a monocarboxylic acid transporter.[20] The mechanisms by which BD-AcAc$_2$ exerts its anti-OT effect might be associated with these increased ketone bodies in

the blood. It is widely accepted that increased production of reactive oxygen species (ROS) makes a major contribution to the development of CNS-OT.[6] Ketone body metabolism can improve the function of mitochondria and reduce ROS production, thus inhibiting ROS-related cell damage and reducing oxidative stress injury.[21,22]

Ketone bodies might also be associated with adenosine metabolism. Therapeutic ketone bodies have been reported to significantly inhibit seizures in mice, but not in A1 adenosine receptor (A$_1$R) knockout mice.[23] Ketone bodies can increase adenosine content and A$_1$R activity in the brain, reduce adenosine kinase (ADK) activity and inhibit the release of the excitatory neurotransmitter glutamate.[24] It is also known that the balance of glutamate (Glu) and γ-aminobutyric acid (GABA) plays an important role in the pathophysiological process of seizures. AcAc may not only inhibit Glu release[25] and promote its conversion to GABA[26], thus reducing neuronal excitability, but it may also enhance the anti-oxidation ability of hippocampal neurons and inhibit seizure activity.[27] βHB has a similar structure to GABA; it may mimic the effects of GABA and reduce neuronal excitability.[28]

The present study showed that oral administration of BD-AcAc$_2$ significantly delayed the onset of oxygen convulsions in a dose dependent manner. The strongest effect accrued from the highest dose (10.0 g·kg^{-1} body

weight), in which the mean convulsion latency was substantially extended from 9.6 min to 39.8 min. Exposure to HBO significantly increased levels of MDA; a sensitive indicator of oxidative stress injury in brain tissue. BD-AcAc$_2$ significantly reversed this change, suggesting (though not proving) that alleviation of oxidative stress may be the underlying mechanism of delaying CNS-OT. As it is proven that CNS-OT is a self-limiting disease and does not cause brain tissue histopathological changes,[29,30] brain tissue H&E staining was not performed in this study.

The mechanism of CNS-OT concomitant ALI is far from clear. Studies have shown that excessive oxygen exposure can increase ROS production in the lung, which may result in cell injury, increased endothelial permeability and eventually pulmonary oedema.[31] However, the role of oxidative injury in CNS-OT concomitant ALI has not been properly studied. In our study, microscopy on stained lung sections from Null or Vehicle group mice showed that 30 min of 600 kPa HBO exposure resulted in severe lung damage manifest as oedema and congestion, accompanied by inflammatory cell infiltration and lung tissue structure distortion. In contrast, BD-AcAc$_2$-treated mice only displayed slight pulmonary vasodilatation. These results indicated that BD-AcAc$_2$ was effective in CNS-OT concomitant ALI prevention. However, as MDA content in lung tissue did not increase after HBO exposure, this may indicate that oxidative injury did not play a role in CNS-OT concomitant ALI CNS-OT. Concomitant ALI has been associated with a sharp increase in pulmonary vascular pressure.[32] Toxic levels of HBO exposure can lead to a large sympathetic activation of the central nervous system, resulting in the occurrence of pulmonary hypertension. Concurrently, sympathetic activation can release a large amount of catecholamine, disturbing the autonomic nervous balance and resulting in acute lung injury.[33] Adenosine may also play an important role in preventing this pathological process. Studies have shown that adenosine can significantly reduce lung surface haemorrhage and tissue damage, reduce lung tissue leakage, reduce pulmonary oedema, and effectively alleviate acute lung injury induced by oxygen convulsions.[34] Ketoesters may work by modulating adenosine, as discussed above. However, whether this is the underlying protective mechanism needs further investigation.

In conclusion, this study demonstrated that oral administration of the ketone ester BD-AcAc$_2$ could significantly protect mice from CNS-OT and concomitant ALI. However, the underlying mechanisms remain unclear, so further investigations are needed to provide more evidence for its possible future application in diving operations and HBOT.

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Review article

Vibration and bubbles: a systematic review of the effects of helicopter retrieval on injured divers

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Abstract

(Blake DF, Crowe M, Mitchell SJ, Aitken P, Pollock NW. Vibration and bubbles: a systematic review of the effects of helicopter retrieval on injured divers. Diving and Hyperbaric Medicine. 2018 December 24;48(4):241−251. doi: 10.28920/dhm48.4.241-251. PMID: 30517957.)

Introduction: Vibration from a helicopter during aeromedical retrieval of divers may increase venous gas emboli (VGE) production, evolution or distribution, potentially worsening the patient's condition.

Aim: To review the literature surrounding the helicopter transport of injured divers and establish if vibration contributes to increased VGE.

Method: A systematic literature search of key databases was conducted to identify articles investigating vibration and bubbles during helicopter retrieval of divers. Level of evidence was graded using the Oxford Centre for Evidence-Based Medicine guidelines. A modified quality assessment tool for studies with diverse designs (QATSDD) was used to assess the overall quality of evidence.

Results: Seven studies were included in the review. An *in vitro* research paper provided some evidence of bubble formation with gas supersaturation and vibration. Only one prospective intervention study was identified which examined the effect of vibration on VGE formation. Bubble duration was used to quantify VGE load with no difference found between the vibration and non-vibration time periods. This study was published in 1980 and technological advances since that time suggest cautious interpretation of the results. The remaining studies were retrospective chart reviews of helicopter retrieval of divers. Mode of transport, altitude exposure, oxygen and intravenous fluids use were examined.

Conclusion: There is some physical evidence that vibration leads to bubble formation although there is a paucity of research on the specific effects of helicopter vibration and VGE in divers. Technological advances have led to improved assessment of VGE in divers and will aid in further research.

Introduction

Although scuba diving is a relatively safe sport, 15 divers died in Australia in 2011[1] and 168 divers were treated for decompression illness (DCI), 33 of these in Queensland.[2] The possibility of a diver having DCI often necessitates retrieval for medical assessment and possible recompression. Retrieval options commonly include: water (dive boats, police, and coast guard), land (ambulance, private car, bus[3]) and air (rotary or fixed wing aircraft). Many factors are considered when deciding on the most appropriate retrieval platform: patient location and access, acuity, distance to definitive care, response time, speed, weather, time of day, altitude exposure, crew skill mix and platform availability.[4] Owing to dive site remoteness, helicopter aeromedical retrieval is often used for short-haul transport of divers. It has been suggested that the vibration associated with helicopter transport may cause increased bubble generation in divers potentially worsening DCI.[5–8]

DCI is a collective term encompassing the clinical consequences of two different pathophysiological processes:

introduction of bubbles to the arterial circulation by pulmonary barotrauma (referred to as arterial gas embolism (AGE)); and formation of bubbles from inert gas (referred to as decompression sickness (DCS)). Both involve bubbles as presumed primary vectors of injury. There is potential difficulty in clinical distinction between them, and the modern trend in therapy is to treat both in the same manner.[9] These considerations provided the motivation for referring to them collectively as DCI with the nomenclature describing the clinical picture: acute/chronic, evolution, organ system involved, and degree of severity (e.g., acute, stable, mild neurological DCI).[10]

AGE occurs secondarily to pulmonary barotrauma, where expanding alveolar gas enters the systemic arterial circulation.[9] If these bubbles enter the cerebral circulation and cause central neurological symptoms it is called cerebral arterial gas embolism (CAGE). DCS, on the other hand, is presumably caused by the formation of bubbles in the extravascular space or within tissue capillaries primarily from dissolved inert gas. These capillary bubbles subsequently appear in the systemic veins as venous gas emboli (VGE). The *in vivo* formation of bubbles at levels of supersaturation that are vastly lower than predicted to be required for bubble formation in pure solutions suggests the existence of surfactant-stabilized gas micronuclei into which supersaturated gas diffuses to form larger bubbles.

The exact mechanism by which these bubbles cause the symptoms of DCS is not clear. Indeed, they do not always cause problems, but when in abundance are commonly held to be the inciting factors in DCS[9] with the probability of DCS correlated with the bubble load detected.[11] Bubble formation is generally accepted as an indicator of decompression stress and DCS risk in research where the generation of clinical DCS would be an unacceptable end point.[11]

Post-dive risk factors for increased bubble production and DCS include elevated temperature exposure, altitude exposure and exercise. Hot showers post-diving cause vasodilation and decreased inert gas solubility potentially leading to increased bubbles and possible DCS.[12] The decreased ambient pressure accompanying altitude exposure increases any tissue supersaturation which in turn will increase bubble formation. If bubbles were already present the decreased ambient pressure also promotes bubble growth.[13,14] VGE have been detected in some divers during a commercial flight 24 hours after completing their last dive, even though no pre-flight VGE were detected.[15] Exercise post-diving may cause small bubbles to grow and promote new bubbles by physical excitation of the tissues by the process of tribonucleation.[16] Tribonucleation is the process of bubble formation when solid surfaces immersed in a liquid are pulled apart.[17] It has been stated that vibration can lead to tribonucleation and, especially in supersaturated tissues, lead to increased gas bubble loads.[18,19] Pre-dive vibration at 35–40 Hz[20,21] and impact exercise[22] reduced bubble formation, presumably by dislodging pre-existing micronuclei from

crevices or enhancing lymphatic elimination of gas nuclei.[20] Low frequency vibration[20] and movement[23] post-dive have been reported to increase VGE presumably by a similar mechanism of dislodging micronuclei that are growing as inert gas from the surrounding supersaturated tissue diffuses into them.

Low frequency vibration of this nature can be encountered during helicopter transport. It has been hypothesized that this may increase VGE generation, evolution or distribution in a diver, potentially worsening their condition.[5–8] We are not aware of any evidence that could guide the clinician regarding the related risks of helicopter retrieval of scuba divers. Therefore, the purpose of this report is to systematically review the literature surrounding helicopter transport of injured divers to find any evidence that vibration contributes to increased VGE formation and worsening DCS.

Methods

A systematic search of the literature was conducted to identify articles investigating vibration and bubble generation during helicopter retrieval of divers. Databases searched included MEDLINE, CINAHL, Informit, EMBASE, Scopus, Web of Science, Rubicon Foundation and Cochrane Central, with no date limits (i.e., from database inception to April 2018). Medical subject headings (MeSH) and key words used as search terms included: diving* OR scuba OR "*self contained underwater breathing apparatus*"; bends OR "*caisson disease*" OR "*decompression sickness*"; helicopter* OR "*air ambulance*" OR "*emergency helicopter*" OR "*helicopter ambulance*"; vibration AND "*transportation of patients*". Results of the search were exported to a reference managing database (EndNote X7). Preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines were followed (Figure 1).[24] All authors participated in the development of the inclusion and exclusion criteria.

Titles and abstracts were screened and the full texts of potentially relevant articles were obtained for review. Inclusion criteria were helicopter retrieval, scuba divers, bubble production, and DCS. Owing to the limited number of articles on vibration and bubble formation, inclusion criteria were broadened to include articles that only presented data on helicopter retrieval of divers. Exclusion criteria for each stage of the review are listed in Figure 1. Exclusion in the initial screening (DFB) included articles pertaining to animals that dive, mechanical vibrations of platforms and equipment, helicopter underwater escape training, and syndromes from using equipment that vibrates such as a jack hammer. Full text review exclusions included articles with no data and only expert opinion, consensus statements, historical review of retrieval services and listing of patient presentations with no differentiation between modes of arrival.

Reference lists of identified publications were reviewed for additional relevant articles. All non-English language

Figure 1
PRISMA flow diagram

| **Identification** | Cochrane Central (*n* = 2) | Medline (*n* = 139) | Informit (*n* = 79) | EMBASE (*n* = 177) | CINAHL (*n* = 9) | Scopus (*n* = 310) | Web of Science (*n* = 105) | Rubicon (*n* = 77) |

Records after duplicates removed
(*n* = 456)

Additional records identified through other sources
(*n* = 6)

Screening

Records screened
(*n* = 462)

Records excluded
(*n* = 426)
Unrelated animal research
Mechanical not helicopter
HUET/submarine escape
Sound levels/speech
Springboard diving
Temperature
Helicopter crashes
Equipment/alarms
Vibration syndromes
Space travel
Flying after diving/treatment
Pre-dive conditioning
Retrieval and vibration,-no diving
Altitude,-no vibration
Diving incident reports
Dive preparedness
Transfer under pressure

Eligibility

Full-text articles excluded, with reasons
(*n* = 29)
Expert opinion *n* = 6
Historical review *n* = 3
Consensus statements *n* = 5
Diving incident report *n* = 1
Portable recompression chambers *n* = 1
Altitude exposure *n* = 1
No DCI *n* = 1
No helicopter retrieval *n* = 2
No differentiation between mode of transport *n* = 5
Already reported data *n* = 1
Pre-dive vibration *n* = 1
Phone advice *n* = 1
Mathematical modeling n = 1

Full-text articles assessed for eligibility
(*n* = 36)

Included

Studies included in qualitative synthesis
(*n* = 7)

(Danish, French, German, Italian and Japanese) abstracts and relevant articles were translated. Two authors (DFB, MC) reviewed the literature; the content experts (SJM, PA, NWP) also being tasked to identify any further missing literature (none were found). The manuscript and tables were critically reviewed by all authors.

Included articles were evaluated and their initial level of evidence (LOE) was determined based on the reported research methodology utilized by the Oxford Centre for Evidence-Based Medicine.[25] A levels-of-evidence table guided the initial grading when assessing each study's research question and methodology. The next step was to adjust the initial grade based on study quality, imprecision, indirectness, inconsistencies and effect size.[26] The grading of

recommendations, assessment, development and evaluation (GRADE) approach was then used to upgrade or downgrade the initial LOE as appropriate.[26] The overall quality of evidence was assessed using the quality assessment tool for studies with diverse designs (QATSDD).[27] This tool allows for the comparison of studies with differing methodological research designs. Papers are graded on a scale of 0 to 3 for each criterion. The score is then summed and expressed as a percentage of the maximum possible score. Each individual paper is given a quality score and the percentage then allows for comparison across the differing methodologies within the same field of research.[27] An interpretation of the scores can then allow for classification into low (< 50%), medium (50–80%) or high (> 80%) quality evidence. The QATSDD tool was modified by excluding one criterion, "*evidence of*

Table 1

Characteristics of studies included in systematic review; [a] LOE – level of evidence: Level 1 high (systematic review), Level 2 (randomized trial), Level 3 (cohort study), Level 4 (case-series), Level 5 low (Mechanism-based reasoning)[25]; [b]Type 2 DCS is considered more serious and includes central nervous system (CNS), inner ear and cardiorespiratory symptoms; AGE – arterial gas embolism; CAGE – cerebral arterial gas embolism; CNS – central nervous system; DCI – decompression illness; DCS – decompression sickness; Hz – hertz; HBO – hyperbaric oxygen; kPa – kilopascal; IV – intravenous; SANDHOG – San Diego Diving and Hyperbaric Organizations; VGE – venous gas emboli; W – watt

Study	LOE[a]	Study design	Participants	Methods	Results
Bubbles and vibration					
Ikels[17]	5	in vitro	Liquids of various viscosities	Liquids of varying viscosity saturated with various gases (including nitrogen) at ambient pressure; bubble production observed during hypobaria and from steel ball rolled down a tube	High steel ball velocity facilitated bubble formation. Extreme hypobaria for bubbles to form in human blood. Hypobaria for bubble formation increased as $P_{gas} \downarrow$. Hypobaria for bubble formation decreased as viscosity increased
Balldin[6]	3	Block randomized (1–5 vibrate/rest; 6–10 rest/vibrate)	10 healthy male scuba divers 28 ± 7 years (y) (range 18 to 38 y)	1. 250 kPa, 100 minutes (min), hyperbaric chamber; cycling (75 W), 2 min work/2 min rest; 2. 70 kPa, 120 min + vibration 15 Hz, 15 min on/off, seated. *"some further participants"* vibrated @ 25 Hz Precordial Doppler	No significant difference in VGE detection
Retrieval method comparison					
Bennett[7]	4	Retrospective consecutive case series	107–109 patients with DCI (clinical diagnosis) 78% male, average age 29 y	2-year review; retrieval method; time of symptoms to treatment; 6-week follow up; 131 reviewed, 16 excluded – insufficient data or failure to follow up = 115	Helicopter for intermediate distance, difficult access, worse injured, shorter time to treatment, no worse outcome
Bennett[4]	4	Retrospective consecutive case series	133 patients with DCI (clinical diagnosis) No sex/age data	Time frame not reported; transport platform, time to recompression, altitude stress and resolution of symptoms at discharge	Helicopter shorter time to treatment, outcomes similar in all modes of transport
Helicopter retrieval of divers					
Cristina[8]	4	Retrospective case series	25 male divers 24 DCS type 2[b] age 31 ± 8 y 1 AGE, age 35 y	7 years of helicopter retrievals by the Italian military	2.8% of missions for divers. 24 DCS type 2[b], 1 AGE. All treated with oxygen and IV fluids. No deterioration enroute, all flights < 300 metres, transport time 53 ± 9 min
Oode[30]	4	Retrospective consecutive case series	28 patients with DCI (SANDHOG criteria[31]) 71% male, 45 ± 2 y	4 years of transportation in physician-staffed helicopters 34 identified; 6 excluded due to cardiopulmonary arrest at scene	0 deterioration, 20 no change, 8 improved during flight. All received oxygen; all but 1 received IV fluids; all flights < 300 metres
Reddick[13]	5	Retrospective case series	6 males; average age 28 y	Cases of DCS over 18 months in altitude chamber participants	5 pain and cutaneous DCS, 1 peripheral paraesthesia developed into CNS DCS; all transported by helicopter and received oxygen; 3 worsened in flight, all improved with lower altitude

Table 1 (continued)

Study	Conclusions	Limitations
Bubbles and vibration		
Ikels[17]	Tribonucleation may lead to bubble formation where surfaces rub across one another such as joints, small blood vessels expanding/contracting or muscle tendon insertion sites.	Blood is a complex liquid and was only used in initial decompression studies with no saturation or vibration.
Balldin[6]	Differences were inter-individual. Helicopter vibrations complex. No effect on release of VGE with vibration frequencies used.	Lack of allocation concealment and blinding; Wet (immersed) vs dry hyperbaric exposure not outlined; Timing of Doppler unable to be determined; No grading of Doppler signal, only length of time VGE heard; No blinded independent review of Doppler signals; Only used 15 and 25 Hz sitting in vibrating chair; Altitude as confounder, may obscure any increase with vibration.
Retrieval method comparison		
Bennett[7]	Early transfer of severe injury may outweigh or disguise risk of helicopter transport.	8 patients with DCS via helicopter, 12 CAGE; Scoring by chart review; Number of HBO treatments not included, nor entry or discharge classification; Classification system not validated; Inconsistent data.
Bennett[4]	Retrieval of divers is complicated, consider physiology and retrieval system. No good outcome evidence to guide strategies.	Most of helicopter group CAGE, so not included in detailed analysis of time to treatment, altitude and recovery grade. Grading scale developed in previous study not used. No information on pre-treatment severity, pre-hospital treatment or number of HBO treatments; No follow-up.
Helicopter retrieval of divers		
Cristina[8]	Considerations for retrieval air speed and vibrations.	No number of HBO treatments or outcome at end of treatment; No retrieval method comparison group; No follow-up.
Oode[27]	Subjective improvement via helicopter if altitude <300 metres + administration of O_2 and fluids.	Unsure if any patients had CAGE; No details on patients requiring intubation ($n = 4$); Oxygen saturation improvement clinically insignificant; No analysis between groups with change in symptoms; No data on HBO treatment or follow up; No retrieval method comparison group.
Reddick[15]	No worsening of symptoms if helicopter stayed below 61 metres above ground level of the take-off point. Retrieval twice as fast air vs land.	Altitude exposure pre-DCS; Not divers; No retrieval method comparison group.

user involvement in design", as it did not appear relevant to the included studies. Two authors (DFB and MC) replicated and agreed on the grading.

Results

The combined searches initially identified 898 records with 456 records remaining after duplicates were removed (Figure 1). After initial screening, 36 full text articles were assessed for eligibility. Limited scientific literature was available on this topic, and only seven articles met the broadened inclusion criteria. Table 1 contains a summary of the seven relevant articles. Study characteristics including participants, study design, methods, results, author conclusions and study limitations are presented. The overall quality of clinical evidence was low with only one clinical article reaching a 50% score on the modified QATSDD assessment (Table 2). An *in vitro* research paper provided some evidence of bubble formation with gas saturation and vibration. Only one prospective intervention study was identified which examined the effect of vibration on VGE formation. The remaining studies were retrospective chart reviews of helicopter retrieval of divers. The articles have been categorised into three groups for comparison: bubbles and vibration, retrieval method comparison and helicopter retrieval only.

BUBBLES AND VIBRATION

Preliminary *in vitro* research into tribonucleation was completed on human blood suggesting that significantly decreased pressure was required for bubbles to form.[17] Further studies were then completed using liquids of varying viscosities saturated with different gases. The liquids were examined for bubble generation under decreasing pressure and varying velocities of a steel ball rolling down the side of a test tube which acted as a vibration stimulus. High steel ball velocity increased bubble formation. The amount of decompression required for bubble formation was inversely proportional to the gas supersaturation, i.e., as the gas supersaturation decreased the amount of pressure reduction required for bubble formation increased.

This article was assessed as a medium quality of evidence with a modified QATSDD score of 79% (Table 2) and a LOE grade 5. Even though this was *in vitro* research, the study provides important insight into the physics of bubble generation. The addition of a mechanical stimulus to the combination of gas saturation and pressure reduction increased bubble production. Only preliminary work was completed on human blood with the more extensive research conducted on liquids of varying viscosities.

Only one prospective intervention study assessing VGE generation in divers exposed to vibration was found.[6] Healthy, young, male scuba divers were enrolled and exposed to 250 kPa for 100 min in a hyperbaric chamber with cycling for 2 min/2 min rest. Within five min of surfacing the divers were taken to 70 kPa (hypobaric) for two hours. During the hypobaric exposure the divers were vibrated for 15-min periods at 15 hertz (Hz) in a seated chair alternated with a 15-min period of non-vibration for a total of 120 min. VGE were detected using precordial Doppler with duration of bubble signal recorded in seconds (sec). All participants were then treated with oxygen (O_2) at 220 kPa for 30 min. Results were reported as duration of bubbles in sec with no significant differences found between the vibrated and non-vibrated conditions.

This article was assessed as having a low quality of evidence with a modified QATSDD score of 26% (Table 2) and a LOE grade 3. This was a block randomized, quasi-crossover study with no allocation concealment or blinding of participants or adjudicators. Dry versus wet (immersed) hyperbaric exposure was not outlined. Participants sat in a vibration chair and were therefore not supine. Sitting would be an unusual position for a patient during helicopter retrieval and not the recommended position for optimal inert gas washout.[28] Helicopter vibration frequencies occur in a wide range of frequencies from 5 to 150 Hz.[29] Only 15 (peak 0.23 ĝz) and 25 (peak 0.64 ĝz) Hz exposures were performed. The peak accelerations used were much lower than peak helicopter accelerations quoted in the literature.[29] The exact timing of the Doppler scans was unclear with no grading scale used. There was no reporting of training of the Doppler technician, how the recordings were saved or whether there was independent blinded review of the recordings. Since this study, technological advances and reporting guidelines have substantially changed,[23] so the results require cautious interpretation. The main limitation is the use of altitude exposure to generate VGE. The altitude exposure, known to produce a great amount of bubbles, may have acted as a confounder and masked any difference in VGE generation between the vibration and non-vibration conditions.

RETRIEVAL METHOD COMPARISON

Two retrospective case series compared outcomes of injured divers treated at a hyperbaric facility stratified by retrieval mode. Retrieval modes included fixed and rotary wing aircraft, road ambulance or self-referral. Both studies were written by the same author and based in Sydney, Australia. Most helicopter retrievals were for divers with CAGE and not DCS.

The medical records of 131 consecutive divers who presented to The Prince Henry Hospital, Sydney, Australia for treatment of DCI over a two-year period were retrospectively reviewed.[7] Cases were classified by retrieval method, time of symptom onset to recompression and a six-week follow-up score designed for the study. Most of the patients were self-referrals. More than half (60%) of the helicopter retrieval group were diagnosed with CAGE, with two deaths in the CAGE group. The discharge scores

Table 2

Quality of clinical evidence for systematic review of vibration and bubbles in divers; * QATSDD – quality assessment tool for studies with diverse designs; 0 = not at all; 1 = very slightly; 2 = moderately; 3 = complete; n/a − not applicable (*in vitro* study)

QATSDD* Criteria	Study						
	Ikels[17]	Balldin[6]	Bennett[7]	Bennett[4]	Cristina[8]	Oode[30]	Reddick[13]
Explicit theoretical framework	3	1	2	2	1	1	0
Statement of aims/objectives in main body of report	2	3	3	1	2	2	0
Clear description of research setting	3	2	3	1	3	3	1
Evidence of sample size in terms of analysis	0	0	0	0	0	0	0
Representative sample of target group of reasonable size	n/a	1	2	1	3	1	1
Description of procedure for data collection	3	1	2	1	1	2	0
Rationale for choice of data collection tool(s)	3	0	2	0	0	0	0
Detailed recruitment data	n/a	0	2	1	2	2	1
Statistical assessment of reliability and validity of measurement tool(s) (Quantitative only)	2	0	0	0	0	0	0
Fit between stated research question and method of data collection (Quantitative only)	3	1	2	2	1	2	0
Fit between research question and method of analysis (Quantitative only)	3	0	1	1	0	2	0
Good justification for analytical method selected	3	0	0	0	0	0	0
Evidence of user involvement in design	Item not included in scoring as not relevant to included studies						
Strengths and limitations critically discussed	1	1	1	1	0	1	0
Total score	26/33	10/39	20/39	11/39	13/39	16/39	3/39
% of maximum possible score	79	26	51	28	33	41	8

for the DCS patients were similar for each retrieval mode. Patients retrieved via helicopter had a significantly shorter time to first recompression (4.9 hours (h) vs > 20 h road and fixed wing).

This was a pilot study[7] and was assessed as medium quality of evidence with a modified QATSDD score of 51% (Table 2) and a LOE grade 4. Only a very small number of

the patients with DCS were transported by helicopter (7%). The discharge classification scoring system was designed for this study, not previously validated and completed by reviewing the medical records. The patients were followed up at six weeks. The author's conclusion was that the speed of retrieval in the helicopter group and the presumed benefit of earlier recompression may possibly mask any increased risk from rotary wing retrieval.

A further retrospective study performed by the same author reviewed 133 cases treated in the same hyperbaric medicine unit.[4] Cases were analysed by transport platform, time to recompression, altitude stress and status at discharge. Helicopter retrievals (23 patients) were, again, predominantly for divers with CAGE and had markedly shorter average times to recompression (5 h vs > 20 h for road and fixed wing) and, therefore, were not included in further analysis. Altitude exposure was similar in the self-referral (200 metres, m), road (200 m) and rotary wing groups (150 m). Two cases were discussed in more detail to highlight the complexity of retrieval decisions.

This article[4] outlined retrieval options and considerations and was given a low quality of evidence modified QATSDD score of 28% (Table 2) and a LOE grade 4. Most patients retrieved by helicopter were diagnosed with CAGE so the author decided to omit them from the more in-depth analysis of time to recompression and symptom resolution. Even though the helicopter-retrieved patients were omitted from further analysis, their outcomes appear to be similar to the other retrieval modes. The grading scale developed in the previous study[7] was not used and discharge outcome was only classified as full or incomplete resolution with no follow up performed. Within the other retrieval modes there was no information on number of DCS versus CAGE cases in the cohort.

HELICOPTER RETRIEVAL OF DIVERS

This third group of articles were all retrospective chart reviews of DCI cases transported by helicopter. Their focus was on change in clinical condition during helicopter flight. The cases in one article were altitude chamber participants not scuba divers. This article was still included in the review as it was frequently referenced in other articles. All three articles were graded as low quality of evidence (Table 2).

Charts were retrospectively reviewed for a seven-year period of helicopter retrieval flights by the Italian Military search and rescue organization.[8] The authors extracted cases associated with diving injuries. Twenty-five cases were identified (2.8% of total missions) as involving divers, comprising 24 DCS and 1 CAGE. All divers were treated with 100% O_2 and intravenous (IV) fluids en route. All flights were at altitudes of less than 300 m. Transport time was 53 ± 9 min (measure of variance not reported). There was no deterioration reported during any of the flights.

This article[8] was of a low quality of evidence based on the modified QATSDD score of 33% (Table 2) and a LOE score of 4. The retrieval service provided exceptional service with rapid transit times, low altitude flights, O_2 and IV fluids for all patients. There were no data presented on pre-flight O_2 or time from symptom onset to retrieval. No information was presented on injury severity or hyperbaric treatment. There was no comparison with other retrieval methods and no follow up.

The charts of 28 consecutive DCI patients retrieved via helicopter over a four-year period from Izu Peninsula, Japan were retrospectively reviewed.[30] Six divers who had cardiopulmonary arrest at the scene were excluded. Diagnosis of DCI was made using the San Diego diving and hyperbaric organizations (SANDHOG) criteria.[31] Patient demographics and dive characteristics were collected. Patients were classified by type of DCS: central nervous system (CNS), 'chokes', 'chokes'/'bends' and 'chokes'/CNS. All patients received O_2 with delivery device and flow reported. All participants were transported in the supine position and all but one subject received IV fluids. All flights were below an altitude of 300 metres above sea level. Changes in vital signs, Glasgow coma scale (GCS) and change of subjective symptoms before and after the flights were compared. A statistically significant improvement in O_2 saturation pre- and post-flight was found. Four patients required intubation but the timing of this intervention is unclear. No patient deteriorated en route, 20 did not change status and eight improved. Time from request for transfer and arrival at the medical facility was recorded.

This article examined the outcomes of patients diagnosed with DCI in a physician-staffed helicopter retrieval service.[30] The quality of evidence was low based on the modified QATSDD score of 41% (Table 2) and a LOE grade 4. Inconsistent use of the terms DCS and DCI make it difficult to determine if patients who surfaced suddenly were CAGE or CNS DCS. Duration of O_2 therapy was stated to be similar in all patients with reference to a table that does not contain that data. The significant improvement stated for O_2 saturation was clinically insignificant. There is no detail about the timing or indication for intubation in four patients. No follow up data on hospital treatment or outcome were reported. There was no comparison with other methods of retrieval. No analysis of the differences between the patients in relation to change in subjective symptoms was performed.

Six cases of altitude chamber participants with DCS occurring over an 18-month period were retrospectively reviewed.[13] A synopsis of each case was presented. All patients were retrieved by helicopter and received 100% O_2 from diagnosis to delivery to the hyperbaric facility. Three of the patients had worsening of their symptoms during flight which all resolved on flying at lower altitudes. The article was given a very low level of evidence score based on the modified QATSDD score of 8% (Table 2) and a LOE grade 4. The author stated that aeromedical retrieval was twice as fast as ground transportation however no actual retrieval times by other modes were compared.

Discussion

It has been suggested that the vibration generated during helicopter retrieval of injured divers may lead to worsening of DCS due to an increase in inert gas bubbles.[5–8,18] Knowledge in this area is necessary to assist the clinician in deciding the possible risk of retrieving an ill or injured diver

by a rotary wing platform. This systematic review confirms that there is no published research to help guide the retrieval decision-making process.

Physical evidence shows that vibration leads to bubble formation although there is limited published research on the specific effects of helicopter vibration and VGE in divers. Early *in vitro* research demonstrated that movement of an object in a nitrogen-saturated liquid could lead to bubble production.[17] The addition of a mechanical stimulus increased bubble formation more than increased gas saturation and pressure reduction alone. It is plausible that this process of tribonucleation could lead to VGE production in divers exposed to vibration.

An early study exposing healthy divers to vibration found no increase in VGE.[6] However we now understand that helicopter vibrations are much more complex than those simulated in this study with a wider range of frequencies and amplitudes.[29] The range of vibration frequency generated by a helicopter is 5 to 150 Hz,[29] while the two frequencies (15 and 25 Hz) used in this study were too close together to be classified as separate measurements. The peak accelerations used were significantly less than those experienced in a helicopter. The frequencies and amplitudes used in this study do not reflect actual measured helicopter vibrations and may not have been a strong enough stimulus for VGE generation.[29]

Divers are exposed to both altitude and vibration during helicopter retrieval. One of the included articles found that exposure to altitude during the helicopter retrieval of patients with DCS led to worsening symptoms.[13] Improvement in symptoms was seen in divers treated with O_2, IV fluids and restricting the flying altitude to below 300 metres.[8,30] Positioning divers in areas with less vibration during aeromedical retrieval has been suggested with an acknowledgement of little evidence on which to base this recommendation.[18] The previous South Pacific Underwater Medicine Society (SPUMS) policy for initial management of injured or ill divers recommended O_2 administration, IV fluids and to fly as low as possible with 300 metres considered the maximum.[32] However, the most recent consensus guidelines for the pre-hospital management of decompression illness suggests that flying at less than 150 metres above pick-up location is preferred, though no reference for this recommendation is given.[33]

Both AGE and DCS are caused by bubble generation although their mechanisms are significantly different and this limits the generalization of the CAGE outcomes to divers with DCS. The two retrospective articles comparing retrieval methods stated that most of the injured divers retrieved by helicopter were diagnosed with CAGE.[4,7] Another article used confusing nomenclature making it difficult to determine if the included patients had CAGE or DCS.[30] Patients with CAGE tend to be sicker and with symptom onset earlier than patients with DCS. Request for helicopter transport

in these divers may be due to clinical urgency. Speed of retrieval may mask any increased risk from the rotary wing retrieval.[7] CAGE can occur in circumstances of lower inert gas load than serious DCS, meaning that patients may be at less risk of vibration-induced bubble formation if transported by helicopter.

FUTURE RESEARCH

Though the evidence is limited, these articles provide a framework for further research into this area. Technological advances have occurred allowing for better assessment of bubbles in divers.[23] Helicopter vibrations are complex,[29] but measurements of helicopter vibrations during a flight have been acquired.[29] This will lead to the ability to replicate and expose a diver to the position and vibrations encountered during actual aeromedical retrieval. Administration of O_2 pre-vibration should be considered.[8,30,34] Altitude exposure should be eliminated so that the effect of vibration on bubble production can be isolated. Research into this area can lead to a better understanding of the effects of vibrations on divers and provide evidence to guide clinical decisions surrounding the risk/benefit of helicopter retrieval of injured divers.

LIMITATIONS

There is very little specific literature on bubble generation induced by vibration, and none during helicopter retrieval of divers. Studies identified were of diverse designs so the modified QATSDD tool was used to better compare the levels of evidence. The quality of evidence was low with most articles being retrospective reviews. Older investigative techniques were poorly described and therefore it was difficult to interpret the results.[6] Articles were of varying languages but translation allowed for their inclusion. There may be an element of selection bias as only articles found were reviewed. However, the Rubicon Foundation research repository was searched and provided access to some military research documents and many conference proceedings.

VGE are relatively easy to detect using ultrasonic methods by trained technicians and are often used as an outcome measurement in diving research.[35] Even though VGE may be responsible for some symptoms of DCS, they commonly occur after dives without DCS.[36] Nevertheless, higher VGE grades correlate with an increased risk of DCS.[37] Ultrasound techniques are only able to assess intravascular bubbles and therefore provide an incomplete picture of conditions in the whole body. Studies using VGE as a surrogate marker for DCS risk need to use paired comparisons and be well powered.[35]

Conclusions

There is some physical evidence that vibration leads to bubble formation although there is a paucity of research on the specific effects of helicopter vibration and VGE in divers.

Technological advances have led to improved assessment of bubbles in divers and will aid in further research.

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Technical report

Validation of algorithms used in commercial off-the-shelf dive computers

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Abstract

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Introduction: Whilst the US Navy has been very systematic about validating Navy dive computer algorithms, there has been little documented or published evidence of rigorous testing of the algorithms in commercial off-the-shelf dive computers. This paper reports the evaluation of four algorithms used in these – Bühlmann ZHL-16C; VPM-B; Suunto-RGBM; EMC-20H – by comparison with US Navy experimental dives with known decompression sickness outcomes.

Methods: Three specific tests were developed to test the algorithms' ability to mitigate decompression sickness: Total decompression time; no stop times and first stop depth. Output of commercial decompression algorithms were compared to either the probability of decompression sickness (P_{DCS}) results from US Navy man-trials or statistical models derived from P_{DCS} data. The algorithms were first tested with default conservative factors, then these factors were adjusted if the tests were not initially passed. The last verification step was to compare the output of the wrist computer with that of the full desktop algorithm.

Results: This testing indicated that, whilst none of the four passed all of the proposed tests with factory-default conservatism, ZHL-16C and Suunto-RGBM could be made to pass by adjusting user-defined settings.

Conclusions: Man-trial data on P_{DCS} is available to the non-US Navy scientific community for testing of commercial decompression algorithms. This type of validation testing can be very informative on how to best use available commercial dive computers to improve diver safety.

Introduction

In the last few decades, there has been considerable work on the development of new decompression algorithms, as well as variations on existing algorithms. Many of these algorithms are used in commercial off-the-shelf dive computers. During this time, the US Navy Experimental Diving Unit (NEDU) has systematically validated Navy dive computer algorithms against the incidence of decompression sickness. However, there has been little documented or published evidence of rigorous testing of the algorithms in commercial off-the-shelf dive computers used by recreational divers. In a meeting abstract, testing was reported of a wide sampling of commercial dive computers against the few dive profiles for which there are relatively good estimates of the probability of decompression sickness (P_{DCS}).[1] Since that brief report, there have been comparative studies that have simply compared different commercial off-the-shelf dive computers against each other using the same depths and bottom times (defined as the difference between the time the diver leaves the surface and the time at which the diver leaves the maximum depth).[2,3] These two studies showed a wide variation of results in terms of no-stop times (NST, defined as the maximum bottom time for a given depth for which a direct ascent is allowed with no staged decompression) and total decompression time (TDT, defined as the sum of times spent at decompression stops plus the time to travel from depth to surface) varying by up to a factor of two. However, the issue of whether any specific computer (or algorithm) provided too little decompression time or too much was not addressed. This paper assesses some of the algorithms used in commercial off-the-shelf dive computers

Methods

The recommendations from reference 4 were used to develop the testing process: 1) define requirements; 2) validate each algorithm against those requirements; 3) verify to the best extent possible that the algorithms evaluated are the same as those implemented in the chosen dive computers.[4] The main requirements were that the algorithms prescribe decompression schedules that result in an acceptably low P_{DCS}. In addition to this main requirement, one would like this dive profile to be efficient, or not significantly longer than needed as determined by TDT for a given P_{DCS}. Any algorithm can always result in lower P_{DCS} by arbitrarily increasing shallow-depth stop times. However, this can introduce other hazards to the diver, such as running out

Table 1
Overview of decompression algorithms assessed; Exp – exponential

Algorithm	Category	On-gas/off-gas	Number of compartments	Desktop software	Wrist unit used for verification
ZHL-16C	Dissolved gas (Haldanean)	Exp/exp	16	MultiDeco 4.12	Shearwater Perdix
EMC-20H	Dissolved gas	Exp/linear	20	Analyst 4.01	N/A
VPM-B	Dual phase	Exp/exp	16	MultiDeco 4.12	Shearwater Perdix
Suunto-RGBM	Dual phase	Exp/delayed exp	9	DM5 V1.2.47	Zoop-Novo

of breathing gas or, in cold water, hypothermia. Since the ultimate objective was to reduce P_{DCS} in divers, this study included higher-risk dive profiles, where the product of the **p**ressure (in bar) and the square **r**oot of the bottom **t**ime (minutes) or PRT was greater than 25.[5]

Four different decompression algorithms were tested (Table 1). These algorithms were chosen for evaluation owing to the availability of implementation in desktop computer decompression planning software. More details of these algorithms are available in the literature; some algorithms are better documented in the open literature than others.[6–8] Some details on EMC-20H came directly from Cochran Undersea Technology (Corso J, personal communication, 2016). For purposes of this study, the different algorithms were tested as 'black boxes' with known inputs and outputs, using desktop versions of the algorithms.

The four algorithms tested were all deterministic in nature, based on either gas loading or bubble formation in multiple tissue-type compartments. In this study, the four algorithms were validated against predictions made by probabilistic models derived from man-trial P_{DCS} data and by using the data directly. Since we know from the previous comparative studies that these different algorithms will produce markedly different results, the basic differences between the various approaches were looked at first. Two of the algorithms (ZHL-16C and EMC-20H) were in the category of 'dissolved gas' or 'tissue loading algorithms' (Haldanean) and the other two (VPM-B and Suunto-RGBM) were dual-phase (bubble) algorithms. Also, within each of these two physiological-based paradigms, there are still major differences, not just the number of compartments, but more importantly the functional form of the tissue loading process.

The algorithms were evaluated as implemented in the following software packages: MultiDeco 4.12 (Bühlmann ZHL-16C and VPM-B), DM5V1.2.47 (Suunto-RGBM) and Analyst 4.01 (EMC-20H). All dive computers tested employed some form of conservatism factor, a

user-adjustable parameter that changed the computed decompression requirements. The ZHL-16C algorithm employed a two-dimensional conservatism factor, referred to as a 'gradient factor' (GF). GFs, ranging from 0% to 100%, were added to the ZHL-16C algorithm such that they modify the M-value equations in the Bühlmann model, and hence alter the prescribed decompression profile.[9] The lower gradient factor (GF-Lo) controls the depth of the first stop. The higher gradient factor (GF-Hi) affects total decompression time. More details on this topic can be found elsewhere.[9] Setting values of GF-Hi = GF-Lo = 100 results in the original ZHL-16C model. EMC-20H used a single-value conservatism factor, input as a percentage ranging from 0% (default) to 50%. Suunto-RGBM utilised a single-value conservatism factor which could be set to 0 (default), +1 or +2. The VPM-B implementation used in MultiDeco enabled a similar single-value conservatism factor, but with a range from 0 (default) to +5. All algorithms were initially evaluated with their default conservatism factors: GF-Lo = GF-Hi = 100% for ZHL-16C, 0% for EMC-20H, and 0 for VPM-B and Suunto-RGBM. If any algorithm failed a test then its conservatism factor(s) was/were adjusted iteratively to determine whether a suitable setting could be found to allow the algorithm to pass the test.

TOTAL DECOMPRESSION TIME

To assess these algorithms against the requirement of low P_{DCS}, we first re-visited methods and models designed to specifically assess the suitability of the US Navy's decompression schedule (used at that time). Data on single-level, non-repetitive, nitrogen-oxygen dives from the US Navy Decompression Database were fitted to a logistic regression that resulted in P_{DCS} isopleths as a function of bottom time and TDT.[10,11] As in the original paper, it was postulated that after depth and bottom time, TDT is a strong candidate for the most influential variable in modeling DCS (as compared to profile/stop-time combinations),[10] and has been corroborated by other studies.[12,13]

Figure 1

Reproduction of Figure 8D from reference 11. P_{DCS} isopleth contours from the StandAir model plotted with the data the model was derived from; depth is 150 fsw; see text for explanation of symbols
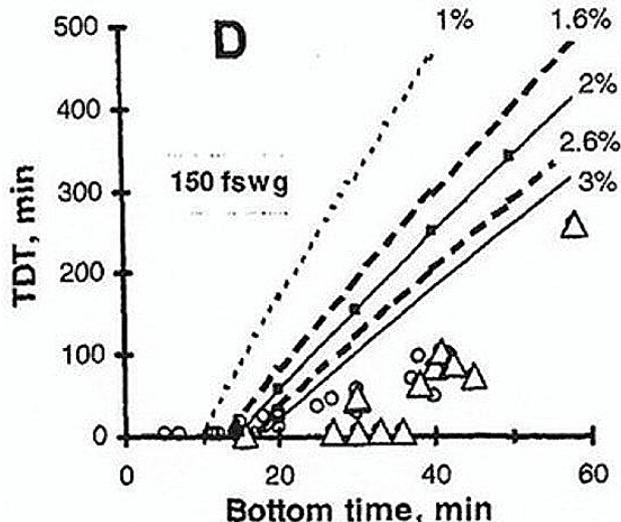
Figure 2

Reproduction of Figure 5 from reference 14. 0.2% $P_{(CNS)DCS}$ isopleth curve from Model2 plotted with data symbols the model was derived from.
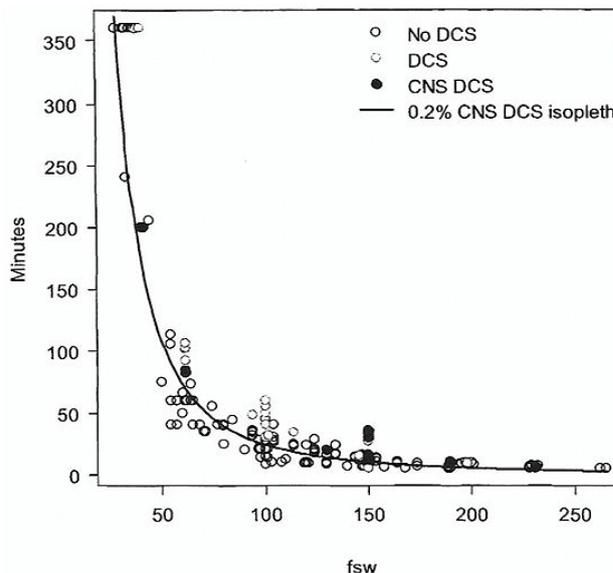
The domain of applicability of the current study was limited to 13 < PRT< 37, so the 'StandAir' model, which is based on data from standard air dives which had depths of less than 190 feet' sea water (fsw) and bottom times of less than 720 minutes (min), was utilised.[10] This model was considered to be reasonable except at the two depth extremes (nominally < 60 fsw and > 190 fsw). In the original study, the TDT required by the algorithm-under-test was compared to the P_{DCS} isopleths from the statistical model. It was found that the TDT required by the algorithm-under-test lay between the 2% and 3% P_{DCS} isopleths and thus the algorithm was deemed acceptable for US Navy use.[10] Figure 1(reproduced from Figure 8D from an additional reference[11]) shows graphically the relationship between the computed P_{DCS} isopleths from the StandAir model to the original data underlying the model.[11] The symbols show grouped data from Navy trials that resulted in DCS incidence at depths between 145 and 154 fsw and bottom times were rounded to the nearest 5 min. The figure/symbology is described as follows:

"*Triangles locate the bottom times and TDTs of particular dive trials that resulted in at least one case of DCS. Circles show trials that produced no DCS in any divers*."[11]

In the current study, we selected the 3% P_{DCS} isopleth as an initial standard of comparison as a compromise between managing DCS risk while not requiring excessive total decompression times.

NO-STOP TIMES

As a special/limiting case these algorithms were also evaluated for NST, i.e., for a given depth what is the maximum bottom time for the algorithm that allows a direct ascent?. For the NST limit test, extensive research performed by NEDU in 2009 was leveraged.[14]   In this research, the basic methodology used was to compare no-stop decompression data from many well-documented experimental man-trials and fit a logistic model of the $P_{(CNS)DCS}$ to the data.[14] The resultant model, 'Model2' was used to generate $P_{(CNS)DCS}$ isopleth curves. Figure 2 (reproduced from Figure 5 from reference 14) shows graphically the relationship between the computed 0.2% $P_{(CNS)DCS}$ isopleth to data symbols that represent dive profile summaries. The data covered a range of depths from 30 to 260 fsw. From Figure 2 we observed that a large fraction of the data is between 50 and 200 fsw and thus covers the domain of applicability of this study. The authors' chi-square goodness-of-fit analysis "*motivates retention of the hypothesis that Model2 provides a valid summary of results from all the dive trials across all depths*."[14]   This 0.2% $P_{(CNS)DCS}$ isopleth is used as the NST test in the present study. This special case serves to test the various algorithms at the lower range of PRT (~17 to 20) and thus is the most relevant for recreational divers. For both NST and TDT, statistical regression models, derived from man-trial data as a standard of comparison, were used rather than the individual data points themselves.

FIRST SIGNIFICANT STOP

While it has been shown that TDT was more important than how that total time was distributed amongst the stops, the second important variable was "*how deep the schedule starts*."[13]   To assess this, data were used from a NEDU controlled experiment which compared P_{DCS} outcomes of dives of the same depth, bottom time and same TDT but with different stop profiles.[15] This NEDU study was performed

Figure 3

Total decompression time (TDT) vs. bottom time; results for 120 fsw dives
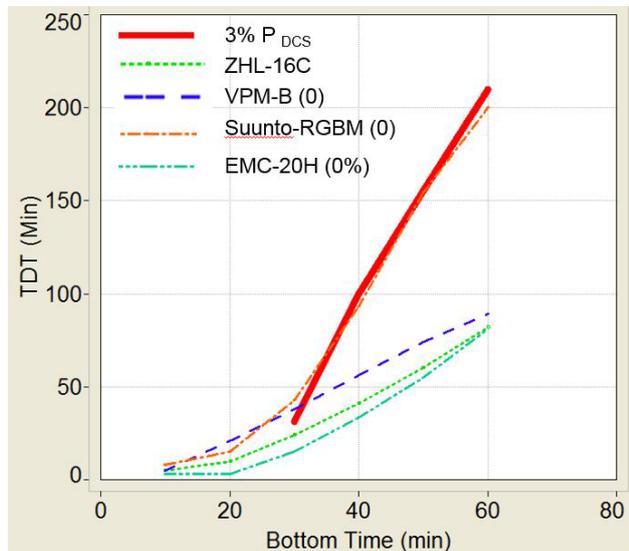
Figure 4

Total decompression time (TDT) vs. bottom time; results for 150 fsw dives
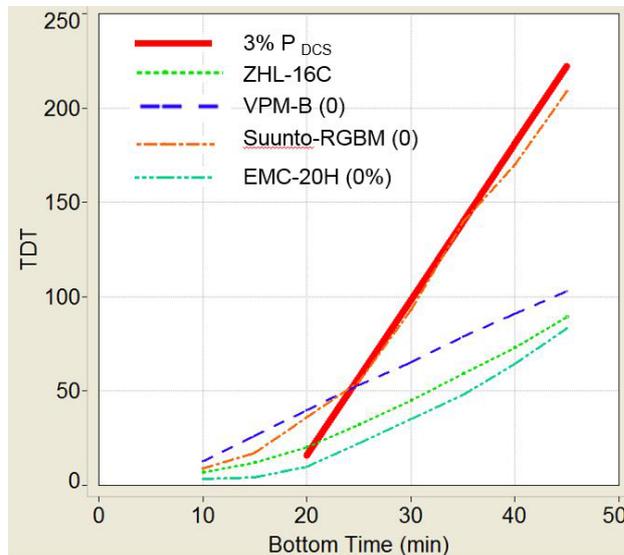
specifically to assess the effect of deep versus shallow profiles as dictated by dual-phase versus tissue-loading algorithms. In the NEDU study, the maximum depth was 170 fsw, the bottom time was 30 min, the ascent rate was 30 fsw·min⁻¹ and the TDT for both profiles was 180 min. The group of divers who started their first stop at 40 fsw (for 9 min) had significantly lower P_{DCS} ($P = 0.0489$ one-sided, Fisher Exact Test) than the group that stopped first at 70 fsw for 12 min. Since there is not sufficient information to know exactly where between the two tested depths might be optimal, this test case used the two depths as a maximum and minimum for the first stop criterion. In applying this test, care must be taken owing to the inter-relationship between the effects of ascent rate and first stop depth. So this last test is better called a first 'significant' stop depth test, where significant is tentatively defined as ≥ 1 min.

VERIFICATION

In the verification step, the objective was to assess whether the algorithms as implemented into the wrist computer hardware are a faithful representation of the full/baseline algorithm used in the desktop planning software. The specific wrist units under test are listed in Table 1. But not all wrist computers have a dive planning mode. If they do not, then the best approach would be to test the wrist unit in a hyperbaric chamber, reproducing a particular dive-pressure profile.[1] This type of testing was beyond the scope of the present research and verification will be limited to comparing desktop numerical results to wrist-unit implementation where the wrist computers have a dive planning mode. We compared numerical results (output) of TDT (or NST) between that given by the desktop planning version and the actual wrist unit, given the same inputs. While this was not an exhaustive/conclusive test, this criterion is necessary but

not sufficient for good verification, and, thus, it is a logical first step in the verification process.

Both ZHL-16C and VPM-B were easily tested by comparing the desktop version in MultiDeco to the wrist computer implementation of the Shearwater Perdix unit operated in dive planning mode. We verified Suunto-RGBM by comparison to the DM5 software. The EMC-20H algorithm could not be verified using this method because the wrist dive computer does not have a dive planning mode.[8]

Results

TOTAL DECOMPRESSION TIME

Total decompression times required by the various algorithms under test (at default conservatism levels) are plotted versus bottom time for the two depths (Figure 3 and Figure 4). The 3% P_{DCS} isopleth as predicted by the StandAir model of Reference 10 is included as a guideline. From Figures 3 and 4, it is reasonable to tentatively infer the following:

- Suunto-RGBM seems to prescribe sufficient TDT over a fairly wide range of bottom times at both depths;
- VPM-B appears to prescribe adequate TDT for bottom times up to 30 min at 120 fsw and 29 min at 150fsw (corresponding to PRT values of 25 and 30);
- ZHL-16C seems to prescribe insufficient TDT for bottom times of 28 min (120fsw) and 21 min (150 fsw) which equates to a PRT value of 25;
- EMC-20H seems to prescribe insufficient TDT for bottom times of greater than 26 min (120 fsw) and 19 min (150fsw), which roughly corresponds to a PRT value of 24.

Figure 5
No-stop times versus depth for the tested algorithms
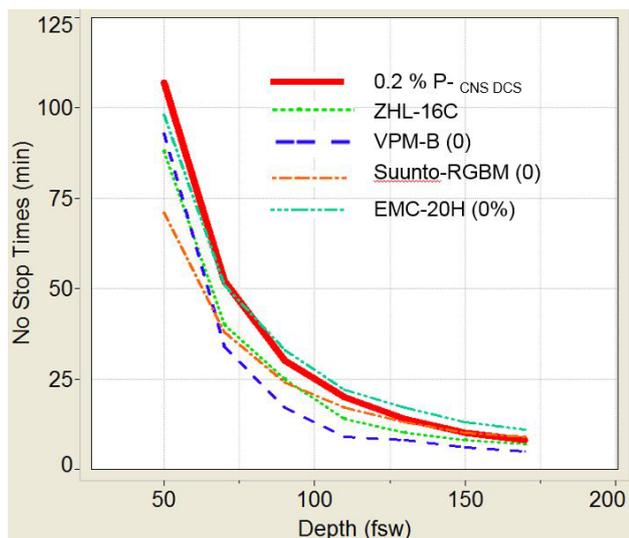
Through iterative adjustment of the conservatism settings, the three algorithms ZHL-16C, VPM-B and EMC-20H could be tuned to require adequate TDT, but for all three, the conservatism factor required depends on the PRT of the dive. Using the most conservative setting of 50%, EMC-20H could be made to provide sufficient TDT up to a PRT of about 30. At the high end of the domain of applicability in this study of 37, VPM-B could be made to provide sufficient TDT according to the requirements of the test as described here by setting the conservatism factor to +5. ZHL-16C should use a GF-Hi (which mainly affects TDT) < = 70.

NO-STOP TIMES

The results for NST are shown in Figure 5. From this figure, we can observe that both ZHL-16C GF-Hi=GF-Lo = 100 and VPM-B (conservatism = 0) specify sufficiently short NST over this fairly wide range of depths. EMC-20H seems to prescribe adequate NST for depths less than 80 fsw, but at greater depths, the NST may be too long. The Suunto-RGBM algorithm is more difficult to interpret for this metric, since it adds a 3 min/10 fsw 'safety stop' to all dives with a depth greater than 30 fsw.[16] Therefore, it was assumed the NST for Suunto-RGBM using the DM5 software to be the bottom time at which the prescribed 10 fsw stop time was increased from 3 to 4 minutes, or if an additional stop was added deeper than 10 fsw. Suunto-RGBM appears to prescribe sufficiently short NST at the shallower depths, then approaches the NEDU-suggested limit around 130 fsw. Suunto-RGBM may be too conservative over the lower range of depths. EMC-20H was adjusted to require adequately low NST with a conservatism factor of > 25%.

FIRST SIGNIFICANT STOP

Given that the conservatism factors of all of these algorithms have been modified to provide adequate TDT, the various profiles differ considerably with respect to the profile or depths of the various decompression stops. As described above, the four decompression algorithms were run to calculate the depth of the first significant stop using the depth-bottom time from reference 15.[15] VPM-B with the default conservatism of 0 calls for a first stop of 90 fsw, which is deeper than the 70 fsw depth that resulted in higher P_{DCS} in the trial. Increasing the conservatism factor did not help; the first significant stop increased to 100 fsw for values of four or greater. EMC-20H was first evaluated with a conservatism of 25% as suggested by the NST test. At this level, the first significant stop was 70 fsw. This first stop can be brought down to 60 fsw with a conservatism value of 5% or less. However, at this conservatism value, the algorithm does not prescribe sufficient TDT and NST.

For Suunto-RGBM in default mode of conservatism factor of 0 and using the 'deep-stop' option yields a first stop depth of 113 fsw, which is significantly deeper than both of the NEDU profiles. Turning off the deep-stop mode reduces the first stop depth to 59 fsw.

We first evaluated ZHL-16C using GF-Hi = GF-Lo = 70, since the TDT test suggested lowering GF-Hi to 70. With these GF values, the first significant stop was at 60 fsw, which is inside the desired range. Keeping GF-Hi at 70, we can lower GF-Lo down to 55 and still maintain a first significant stop depth of shallower than 70 fsw.

VERIFICATION

For the verification of ZHL-16C and VPM-B, the computed TDT for two depth-time combinations (120 fsw/50 min and 150 fsw/40 min) agreed within 4.5 to 6.8%, which is within the inherent uncertainty of the validation.

Verifying the Suunto-RGBM algorithm was more complex. While the desktop dive planning software DM5 enables the user to plan decompression dives with the Suunto-RGBM version of the algorithm, the low-end wrist computers that use this algorithm (in this study, Zoop-Novo) only enable the planning of non-decompression dives. As mentioned above, Suunto-RGBM adds a 10 fsw/3 min safety stop to all dives with a depth greater than 30 fsw. The manual states that this is an optional safety stop.[16] As before, NST time using the DM5 software was assumed to be the bottom time at which the 10 fsw stop time was increased from 3 to 4 minutes, or if an additional stop was added deeper than 10 fsw. Useful data were only obtained if the deep stop option in the DM5 planner was turned off; see Table 2 for a summary of the verification test performed on Suunto-RGBM.

OVERALL ASSESSMENT

The assessment results are summarised in Table 3. Based on an initial analysis, it can be inferred reasonably that none of the four algorithms evaluated passed all of the tests with default settings. ZHL-16C could be adjusted to pass all of the

Table 2
Verification results of the no-stop times using the Suunto-RGBM algorithm; fsw – feet sea water

Depth (fsw)	Zoop-Novo wrist unit (min)	DM5-deep stop on (min)	DM5- deep stop off (min)	Difference (%)
50	63	71	71	11
70	33	Added stop @ 35 fsw	38	13
90	19	Added deeper stop	24	21
110	11	Added deeper stop	17	35
130	7	Added deeper stop	13	46
150	5	Added deeper stop	10	50

Table 3
Summary of preliminary findings; Cons – conservatism setting; GF – gradient factor; NST – no stop time;
PRT – see text (p.251) for definition; TDT – total dive time

Algorithm	TDT	NST	Profile	Verification
ZHL-16C	Default OK for PRT ≤ ~25; suggest GF-Hi ≤ 70 for higher	OK with GF 100/100	Suggest GF-Lo ≥ 55	Good; agreement better than 10%
VPM-B	Default OK for PRT < 28; Above this may need to increase Cons. with PRT	OK	Cannot tune to address this	Good; agreement better than 10%
Suunto-RGBM	Default OK for wide range of PRT	OK	Suggest turn off deep stop option	Partial test; only able to compare NST; agreement 11−50%
EMC-20H	Default OK for PRT < 24; above this, may need to increase Cons. with PRT	Default not sufficient > 70 fsw; okay with Cons > 25%	Default OK, but marginal with Cons ≥ 5%	N/A; computer does not have plan-mode

tests with GF-Hi < = 70 and GF-Lo > = 55. Suunto-RGBM could be made to pass all of the tests by simply turning off the deep stop option, which is easily done on the wrist unit tested. VPM-B could be adjusted to prescribe sufficient TDT (the required conservatism factor depends on the depth-bottom time of the dive) but could not be adjusted to pass the first significant stop test; the first stop was always too deep. EMC-20H could be tuned to pass the TDT test (conservatism ~ 50%) and the first stop test (conservatism < 5%) independently, but not simultaneously.

Discussion

The domain of applicability of the current study was dives with PRT values in the range of 13 to 37, which covers no-stop dives (generally PRT < 20), and low-risk decompression dives (20 < PRT < 25), as well as some historically higher risk dives (25 < PRT < 37). One obvious limitation of this study is that we used US Navy test data and

there are significantly different risk factors between Navy dives and recreational dives, such as bottom work-load, potentially low water temperature and high currents. One complicating factor in this study was the lack of software configuration control in these algorithms. Variants exist and these different variants are not well identified or documented, which impedes the validation and verification process.[17,18] In the future, the concepts and procedures of model configuration management and verification should be more rigorously implemented into algorithms used for commercial off-the-shelf dive computers. The initial verification tests in this study only cover a few pairs of possible tests that should be performed.

This study presents how man-trial data with known P_{DCS} can be used by the non-Navy scientific community for testing of commercial decompression algorithms. This type of validation testing informs how to best use available commercial dive computers to improve diver safety. More

research on how to structure and improve these tests is needed, specifically on the first significant stop test. Lastly, for algorithms used in commercial dive computers where desktop dive planners are not available, similar testing as described here can be performed by simulating dives with wrist units in hyperbaric chambers.

Summary

Commercial off-the-shelf dive computer algorithms were evaluated by comparison with US Navy experimental dives with known decompression sickness outcomes and resultant statistical models. Four algorithms were evaluated: Bühlmann ZHL-16C, VPM-B, Suunto-RGBM and EMC-20C. This preliminary testing indicates that while none of the four passed all of these proposed tests with factory default settings, ZHL-16C and Suunto-RGBM could be made to pass by adjusting user-defined settings.

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Conflicts of interest: The author has no commercial interest or link with any dive computer manufacturer.

Case reports

A diver with immersion pulmonary oedema and prolonged respiratory symptoms

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Abstract

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Immersion pulmonary oedema (IPE) is particularly associated with an excessive reaction to exercise and/or cold stress. IPE usually resolves without recompression therapy within a day or two. Herein we report a diver diagnosed with IPE, in whom symptoms persisted for five days. A 58-year-old man presented with sudden onset of dyspnoea, cough and haemoptysis after surfacing. He was an experienced diving instructor with a history of moderate mitral valve regurgitation. While IPE was diagnosed and oxygen administered, respiratory symptoms deteriorated, and serum C-reactive protein elevated. No evidence of infection was seen. Three hyperbaric oxygen treatments were given on the basis of suspected decompression sickness, and symptoms subsequently resolved. The recently diagnosed mitral valve regurgitation and inflammatory response were considered to have contributed to the prolongation of symptoms.

Introduction

The concept of immersion pulmonary oedema (IPE) was established in recent years following a first report in 1981.[1] The condition is especially associated with an excessive reaction to exercise or cold stress.[2] IPE usually resolves quickly with rest and oxygen administration, according to previous reports.[2–4] We present a case of IPE in which respiratory symptoms lasted five days, and which improved after three hyperbaric oxygen treatments (HBOT), along with administration of antibiotics and diuretics. Such protraction of respiratory manifestations in a patient with IPE does not appear to have been reported previously. We consider the possible pathophysiological mechanisms underlying this unusual clinical situation.

Case report

The patient was a 58-year-old man with 20 years' experience as a scuba diving instructor. He had been diagnosed with moderate mitral valve regurgitation (MR) six months before the current event, but had not been prohibited from diving. Cough-variant asthma had once been suspected when he complained of chronic cough after an upper respiratory tract infection. He had a 20-pack-year history of smoking, but had quit 10 years earlier. He had no history of allergy and was not on any medications.

He went air scuba diving on an autumn morning using appropriately prepared equipment and wearing a wetsuit. He had been aware of mild dyspnoea and cough for two days before diving. He had felt substantial dyspnoea after swimming to a boat but this resolved after a short rest. The sea temperature was 20.4°C and the wave height was 2 m. His first dive of the day reached a maximum depth of 18.7 metres' sea water (msw) and lasted 56 minutes (min), including a three-minute safety stop at 5 msw. He felt the temperature was a bit cold and exercise intensity was light. He changed his ordinary air regulator to an oxygen regulator on the decompression stop in 5 msw, to demonstrate oxygen decompression for educational purposes. Just after restarting the ascent, he experienced intense shortness of breath that worsened on surfacing. Although he took off his wetsuit as it felt tight, symptoms did not resolve. He rested while inhaling pure oxygen but symptoms deteriorated and became accompanied by irresistible coughing and haemoptysis, and he was transported to a local hospital. He denied experiencing any marine animal stings or aspiration during

Figure 1

Chest X-ray on admission reveals bilateral lung base-dominant infiltrations

Figure 2

Chest CT on admission (2.5 h after onset) shows ground glass opacity in a central peribronchial-vascular distribution and smooth thickening of the interlobular septae in the lung bases bilaterally, compatible with pulmonary oedema Two days later (right, 35 h after onset), Chest CT findings worsened, revealing bilateral consolidation and pleural effusions

2.5 hours after onset   35 hours after onset

the dive, even when he exchanged his regulator.

On admission, heart rate was 138 beats per min, respiratory rate 24 breaths per min and oxygen saturation was 96% on oxygen at 6 L·min^{-1}. Vesicular breath sounds were diminished in both lung fields on auscultation. Cardiac examination revealed a systolic murmur (Levine 3/6) at the apex radiating to the axilla. No peripheral oedema was evident and jugular venous distention was not seen. Electrocardiography showed sinus tachycardia. Chest X-ray showed bilateral dependent oedema in the lower lung fields (Figure 1). Chest computed tomography (CT) revealed ground glass opacities with a central peribronchial-vascular distribution and smooth thickening of the interlobular septae in bilateral lung bases and apices (Figure 2). Laboratory studies showed a serum troponin I concentration of 0.020 ng·mL^{-1} and a brain natriuretic peptide (BNP) level of 150.8 pg·mL^{-1}. He was admitted to hospital to ensure rest and to administer oxygen, on suspicion of IPE. Ceftriaxone and azithromycin were administered from the day of admission because community-acquired pneumonia (CAP) was also being considered.

Hypoxia worsened, accompanied by productive cough during the night on the second hospital day. Transthoracic echocardiography (TTE) performed at this time revealed severe MR with tendon rupture, normal left ventricular wall motion and no evidence of right heart strain. Repeat chest CT revealed progression of pulmonary oedema and bilateral pleural effusions (Figure 2). Diuretics were administered and hypoxia improved slightly. However, fever increased to 39°C and laboratory studies revealed a strong inflammatory response (C-reactive protein (CRP) 20.7 mg·dL^{-1}). As the clinical course was atypical for IPE, heart failure and CAP,
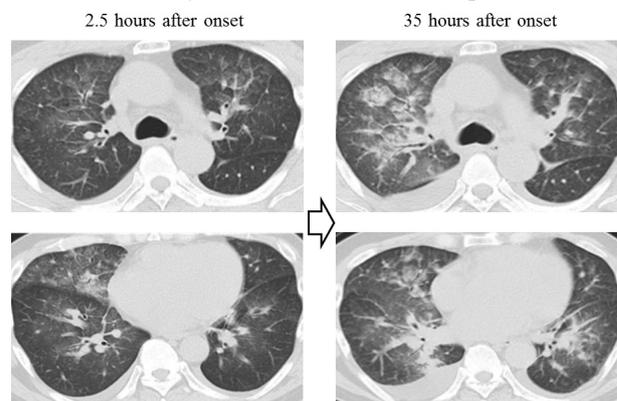
the patient was transported to our hospital on suspicion of decompression sickness (DCS).

On admission, heart rate was 114 beats per min, respiratory rate 25 breaths per min, blood pressure 113/68 mmHg, body temperature 38.4°C and oxygen saturation 95% on oxygen at 3 L·min^{-1}. Chest auscultation revealed fine crackles in the basilar regions bilaterally. No peripheral oedema or cyanosis were noted. Laboratory studies showed a CRP level of 22 mg·dL^{-1}, a BNP level of 134 pg·mL^{-1}, and otherwise normal results, including liver enzymes. Negative results were obtained for sputum and blood cultures, urine pneumococcal antigen, legionella antigen and pharyngeal mycoplasma antigen. Sputum cytology showed a markedly increased eosinophil count.

We suspected respiratory DCS and started recompression therapy according to United States Navy treatment table 6 (USN TT6).[5] Hypoxia, tachycardia, and tachypnea improved during the first recompression therapy, but response was incomplete. This first HBOT was extended. Two further USN TT6 resolved all symptoms, CT findings, fever and inflammatory response. Ceftriaxone was discontinued on day five as elevated liver enzymes were noted, ampicillin/sulbactam was administered instead. The patient was discharged without any further complications and subsequently underwent mitral valve replacement.

Discussion

We initially diagnosed IPE based on the typical presentation and radiological findings. However, despite appropriate initial therapy for IPE, symptoms deteriorated and we needed to consider other potential diagnoses. In addition to the use of antibiotics and diuretics, three HBOT were performed on the suspicion of DCS and symptoms had resolved by the completion of these.

The pathophysiological mechanism leading to IPE is elevation of lung capillary pressure, caused by a cold- or exertion-induced hypertensive response.[1,6] The pre-existing MR in our patient represents a known risk factor for pulmonary oedema. During immersion, the increased preload caused by the cold environment may have contributed to the development of pulmonary oedema.[2] On the other hand, shortness of breath just after swimming but before diving could have been an early manifestation of IPE, as in a previous report.[3]

The protraction of IPE into a five-day pulmonary disorder and pronounced inflammatory response were prominent features in the present case. Most reported cases of IPE resolved faster, without antibiotics or a need for recompression therapy.[3,4] The possibility of an infectious pathogenesis contributing to the high fever and inflammatory response could not be excluded in this case. On the other hand, the findings were sufficient to look for aetiologies other than infection because antibiotic administration appeared ineffective and no evidence of infectious pathogens was detected from microbiological examinations.

Another possible diagnosis was heart failure from reversible stress cardiomyopathy (Takotsubo cardiomyopathy), acute myocarditis or acute exacerbation of known MR.[8,9] However, there was little possibility of myocarditis or Takotsubo cardiomyopathy given the lack of myocardial strain findings on ECG, the normal wall motion seen on TTE on hospital day two in the previous institute and the negative results for serum troponin I.

Pre-existing MR could plausibly have contributed to this prolongation of respiratory symptoms. MR contributes to elevation of pulmonary artery pressure and subsequent pulmonary oedema. When his MR had deteriorated owing to tendon rupture was unclear, although the rupture had occurred at least since his first visit to the previous institute. According to his cardiologist, the pulmonary oedema could not have been owing only to MR because there was no evidence of right heart strain or hypotension.

This diver had the possibility of having respiratory DCS. Respiratory DCS has been described anecdotally as "*the chokes*". This is a rare presentation of DCS and case reports of respiratory DCS are limited.[10] Respiratory DCS has been considered to arise from the formation of a large quantity of gas bubbles and subsequent congestion of the pulmonary circulation.[5] The patient's diving log indicated a bottom time of 48 min and a maximum depth of 18.7 m. This suggests his safety stop was made within the no-decompression limit,[5] and the probability of profuse gas bubbles in pulmonary vessels seemed low. Consideration of this condition as respiratory DCS was thus difficult in the classical context, although one report has described a case thought to represent respiratory DCS with insufficient inert gas loading to form gas bubbles.[10] The clinical course of our patient, employing oxygen during decompression, post-dive, during transport

and three USN TT, also reduces the likelihood of DCS as an explanation.

In conclusion, pre-existing MR could have contributed to protraction of respiratory symptoms and the slow response to recompression therapy in this case. Simultaneously, IPE itself may sometimes be more protracted than we have previously assumed in such situations. The inflammatory response could have been evoked by oxygen, infection or lung overinflation.

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The on-site differential diagnosis of decompression sickness from endogenous cerebral ischaemia in an elderly Ama diver using ultrasound

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Abstract

(Yanagawa Y, Omori K, Takeuchi I, Jitsuiki K, Ohsaka H, Ishikawa K. The on-site differential diagnosis of decompression sickness from endogenous cerebral ischaemia in an elderly Ama diver using ultrasound. Diving and Hyperbaric Medicine. 2018 December 24;48(4):262–263. doi: 10 28920/dhm48.4.262-263. PMID: 30517960.)
Commercial or occupational breath-hold (BH) harvest divers along the coast and islands of Japan are collectively called Ama divers. Repetitive BH diving by Ama divers may place them at risk of developing neurological decompression sickness (DCS). We report a 74-year-old Ama diver who demonstrated right hemiparesis during an ascent after free diving at a depth of 5 metres' sea water. This report suggests the usefulness of on-site ultrasound for making a differential diagnosis of DCS from endogenous cerebral ischaemia. Further clinical studies of this management approach are warranted.

Introduction

It was previously believed that a diver must be exposed to a certain minimum depth before bubbles could form and that a diver could spend an unlimited amount of time at shallow depths (< 10 meters' sea water, msw).[1] However, it is now known that bubble formation can occur even after shallow dives and that it is inappropriate to exclude the diagnosis of decompression sickness (DCS) based on a perceived minimum depth.[1] Even when a diver follows the decompression tables, excess nitrogen remains in the blood for hours after a dive. Doppler studies have shown that up to 10% of remaining nitrogen may be released as bubbles after a normal dive.[2]

Commercial or occupational breath-hold (BH) harvest divers along the coast and islands of Japan are collectively known as Ama divers.[3] Repetitive BH diving can lead to the accumulation of nitrogen (N_2) in blood and tissues, which may give rise to DCS,[4] and previous studies have suggested that Ama divers are at risk of neurological DCS.[3–5] We report a patient with endogenous transient ischaemia who demonstrated right hemiparesis during her ascent after free diving to a depth of 5 metres' sea water (msw).

Case report

A 74-year-old Japanese female Ama diver was rescued by colleagues after she developed right hemiparesis during ascent while free diving to a depth of 5 msw. She had made multiple dives over three hours to similar depths collecting turban shells and abalone. The exact times of the dives could not be determined. She had known hypertension on medication. She was taken ashore, and an emergency call resulted in an ambulance being dispatched. A physician-staffed helicopter from Eastern Shizuoka was also requested, as divers with DCS are transported to a hospital located further away for recompression since there are no suitable hospitals on the Izu Peninsula.

When examined on site by a physician an hour post dive, she was asymptomatic. As portable ultrasound (Vscan®; General Electric, USA) using the longitudinal sub-xiphoid window did not reveal any bubbles in her inferior vena cava, she was diagnosed with endogenous cerebral ischaemia not induced by DCS or air embolism from barotrauma. She was transported to our hospital (which does not have recompression facilities) by helicopter whilst receiving intravenous Ringer's solution and on oxygen.

On arrival, she was fully conscious and orientated, with isocoric, reactive pupils. Neurological examination was unremarkable. Whole-body computed tomography (CT) revealed no gas in her body, and there were no blebs, small bullae or air trapping in the lungs. Magnetic resonance imaging (MRI) of her head two hours after the incident, including diffusion-weighted imaging and angiography, were negative. Biochemical analyses of blood revealed no abnormalities, including for D-dimer. Right-to-left shunt, including a persistent foramen ovale (PFO), was excluded on

ultrasound. A dagnosis of a transient endogenous ischaemic attack and hypertension was made, and she received anti-platelet therapy without recompression treatment. Her post-admission course was uneventful and she was discharged on hospital day five without sequelae.

Discussion

This report suggests the potential usefulness of on-site ultrasound for the differential diagnosis of a central neurological event. The different diagnosis of DCS, air embolism from pulmonary barotrauma or endogenous stroke was necessary in the present case. Free diving as performed by Japanese Ama is a rare cause of DCS but still one that should be considered.[3] The mechanism of stroke induced by DCS or air embolism is intravascular gas produced by abrupt decompression or barotrauma. Also there is a strong correlation between DCS and PFO, where venous bubbles can become arterialized via the right-to-left shunt and, thus, result in cerebral symptoms.[6] Repetitive deep breath-hold diving, long dive times and short surface intervals result in N_2 loading predisposing divers to DCS, which characteristically manifests as cerebral stroke.[3,7–9] This elderly Ama had no air bubbles on ultrasound at the scene or on subsequent CT, a negative D-dimer level and no right-to-left shunt, so the possibility of DCS was unlikely.

Even in asymptomatic divers, gas bubbles can be detected by ultrasound immediately after diving.[10] Loss of consciousness during diving may be from decompression illness (DCI), hypoxia induced by drowning, immersion pulmonary oedema or a cardiac event from immersion and exercise.[11] In such cases, physicians on board the Eastern Shizuoka helicopter routinely use ultrasound to make a differential diagnosis at the scene in order to decide to which hospital the diver will be transported, dependent on the availability of recompression facilities. However, the use of this device in making a differential diagnosis has not been validated in the literature. There may be criticism that the absence of bubbles in the inferior vena cava, even in the acute phase, cannot exclude the presence of bubbles in the whole body of a diver, so divers with focal neurological signs should always be transported to a hospital with a recompression chamber as a precaution.

Conclusion

This report suggests the usefulness of on-site ultrasound for the differential diagnosis of DCS from endogenous cerebral ischaemia in an elderly Ama diver. Further clinical experience with ultrasound for the acute differential diagnosis of divers with neurological symptoms and signs is merited.

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Conflicts of interest: nil

The world as it is

British Sub-Aqua Club (BSAC) diving incidents report 2017

Compiled by Brian Cumming and Jim Watson, Diving Safety and Incidents Advisors
Summary of the 2017 report prepared by Colin Wilson

BSAC has been reporting annually on diving incidents for over 25 years.[1,2] The quality of information collected has improved over these years. Although these data are mainly from reports by club members, other sources are also used. BSAC continues to try to identify common errors, directing changes in training. The reports have been summarised in this journal since 2006 with earlier reports detailing the data collecting methods.[3]

The 2017 report includes 205 incidents, mainly in the United Kingdom (UK), with a few overseas incidents, and continues the decline in the numbers of reports over the past decade. BSAC believes this decline is unlikely to be owing to a fall in reporting or to the non-inclusion of RNLI reports (see below). However, there were more ascent incidents, with 44 documented. The fewest incidents are seen in the northern hemisphere winter months and, as in 2016, without a springtime surge, suggesting better care by divers as they return to diving at the beginning of the summer season. Coastguard involvement was the same as 2016 but Search and Rescue (SAR) helicopter usage fell by a third. The 2017 data do not include the Royal National Lifeboat Institution (RNLI) whose involvement is mainly in support of divers with disabled boats and in search and recovery of divers.

Fatalities

There were 11 fatality reports, only two being of BSAC members (well below the previous 10-year BSAC average of six). The reports sometimes lack the quality and depth of information of other diving fatalities reports,[4] so there was often insufficient information to be clear about the exact chain of events and root causes, though an educated assessment was made. Multiple causes are often at play when disaster happens. Broadly the causes were similar to previous years:
- Ten divers were aged between 36 and 72 years, average 55 years (one unknown); older than in previous years.
- Seven cases involved loss of consciousness underwater.
- No confirmed cases of a non-diving-related medical incident (e.g., heart attack); however, medical factors could be implicated in six with insufficient information on another two. Immersion pulmonary oedema (IPE) might have been a contributory factor in five.
- Two cases involved divers diving in a threesome.
- One involved a rapid ascent.
- Six cases involved separation of some kind.
- One open-circuit diver was diving solo.
- One lone snorkeller;
- Two cases involved rebreather divers.

From the fatalities section:

Case 1

A group of six divers, four using rebreathers and two using open circuit with one on trimix, carried out a hardboat dive on a wreck. A maximum one hour runtime was agreed with the skipper and all buddy checks, gas and computer checks and prebreathes were carried out. The divers were split into two groups and a diver from the first group descended, tied the shot into the wreck, checked conditions and sent up a signal buoy as an 'OK' for the dive to go ahead. The first group carried out their dive and ascended completing their decompression with a run time of 50 min to a maximum depth of 45 msw [metres' sea water]. *The second group, three on rebreathers carrying two bailout cylinders of air and nitrox 50, and one on open circuit trimix entered the water but the trimix diver experienced a flooded suit so aborted the dive. The three rebreather divers continued the dive and descended but at some point one of the group became separated from the others and was the first to surface using the shotline and was recovered by the boat. Two DSMBs* [diver surface marker buoys] *arrived on the surface and it was assumed these were those of the two other divers. The skipper went to recover the shot but this was interrupted when only one diver surfaced with his DSMB. The diver reported that he and the third diver had deployed their DSMBs and begun their ascent after becoming separated from the first diver. During the ascent the third diver had let go of his DSMB and moved to grab the other diver's arm. The diver asked him if he was 'OK' and received an 'OK' in response although he did not seem it. The third diver then let go and slid off the diver's arm and sank down to the seabed where he was unresponsive, his loop was out of his mouth and he appeared deceased. The diver tried to lift the third diver but he remained negatively buoyant and the diver was unable to raise him to the surface. He tied him to the shotline and made the decision to go up for help. He ascended, completed decompression and surfaced in a distressed state shouting for help. The boat picked him up, the diver calmed down and with the facts established the Coastguard was contacted. One of the rebreather divers ... after around 30 minutes on the surface returned to the wreck to search for the missing diver who was no longer tied to the shotline. Although an extensive search was made the diver was not located and the search stood down. Eight weeks later police confirmed that they had recovered a body from the sea thought to be that of the missing diver.*

Case 2

A diver using nitrox 32 carried out a wreck dive from a boat with two buddies. The dive plan was to a maximum depth of 24 m with a bottom time of 35 min and a total run time of 45 min. The diver was using twin 12 litre cylinders and a side slung decompression cylinder with nitrox 80. The group ascended but were separated on the surface due to strong currents. The instructor and one student let go of the ascent line and inflated their DSMBs. The instructor tried to tell the diver (the casualty) to let go of the line, too. The dive boat picked up the instructor and one student. The instructor told the skipper to get back to the shot line where he expected to find the casualty. They arrived at the line and the instructor got back in the water where he found the casualty on the surface without mask and regulator, face down and unconscious. The casualty was brought onto the boat and CPR with oxygenated air attempted. This was between 10 to 15 min after the group surfaced. A helicopter was called and the winchman arrived on the vessel approximately 10 min after CPR was started. He took over and tried to revive the casualty. The skipper believed the diver had completed all required decompression stops. The diver was reported to have frantically attempted to remove his equipment on the final stage of his ascent, surfaced alone and lost consciousness. When the rescuer reached the diver his equipment just fell away leaving just the buoyancy of his drysuit. The diver was airlifted to hospital and subsequently pronounced dead.

Decompression illness (DCI)

There were 56 decompression incident reports with some involving more than one casualty. More than one causal factor was implicated in some. Since 2013, DCI incidents appear to have levelled off. Analyses of the incidents are similar to previous reports:

- within the limits of tables or computers – 23;
- repetitive diving – 23;
- depths greater than 30 msw – 25;
- rapid ascents – 11;
- missed decompression stops – 4.

This list is almost identical to previous years. A number of the "*diver injury/illness*" reports (37) were probably DCI. It is noted that there are more DCI cases arising from dives reported to be within decompression limits.

From the DCI section:

Case 3

A day had been organised for members of a dive club to undertake a hard hat try-dive in a pool and a hyperbaric chamber dive. The group were split into two with the first group carrying out the hard hat dive first followed by the chamber dive and the second group carrying out the chamber dive followed by the hard hat dive in a pool. One of the divers in the second group did the chamber dive to a maximum depth of 50 msw [equivalent] with a dive time of 33 min. The group had a lunch break and then [the diver] undertook the hard hat dive reaching a maximum depth of 4 msw with a dive time of 8 min. The diver returned home but later that evening she had a bad headache and was in constant pain so took pain killers. She stayed in bed until mid-day the following morning still with a bad headache and pain in her hips and knees so took more painkillers. The diver went to work but that evening she could not see properly, was disorientated and still had pain in her legs. She contacted a local hyperbaric chamber who told her to attend straight away and during the journey it was noted that she had become confused, still had the headache and pains in her legs, knees and right foot. The diver was assessed at the chamber and given recompression treatment. She was discharged and advised not to dive for four weeks.

Case 4

A diver, using a rebreather with air diluent, carried out a boat dive to a maximum depth of 30 m and a run time of 60 min. The diver reported a normal ascent with no missed decompression stops. Back aboard the diver started vomiting, with blood seen in the vomit. The boat broadcast a 'Pan Pan' requesting Coastguard assistance. The Coastguard arranged a connect call with a duty dive doctor who advised that the diver be evacuated by helicopter to a recompression chamber. The diver at this point began to experience headaches, chest pain and shortness of breath. The boat skipper upgraded the incident to a 'Mayday'. The update on the diver's condition was relayed to the doctor who advised that the symptoms were not consistent with a cardiac issue and he still wanted the diver to be taken to the chamber. The diver's symptoms progressed to a worsening of his chest pain, tightness in the top of his shoulders and loss of the ability to stand. The diver was still conscious and responsive and an update was given to the doctor. The helicopter arrived on the scene and evacuated the diver. The nearest hyperbaric chamber was at capacity so the diver was transferred to another chamber once they could offer a space. There had been problems arranging a chamber space due to most local facilities being full to the extent that a chamber on a nearby warship was considered and prepared. (Coastguard report).

Case 5

A diver had carried out four boat dives over a weekend, two [each day]. The first dive on the Sunday was to a maximum depth of 28 msw with a dive duration of 42 min including a 2 min stop at 17 msw and a 3 min stop at 6 msw. After a 2 hour surface interval and using nitrox 32 the second dive was to a maximum depth of 32 msw with a dive duration of 36 min including a 2 min stop at 17 msw and a 3 min stop at 6 msw. The stops at 17 m on both dives were non-mandatory

profile dependant intermediate stops (PDIS). Back aboard and after de-kitting and having a cup of coffee, the diver and his buddy sat in the wheelhouse. The diver commented to his buddy that he felt really tired and uncomfortable after the pasty he had eaten for lunch. Around 30 min after surfacing the diver decided to go out on deck as he was feeling a little sick but he could not stand up nor could he feel or move his legs. The skipper contacted the Coastguard and it was decided that when the boat returned to harbour it would be met by an ambulance to transfer the diver to a hyperbaric chamber. During the journey back and after 20 min on oxygen the diver was able to move his toes and 1 hour and 15 min later he was able to walk from the boat to the ambulance. At the chamber the diver was diagnosed with a suspected spinal DCI and underwent recompression treatment followed by further treatment the following day. He was asked to stay in the area for another twenty-four hours in case of a relapse but was discharged after further neurological tests. The diver was advised not to dive for three months and to have a diving medical before diving again.

Discussion

IPE in divers may be less rare than previously thought, being suspected in 15 reported incidents, of which five were fatalities. To increase awareness of this potentially fatal condition, advice is provided in the BSAC report on symptoms and initial management of this condition.

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Book review

Textbook of chronic wound care: an evidence-based approach for diagnosis and treatment

Jayesh B Shah, Paul J Sheffield, Caroline E Fife (editors)

A while ago, the Editor asked for someone to volunteer to review a newly released book on wound healing and wound care management. Never volunteer, I thought, the first moment I received the eBook – 936 pages long! The Editor answered calmly that perhaps I was lucky because there are many pictures in there. Well, there are, and in the end I am happy that I had the chance to get through this book as it was interesting and useful and in no way as painful to review as I first thought it might be.

The book was released in early 2018 and, therefore, is up-to-date. Going through the listed references of the chapters the latest citations are from 2016, which, with regard to the editing process, is very current for a textbook. The single chapters follow a red line in structure, always starting with a short explanation of the key concepts of the chapter, with the most important facts listed in a short, concise manner. After that, the contents of the chapters are listed with headings that help to find special fields of interest. At the end of each chapter is a self-test with usually five multiple choice questions on the contents of the chapter. The correct answers are provided.

There are 38 chapters, divided into eight thematic sections and an appendix with very useful and well-designed flow charts on the clinical decision-making pathways in chronic wound care. From a didactic perspective the sections and also each chapter are well constructed. Section One starts with fundamental knowledge on the anatomy, physiology and biochemistry of wound healing, followed by a section covering the essentials of wound healing. These two well-written and illustrated sections provide a good understanding of the problems encountered in caring for chronic wounds. Section Two, consisting of seven chapters, covers a large field including the aetiology of wound healing, the influence of nutrition, preparation of the wound bed, and current concepts of wound dressing – including closure and plastic surgery. Especially in this section there are many pictures and corresponding case reports illustrating the text and the specific problems behind impaired wound healing.

While Sections One and Two provide a general view on the principles and problems of wound healing, Sections Three and Four (with five and four chapters respectively) address special groups of patients with specific wound healing problems such as diabetic wounds, venous insufficiency ulcers, arthropod-related wounds and radiation effects or burns. Special populations, like paediatric patients and the elderly are covered in Section Five, and the various settings in wound care management (including hyperbaric oxygen therapy, HBOT) are covered in Section Six.

Up to the end of Section Six the book provides excellent information for anyone from medical student to nurse to physicians interested in or having to deal with patients with difficult wounds and impaired wound healing, with an up–to-date and practical approach and excellent illustrations. The authors of the chapters and, most of all, the editors have paid close attention to evidence-based data and, as far as currently known, reliable, verifiable facts.

Section Seven is more for those who have to manage wound care, as the section is entitled "*challenges and opportunities in wound care practice in the new era*". In this section, the question on how research can be better incorporated into wound care and HBOT is addressed, but telemedicine, electronic health recording and other management questions are also discussed.

In Section Eight the book closes with a view to the wound care in other countries such as Australia, Spain, India, Thailand, Malaysia, Korea and Africa. This part is interesting to read and nice to have but in my opinion the value of the book would not be less if this section were omitted. The appendix with the really excellent flow-charts was already mentioned before.

Diving and hyperbaric medicine are the central focuses of this journal and many readers are hyperbaric physicians. So, what about HBOT in the book? HBOT is mentioned several times but it is not the main topic, especially with respect to its physiological background and practical application. In the chapter on radiation wounds (Chapter 17) HBOT is described in four text pages; a rather short explanation of the mechanism of action with little information on the topics of how long, how often, which profile/treatment table and so on. With respect to the mechanisms of action only the effect on hypoxia in the wound is mentioned, but other findings, suggesting effects beyond just diminishing hypoxia such as signal transducing effects[1–4] are not mentioned at all.

In Chapter 24 the organizational challenges and problems of performing HBOT in a physician's office are addressed, not how to perform HBOT adequately. In Chapter 27 the challenges of research in this field are the focus, and in Chapter 31 the decision-making process that might lead

to the introduction of HBOT to a patient's management is discussed, but only briefly. Taken together, from a hyperbaric physician's perspective, HBOT is covered somewhat insufficiently. Nevertheless, as stated above, Sections One to Five in particular offer good, current information to many actors in the field of wound healing and this is not a book specifically about HBOT (there are others). Rather, it is targeted at the wider field of wound healing.

Altogether this book is good value to DHM readers who work in the field of hyperbaric oxygen therapy and treat patients with complicated wound healing, indeed, almost a 'must have' for their library. Those readers who are coming from the diving side and have nothing to do with the problems of wound healing will not need this book; nevertheless even these will find interesting aspects, especially in the first half of the book. Finally, I was glad that I volunteered and got the chance to read it.

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Reprints

Ictal mammalian dive response: a likely cause of sudden unexpected death in epilepsy

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Even though sudden unexpected death in epilepsy (SUDEP) takes the lives of thousands of otherwise healthy epilepsy patients every year, the physiopathology associated with this condition remains unexplained. This article explores important parallels, which exist between the clinical observations and pathological responses associated with SUDEP, and the pathological responses that can develop when a set of autonomic reflexes known as the mammalian dive response (MDR) is deployed. Mostly unknown to physicians, this evolutionarily conserved physiological response to prolonged apnea economizes oxygen for preferential use by the brain. However, the drastic cardiovascular adjustments required for its execution, which include severe bradycardia and the sequestration of a significant portion of the total blood volume inside the cardiopulmonary vasculature, can result in many of the same pathological responses associated with SUDEP. Thus, this article advances the hypothesis that prolonged apneic generalized tonic clonic seizures induce augmented forms of the MDR, which, in the most severe cases, cause SUDEP.

Key words
Sudden unexpected death in epilepsy; SUDEP; Mammalian dive response; MDR; Pulmonary edema; Diving bradycardia; Apnea; Demargination

Toxicopathological effects of the sunscreen UV filter, oxybenzone (benzophenone-3), on coral planulae and cultured primary cells and its environmental contamination in Hawaii and the U.S. Virgin Islands

Downs CA[1], Kramarsky-Winter E[2,3], Segal R[2], Fauth J[4], Knutson S[5], and 11 others

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Abstract

Benzophenone-3 (BP-3; oxybenzone) is an ingredient in sunscreen lotions and personal-care products that protects against the damaging effects of ultraviolet light. Oxybenzone is an emerging contaminant of concern in marine environments – produced by swimmers and municipal, residential, and boat/ship wastewater discharges. We examined the effects of oxybenzone on the larval form (planula) of the coral *Stylophora pistillata*, as well as its toxicity *in vitro* to coral cells from this and six other coral species. Oxybenzone is a photo-toxicant; adverse effects are exacerbated in the light. Whether in darkness or light, oxybenzone transformed planulae from a motile state to a deformed, sessile condition. Planulae exhibited an increasing rate of coral bleaching in response to increasing concentrations of oxybenzone. Oxybenzone is a genotoxicant to corals, exhibiting a positive relationship between DNA-AP lesions and increasing oxybenzone concentrations. Oxybenzone is a skeletal endocrine disruptor; it induced ossification of the planula, encasing the entire planula in its own skeleton. The LC50 of planulae exposed to oxybenzone in the light for an 8- and 24-h exposure was 3.1 mg/L and 139 lg/L, respectively. The LC50s for oxybenzone in darkness for the same time points were 16.8 mg/L and 779 lg/L. Deformity EC20 levels (24 h) of planulae exposed to oxybenzone were 6.5 lg/L in the light and 10 l g/L in darkness. Coral cell LC50s (4 h, in the light) for 7 different coral species ranges from 8 to 340 lg/L, whereas LC20s (4 h, in the light) for the same species ranges from 0.062 to 8 lg/L. Coral reef contamination of oxybenzone in the US Virgin Islands ranged from 75 lg/L to 1.4 mg/L, whereas Hawaiian sites were contaminated between 0.8 and 19.2 lg/L. Oxybenzone poses a hazard to coral reef conservation and threatens the resiliency of coral reefs to climate change.

DHM reviewers in 2018

There are over 180 reviewers now in our database, of whom the following 89 have provided reviews during 2018. Rarely (6%) do people decline a review invitation and they usually have good reason for doing so. Many reviews, even those critical of a paper, are most constructive – a tribute to your dedication. The Editorial Board (especially the Editor) would like to thank everyone for their support. As you can see, we try hard to spread the load (except for your aging Editor!). If I've missed someone, I apologise.

In only one instance has a reviewer's comments drawn an unpleasant response from an author. There is no place for such behaviour in medical or scientific endeavour. We are all in this together – to produce good clinical and applied science reports that expand our understanding of the diving and hyperbaric fields and improve patient care.

Reviewer	Count	Reviewer	Count	Reviewer	Count
Gavin Anthony	1	Karen Hasmiller	1	Monica Rocco	1
Costantino Ballestra	2	Malcolm Hobbs	1	Chris Sames	1
Neil Banham	2	Christoph Klingmann	1	Martin Sayer	1
Amir Beglou	1	Jacek Kot	3	Fiona Seddon	2
Mike Bennett	2	Pierre Lafere	1	Susannah Sherlock	1
Lesley Blogg	4	Tom Kertesz	2	Barbara Shykoff	1
Matthew Bruce	4	Christoph Klingmann	1	Arne Sieber	1
Peter Buzzacott	1	Pierre Lafere	1	David Smart	1
Jim Caruso	1	Karen Lam	1	Gary Smerdon	2
Danilo Cialoni	1	John Leach	1	Oana Tatucu-Babet	1
Susan Coleshaw	1	Peter Lindholm	1	Kay Tetzlaff	1
Mike Davis	17	John Lippmann	1	Steve Thom	2
David Doolette	2	Geoff Loveman	1	Frauke Tillmans	1
Douglas Ebersole	1	Pierre Louge	1	Mike Tipton	1
Chris Edge	3	Leigh Madden	1	Mark Turner	1
Carl Edmonds	2	Robert Marx	1	Greg van der Hulst	2
Caroline Fife	1	Heather Massey	2	Rob van Hulst	2
Arron Fisher	3	Moritz Meyer	2	Pieter van Ooij	2
Nic Flemming	1	Ian Millar	1	Charles van Wiik	1
Andrew Fock	2	Simon Mitchell	6	Jose Vega	1
Neil Hampson	1	Otto Mollvaer	1	Constantin von See	1
Karen Hasmiller	1	Richard Moon	2	Lin Weaver	1
Sam Harding	2	Peter Mueller	1	Jurg Wendling	1
Richard Harris	1	Claus-Martin Muth	1	Matthew White	1
Malcolm Hobbs	1	Heather Murphy-Lavoie	3	David Wilkinson	1
Ian Gawthrope	1	George Perdrizet	1	Matt Wilks	1
Peter Germonpré	1	Neil Pollock	2	Peter Wilmshurst	1
Steve Goble	1	Andrew Proctor	1	Colin Wilson	2
Francois Guerrero	1	Jan Risberg	1		

Notices and news

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

SPUMS President's message
David Smart

The past year has been one of the busiest on record for SPUMS, and certainly for me as President, with considerable progress being made in 2018.

Diving and Hyperbaric Medicine e-Journal (DHMJ)

Thanks to Mike Davis and Nicky Telles, the transition of DHMJ to an e-Journal was successfully completed. The task was more complex than first anticipated and was an eye opener for all concerned. The linkages between various databases and the third-party providers involved in e-publishing is staggering, and still a work in progress. The process has gone way past just the production of a PDF journal. A key milestone was recently achieved with full recognition on PubMed Central. The linkages that have been set up should enhance accessibility and the journal's Impact Factor. There remain a number of areas requiring attention that have surfaced since the journal went electronic, including joint publisher governance. Mike Bennett and John Lippmann have represented SPUMS on the Journal Governance Committee (JGC) with colleagues Phil Bryson and Karin Hasmiller, I offer my thanks for the work they have done. Both publishers must have greater buy-in to DHMJ Governance processes in the future. The work cannot just be left to JGC. The publishers of DHMJ are indeed lucky to have had Mike Davis at the helm as Editor for the last 17 years. He retires this year and passes on the reigns to Simon Mitchell. Congratulations and best wishes Simon. Mike has left the Journal in superb state for the handover. He richly deserves SPUMS life membership which was awarded in 2017. We wish him good health and happiness in retirement.

SPUMS Financial Management

After a protracted time, SPUMS has finally migrated its banking over to ANZ. The inaccessibility and inadequate services provided by St George really became manifest when we tried to close SPUMS accounts. The process took months longer than expected and left ExCom shaking our heads in disbelief. Thanks to Peter Smith, Sarah Lockley and Shirley Bowen for their persistence in this process.

SPUMS had a large financial loss in 2016, owing to multiple reasons: new website development; a later than usual ASM organisation due to cancelling Philippines as a venue; increased print journal costs and the previous years'

ASM just breaking even. This trend has been arrested; SPUMS posted a modest surplus for 2017. The e-Journal implementation was a necessary component of the improved position, but we must avoid complacency in our financial dealings. I offer my sincere thanks to Sarah Lockley and her husband Cal Johnson who have done a terrific job with SPUMS and journal financial processes in addition to implementing an electronic accounting package.

Annual Scientific Meetings

Last year's ASM in Bali was a great success. This year marked the second Tricontinental Meeting co-hosted between EUBS, SPUMS and SAUHMA. Congratulations to Jen Coleman, who represented SPUMS on the convening committee of TRICON2018, and a huge effort from Mike Bennett on the scientific committee. Thanks to the teams from all organisations involved in hosting the meeting, in particular to our South African Colleagues. Following TRICON2018, SPUMS will review our future ASMs, seeking input from membership in this regard. Cathy Meehan has been very active in setting up the next three years. In 2019, we are planning Honiara, Solomon Islands, in 2020 to Tutukaka, NZ and in 2021 (SPUMS 50th ASM), to Okinawa, Japan. In addition, background processes to support SPUMS ASMs have been maintained.

SPUMS Executive Committee

I offer my thanks to all committee members for their continuing voluntary involvement and time to contribute to running our organisation. There have been three changes of SPUMS Executive Committee Membership since last AGM. John Orton retired as ANZHMG Chair and has been replaced by Neil Banham. Sincere thanks to John and welcome to Neil. Tamara Ford ceased to be a member and Greg van der Hulst, New Zealand was co-opted in her place. Thanks to Douglas Falconer for his role as Secretary .

SPUMS recognised courses in diving and hyperbaric medicine (DHM) and the SPUMS Diploma DHM

The ANZHMG Introductory Course in DHM moved to Western Australia this year and was highly successful, with 27 attendees. Thanks to Ian Gawthrope and Neil Banham for taking over running the course. Thanks also to David Wilkinson for continuing to lead the SPUMS DipDHM programme, and also for his role in hosting the Royal Adelaide Hospital course in DHM. These are now

integrated with the Australian and New Zealand College of Anaesthetists (ANZCA) Diploma of Advance Diving and Hyperbaric Medicine (DipAdvDHM).

ANZCA DipAdvDHM

SPUMS members Bennett, Smart, Sherlock, and Hawkins have been heavily involved with setting up this programme from scratch, with terrific support from ANZCA. The first exams were held in June–July 2018, and SPUMS members were very active as examiners; Banham, Trytko, Gawthrope and Sherlock with Smart as Chair of exams. The first graduate from the program was Juan Carlos Ascencio-Lane from Tasmania. There is now an established postgraduate pathway for DHM in the Antipodes, via ANZCA.

SPUMS Website and Administration

Nicky Telles has continued her terrific work developing and maintaining the SPUMS website, with Joel Hissink representing ExCom as Webmaster. Steve Goble has continued his support of SPUMS as Administrator. Thanks Nicky, Joel and Steve.

SPUMS Membership

SPUMS membership remains stable at around 450. Increasing membership to over 500 is desirable for our long-term viability. We have set realistic membership subscription rates and we are keen to increase both full membership numbers as well as associates. Please encourage all doctors and people you know who are interested in diving, to join SPUMS.

Key words
Medical society; General interest

Venue: Solomon Kitano Mendana Hotel, Honiara, Solomon Islands

Keynote Speaker
A/Professor Nigel Stuart Jepson
Senior Staff Specialist and Director of the Cardiac Catheterization Laboratories of the Eastern Heart Clinic, Prince of Wales Public Hospital (POWH), Sydney, Australia

Scientific Convenors:
Clinical Professor David Smart and Professor Michael Bennett
Event Convener
Dr Catherine Meehan cmeehan@mcleodstmed.com.au

Follow this link to get to the event webpage and to register for the event. http://www.cvent.com/d/2bqnpp

Have an abstract or paper you would like to present? - Please email our Scientific Convener:
scientific.convener@spums.org.au

Check out the Facebook page: https://www.facebook.com/events/2112622009000126/

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 to authors' available on the SPUMS website www.spums.org.au or at 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, Auckand;
    Dr Denise Blake, Townsville.

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

Key words

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

ANZ Hyperbaric Medicine Group Introductory Course in Diving and Hyperbaric Medicine 2019

Dates: 18 February–01 March
Venue: Esplanade Hotel, Fremantle, Western Australia
Cost: AUD2,600 (inclusive of GST)

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

Contact for information:
Sue Conlon, Course Administrator
Phone: +61-(0)8-6152-5222
E-mail: fsh.hyperbaric@health.wa.gov.au

ANZCA Diploma of Advanced Diving and Hyperbaric Medicine

Juan C Ascencio-Lane, a Hobart emergency physician, is the first candidate to complete the ANZCA Diploma of Advanced Diving and Hyperbaric Medicine (ANZCA DipAdvDHM). The diploma, launched last year, is a post-specialisation qualification in Australia and New Zealand. Its award ncludes completion of DHM training requirements and a specialist qualification. The qualification is the only one of its kind in Australasia.

Figure
The ANZCA Court of Examiners with Dr Ascencio-Lane and ANZCA president, Rod Mitchell. From left: Michael Jones (Chair examinations), Rod Mitchell (ANZCA President), Juan Carlos Ascencio-Lane (candidate), David Smart (Chair DHM examination), Ian Gawthrope (examiner) and Neil Banham (examiner) photo originally published in the *ANZCA Bulletin* September 2018

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 2019
Written paper   26 June 2019
Viva voce   31 July 2019
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
Holders of the Certificate of DHM and highly experienced practitioners of DHM are eligible for recognition of prior experience towards the ANZCA Dip Adv DHM, as outlined in the guidelines for the transitional award of diploma in Regulation 36. Applications for credit must be made in writing to the ANZCA TA unit and must be submitted prior to 31 January 2019.

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

Suzy Szekely, Chairperson, ANZCA DHM SIG
Suzy.Szekely@health.sa.gov.au

SPUMS 49th Annual Scientific Meeting
Preliminary announcement
April 2020
Tutukaka, Northland, New Zealand

Guest Speaker: Richard Harris, Adelaide

Convenor: Greg van der Hulst

The **Diving and Hyperbaric Medicine Journal**
website is at

www.dhmjournal.com

The latest issues, embargoed for one year, are available for the personal use of society members only. Access is via your SPUMS or EUBS website log-in and password. Please respect that these are restricted access and to distribute their contents within one year of publication is a breach of copyright.

Older issues (from March 2007 to September 2017); articles for immediate release into the public domain; contents lists and the Abstracts of the most recent (embargoed) issues; information about submitting to the Journal; profiles of the Editorial Board and useful links are to be found on the site. This will be expanded progressively as resources allow.

Your membership ensures the continued publication of DHM – thank you for your support of SPUMS and EUBS.

Five-year Impact Factor report for *Diving and Hyperbaric Medicine*
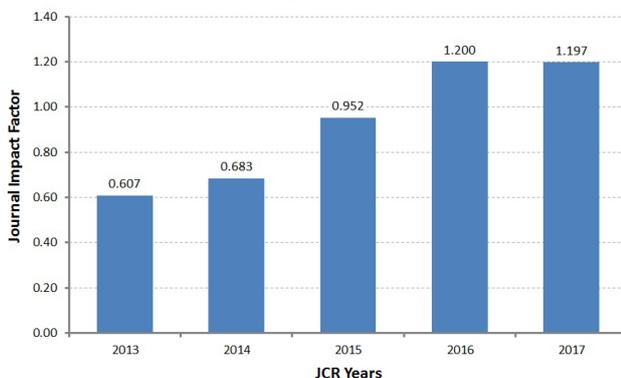
The *Diving and Hyperbaric Medicine* Journal is now on Facebook

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The

SPUMS

website is at
www.spums.org.au

Members are encouraged to log in and keep their personal details up to date.
The latest issues of *Diving and Hyperbaric Medicine* are via your society website login.

Obituary

Albert Hamish Turnbull, MB, BS
17 May 1940–15 July 2018

Hamish was born in Launceston Tasmania, 17 May 1940. His medical training was at the University of Queensland, qualifying in 1966. Although his early medical years were spent in London, he migrated back to Australia in 1978, to set up private practice in Victoria on the Mornington Peninsula, in a small town called Somerville, where he built a very loyal patient following right up until his recent death.

Hamish taught himself to dive as a young boy and regaled many with stories of his beloved Coles Bay. He continued to enjoy his diving until his last SPUMS meeting in Palau in 2015, having attended consistently since his first SPUMS ASM in 1991 in Port Douglas, North Queensland. He made his mark on the meetings and had a dedicated group of diving buddies who always enjoyed his company on and off the dive boat. Hamish was an eloquent and knowledgeable man with a sharp wit. He had a big personality and an equally big heart. He will be missed by us all – Hamish was certainly one of SPUMS's 'characters'!

SPUMS Facebook page

Remember to 'like' us at:
http://www.facebook.com/pages/SPUMS-South-Pacific-Underwater-Medicine-Society/221855494509119

From 01 January 2019 DAN Asia-Pacific will no longer be offering membership, insurance and training out of Australia. This business had been purchased by DAN World, which is an affiliate of DAN America. Due to its larger resources, DAN World will be offering enhanced membership benefits and a greater variety of dive and travel-related insurances to divers in the Asia-Pacific Region. A broad suite of DAN training programmes (non-accredited) will also be offered to local divers. We at DAN AP believe that this has the potential to increase and improved DAN services to divers throughout the Region and urge divers to continue to support DAN.

The funds from this change, together with the existing assets of DAN AP have been transferred to a new entity, the DAN Asia-Pacific Foundation Ltd, which is trading as the Australasian Diving Safety Foundation (ADSF). This new entity, based in Melbourne, is a registered Australian Health Promotion Charity, which means all donations to it are fully tax-deductible. John Lippmann is the Chairman and CEO of this charity and its primary object is to reduce human suffering from diving-related disease and injury. To this end, the ADSF is offering research and safety promotion grants.

Funds have been allocated to support up to AUD100,000 per year for appropriate research projects. In addition, the ADSF plans to offer grants of up to AUD10,000 to fund safety-related projects within the diving industry. Full details of these grants, as well as application forms are available at http://adsf.org.au.

John Lippmann, OAM, has been awarded his Doctorate of Philosophy (Health & Social Development) by Deakin University, Melbourne, Australia. His thesis is entitled:

"*Analysis of scuba diving-related fatalities in Australia*".

John is well known for his publications on diving and first aid and his work for many years as the Founder and Director of Divers Alert Network Asia Pacific. On the basis of his publications record and strong support for the Society, he was elected to full membership of SPUMS some years ago – a recognition bestowed on only a handful of non-medically qualified members. John represents the Society on the Journal Governance Committee.

SPUMS congratulates John on his achievement and the Editor wishes to add his personal appreciation of his contributions to the *Diving and Hyperbaric Medicine* journal as a frequent contributor and as a valued reviewer over many years.

Divers Emergency Service/ADSF Telemedicine Scholarship 2019

This scholarship will provide the successful DHM trainee with AUD4,000 to support their attendance at the next SPUMS ASM.

Criteria
The successful applicant will:
- be a medical practitioner at registrar or fellow level;
- have undertaken a formal attachment to a Diving and Hyperbaric Medicine facility in Australia for a minimum of three-months duration in the year prior to the date of the award;
- use the scholarship within 12-months (i.e., attend the next SPUMS ASM);
- be a registered trainee for the SPUMS Diploma (or completed);
- give a talk at the SPUMS ASM on a topic of their choosing if possible (not mandatory).

Applications should incude:
- a cover letter stating they are applying for the scholarship and including contact details and whether they intend to present at the ASM;
- a current CV;
- a letter of support from their supervisor or Head of Unit confirming their attachment to the unit and their ability to attend the SPUMS ASM;
- a letter confirming they are registered for, or have completed, the SPUMS Diploma.

and be forwarded in writing to
david.wilkinson@sa.gov.au

Applications close 01 March 2019

The successful candidate will be announced mid-March.

Erratum

In relation to the Thai cave rescue, at the end of his President's message in the September 2018 issue, David Smart wrote "*when and if the time is right, all of us in SPUMS are keen to hear Richard's story of the rescue. It is our privilege to have him as a member of SPUMS.*"

He wishes to correct this to read: "*When the time is right at a future ASM, all of us in SPUMS are keen to hear more detail of the rescue. It is our privilege that both Richard and Craig are members of SPUMS.*"

David Smart
President, SPUMS

EUBS Notices and news

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

EUBS President's message
Ole Hyldegaard

Dear members, associates and sponsors of the EUBS, it is with great pleasure and respect that I address you as the new President of the EUBS.

My predecessor Jacek Kot will, as Immediate Past President, continue in the EUBS Excom and I would like to take this opportunity to thank Jacek for his outstanding work for our organization and his long-time commitment to the world of hyperbaric medicine – both in diving and hyperbaric healthcare. With the Durban TRICON2018 meeting fresh in the memory, this year is coming to an end while writing this. On behalf of the EUBS, its members and affiliates, I wish to thank the organizers of TRICON2018 for all their efforts in creating a very successful meeting and all of you who came to Durban and participated in this event.

Diving and Hyperbaric Medicine (DHM) journal

DHM will change its Editor at end of this year, when Simon Mitchell takes over from Mike Davis who was appointed in 2002. DHM has undergone significant improvements in overall quality, visibility and impact in recent years. The journal's peer-reviewed scientific reports from the March 2017 issue onwards are now available on PubMed Central for viewing once they have passed their one year embargo or have been on immediate release. This is a major achievement that will increase the visibility and potential impact of the scientific work published in our journal as well as to promote the societies of EUBS and SPUMS. Mike Davis has been a key contributor to this important work. Personally and on behalf of the EUBS, I wish to express my outmost respect and tribute to the work that Mike has contributed over many years. We wish him all the best for the future and we greatly appreciate the assistance that Mike will give our incoming Editor during the transition period. Whilst appreciating the importance of publishing research in diving and hyperbaric medicine, DHM is also an important tool to promote our societies and to increase the support we need from our members. I call upon all EUBS members who may be able to influence their local, national societies and other organizations involved with diving and hyperbaric medicine to promote DHM, either by displaying adverts on local society websites or circulating the front cover and contents pdf' file of each issue to your members regionally. You will find these files on the EUBS or DHM websites for your free distribution.

EUBS elections

In 2018, we also had elections for the EUBS ExCom. Some good candidates with high quality backgrounds in both research and organizational work in diving and hyperbaric medicine from several regions of Europe and the USA came forward. It is with great pleasure that we congratulate and welcome Jean-Eric Blatteau as new EUBS Vice-President and François Guerrero as the new Member-at-Large. Also, thanks to Virginie Papadopoulou and John S Peter for participating and making their expertise and work available for the EUBS ExCom and elections.

Although our fields of interest and sub-specialisation may seem narrow, the work and specialities of the members of EUBS cover many different areas of the medical society. These range from extreme environmental physiology and human performance in the underwater environment, professional diving operations in off-shore industries and recreational diving in its broadest sense, to the treatment of patients with acute life-threatening or chronic conditions and rehabilitation with improvements of long-term quality of life in cancer patients, wound care and neurorehabilitation. Our challenges are many and it is my intension and goal as EUBS President to represent all these important fields in our organization.

Key words
Medical society; General interest

EUBS news and notices
Peter Germonpre, EUBS Secretary

EUBS Annual General Meeting

The EUBS AGM was held in Durban, South Africa, on Saturday 29 Septmber 2018 from 08.30 to 10.00, prior to the last scientific session and the Closing Ceremony of the 44th EUBS Annual Scientific Meeting – TRICON 2018. There were 69 EUBS members present. The AGM presentation and financial information has been placed on the EUBS website, in the members area section.

EUBS Executive Committee

Ole Hyldegaard (Copenhagen, Denmark) has assumed the position of president of the EUBS, taking over from Jacek Kot, and a new vice-president has been elected by the EUBS Membership: Jean-Eric Blatteau from Toulon, France. Also, to replace Karin Hasmiller after serving a three-year term, François Guerrero from Brest, France, has been elected as a new member-at-large 2018. The Executive Committee wish to express their gratitude for Jacek's and Karin's contributions to the ExCom activities. However, they will both remain active in the ExCom, Jacek as Immediate Past President and Karin as member of the Diving and Hyperbaric Medicine Journal Governance Committee (JGC) and the Research & Education Committee.

TRICON 2018 Meeting

The 2nd Tricontinental Scientific Meeting on Diving and Hyperbaric Medicine was attended by 196 delegates from EUBS, SPUMS, SAUHMA and UHMS, 37 delegates who are not a member of any of these Societies – yet), and 27 spouses. The scientific program, the diving workshops and the social events were greatly appreciated and EUBS would like to thank the Organising and Scientific Committee, as well as our local Congress Organiser Londocor (Ms Leigh Du Plessis and her team), for an outstanding job well done.

CPD certificates were e-mailed out to all participants who had their badges scanned at the start of the morning and afternoon sessions. If you did not receive one, please check your SPAM e-mail folder or search for a message sent on 05 October 2018 by certificates@med-bay.com. If you still do not find the certificate, please contact John Burk at Med-Bay (john@med-bay.com) to request a duplicate. As these CPD credits were awarded by the South Africa HPCSA (Health Professions Council of South Africa), you may have to submit a request for CPD at your own professional association, using this certificate as proof.

EUBS Annual Scientific Meeting 2019

The next EUBS Annual Scientific Meeting will be held in Tel Aviv, Israel, 09–12 September 2019. You can find all the information regarding the conference on the website www.eubs2019.com, or by visiting the EUBS website.

The conference will be jointly organised with the International Conference on Hyperbaric Oxygen and the Brain. The conferences will be hosted by the Israeli Society for Hyperbaric and Diving Medicine.

This period has been chosen as it is after the summer break but just before the high holidays in Israel. Tel Aviv is an exciting hypermodern coastal city, with top-notch medical research and treatment facilities, but also bustling with beaches, restaurants and nightlife.

Please bookmark the dates and register early, as a favourable airfare is dependent on early booking! Why not take advantage of the earlybird registration rates for EUBS 2019? These end on 31 December 2018!

EUBS 2018 Annual Scientific Meeting Awards

At the EUBS Annual General Assembly on 29 September 2018, the Zetterström Committee, composed of Martin Sayer, Michael Bennett and Costantino Balestra, have awarded the Arne Zetterström Award for best poster presentation to: Poster PD-12. "*Hypoxia is not reliably prevented by setting a 60 second apnea limit during exercise: the failure of the "one minute rule" for free diving*". This work was performed by Charlotte Sadler, Kaighley Brett, Aaron Heerboth, Austin Swisher, Nader Mehregani, Ross Touriel, Daniel Cannon, University of California, San Diego and San Diego State University.

Arne Zetterström (1917–1945) is best known for his research with the breathing mixture hydrox for the Swedish Navy. Zetterström first described the use of hydrogen as a breathing gas in 1943. From 1943 to 1944, a total of six ocean dives were made utilising this mixture with the deepest to 160 meters (96% hydrogen and 4% oxygen). On 07 August 1945 Zetterström experienced technical problems diving from HSwMS Belos. His support divers misread his signals and this was followed by a rapid ascent that resulted in severe decompression sickness and hypoxia, resulting in his untimely death.

The Patrick Musimu Award for best contribution, either oral or poster presentation, in the area of breath hold diving, was instituted in 2011 by the Belgian Society for Diving and Hyperbaric Medicine. Patrick Musimu (1970–2011) was a Belgian freediver, sport business manager, marketing and event manager, and physiotherapist. He was born in Kinshasa, Zaire. On 30 June 2005, he beat the previous "No Limits" world record in freediving by almost 40 meters by diving to 209 meters. At his request, this dive was done without the supervision of the International Association for Freediving Agency, from which Musimu dissociated since 2002. According to him, extreme deep freediving should not be considered as a sport but as an adventure. Musimu began freediving in 1999 at the age of 28. His secret lay in years of training and preparation, but special attention should be given to his ear clearing technique: instead of equalizing his ears by the regular manoeuvres, he flooded his air spaces (sinus and middle ears) with seawater before reaching the depth where ordinary equalization would become hard. On 21 July 2011 Musimu died while pool training alone at his home in Brussels, Belgium.

In Durban, the jury decided not to award this prize. It is felt that more research should be conducted in this field, and

the jury, speaking for the Belgian Society for Diving and Hyperbaric Medicine, would like to encourage this further.

EUBS Website

Please visit the EUBS website for the latest news and updates. Specifically, a new "*EUBS History*" section has been added under the menu item "*The Society*". There is still some information missing in the list of EUBS meetings, presidents and members-at-large – please dig into your memories and help us complete this list! By popular demand, EUBS members can now also download the complete abstract book of previous EUBS meetings from the members area.

OXYNET database to be updated

Since 2004, a public online database of European hyperbaric chambers and centres has been available. This was started and initially maintained by the OXYNET Working Group of the COST B14 project of the European Commission, and later maintained by the European Committee for Hyperbaric Medicine (ECHM). The database can be accessed on www. oxynet.org. However, over the past few years, the list and contact information of the OXYNET database has not been maintained regularly, and EUBS ExCom has proposed to take over this task and not only update the information but also to modernize the database and its functionality.

In order to do this, we can use all the help we can get. Please visit the OXYNET site and verify the information that is listed for your own hyperbaric centre. Then, rather than using the online form to correct the information, send an e-mail to oxynet@eubs.org with the updated information. If you could collect information for more than one centre in your area or country, please do. Once the OXYNET database has been relocated and restructured, a direct link will be placed also on the EUBS website, however, we will maintain the address www.oxynet.org as well!

Association Internationale des Centres Hyperbares Francophones (ICHF)

The ICHF is an initiative of EUBS members Rodrigue Pignel (Geneva, Switzerland) and Mathieu Coulange (Marseilles, France) with Thierry Joffre (Lyon, France), comprising a two-monthly video conference meeting with French-language hyperbaric physicians, chamber operators and nurses and has seen an increasing "attendance" for two years. The two to three-hour teleconferences are held on a streaming web platform that allows not only slide presentations, but also video and documents to be shared among all participants. The exchange of information and discussions about case reports, clinical experience (successes and failures), projects and research opportunities is considered a significant educational asset, and more centres

are being added to the ICHF membership. This project shows that using modern technology, all it takes is some creative thinking to increase professional interaction and sharing of knowledge. EUBS applauds this initiative and would like to encourage other similar projects.

The

website is at
<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.

Hyperbaric oxygen lectures

Welcome to: http://www.hyperbaricoxygen.se/ This site offers publications and high-quality lectures from leading investigators in hyperbaric medicine. Please register to obtain a password via email. Once registered, watch online, or download to your iPhone, smart device or computer for later viewing.
For information contact: folke.lind@gmail.se

The Science of Diving

Support EUBS by buying the PHYPODE book "*The science of diving*". Written for anyone with an interest in the latest research in diving physiology and pathology. The royalties from this book are being donated to the EUBS.
Available from: Morebooks https://www.morebooks. de/store/gb/book/the-science-of-diving/isbn/978-3-659-66233-1

DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA

P O Box 347, Dingley Village Victoria, 3172, Australia
E-mail: hdsaustraliapacific@ hotmail.com.au
Website: www.classicdiver.org

Scott Haldane Foundation

As an institute dedicated to education in diving medicine, the Scott Haldane Foundation has organized more than 250 courses all over the world over the past 22 years, increasingly targeting an international audience with courses worldwide

The courses Medical Examiner of Diver (part I and II) 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).

SHF Course Calendar 2019

February: Refresher course, "*Organization diving medical*", The Netherlands
29-30 March: Medical Examiner of Divers part 1, Zeist , NL
4, 5 and 6 April: Medical Examiner of Divers part 2, AUMC, Amsterdam, NL
May: Medical Examiner of Divers part 2, Caribbean
June: Dangerous Marine creatures (level 2d), NL
9-16 November: Medical Examiner of Divers part 1, Nosy be, Madagascar
16-23 November: 27th SHF In-depth course diving medicine (2d): Nosy be, Madagascar
23-30 November: 27th SHF In-depth course diving medicine (2d): Nosy be, Madagascar

On request: Internship different types of diving (DMP), NL
On request: Internship HBOT (DMP certification), NL/Belgium
The course calendar will be supplemented regularly.

For the latest information: www.scotthaldane.org

Copyright

Advertising in *Diving and Hyperbaric Medicine*

The Capita Selecta Diving Medicine annually offers symposia on diving medicine presented by speakers of national and international renown to a multinational audience of diving physicians, paramedics and highly educated diving instructors. The level of the presented material is advanced, i.e., Level 1 and 2d, and often beyond that. The lectures are in English.

30 March 2019: Diving medicine of women, children and divers with a disability

Speakers include: Selina Haas, AT, Ulrike Preiml, AT and Guy Vandenhoven (BE)

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

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 onto the SPUMS website, click on "*Resources*" then on "*GTÜeM 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.

German Society for Diving and Hyperbaric Medicine (GTÜeM)

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

20th International Congress on Hyperbaric Medicine 2020

Dates: 13–16 September 2020
Venue: Rio de Janeiro, Brazil
For preliminary information contact:
Dr Mariza D'Agostino Dias
Email: mariza@hiperbarico.com.br

Diving and Hyperbaric Medicine: Instructions for Authors

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, members of the diving and hyperbaric industries, and divers. Manuscripts must be offered exclusively to *Diving and Hyperbaric Medicine*, unless clearly authenticated copyright exemption accompanies the manuscript. All manuscripts will be subject to peer review. Accepted contributions will also be subject to editing.

Address: The Editor, Diving and Hyperbaric Medicine, P O Box 35, Tai Tapu, Canterbury 7645, New Zealand
Email: editor@dhmjournal.com
Phone: +64-(0)3-329-6857
Mobile: +64-(0)27-433-2218
European Editor: euroeditor@dhmjournal.com
Editorial Assistant: editorialassist@dhmjournal.com
Information: info@dhmjournal.com

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

Types of articles

DHM welcomes contributions of the following types:

Original articles, Technical reports and Case series: up to 3,000 words is preferred, and no more than 30 references (excluded from word count). Longer articles will be considered. These articles should be subdivided into the following sections: an **Abstract** (subdivided into Introduction, Methods, Results and Conclusions) of no more than 250 words (excluded from word count), **Introduction, Methods, Results, Discussion, Conclusions, References,**

Acknowledgements, Funding sources and any Conflicts of interest. Legends / captions for illustrations, figures and tables should be placed at the end of the text file.

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

Case reports, Short communications, Work in progress reports, etc: 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 and historical articles, Commentaries, Consensus and other meeting reports, etc., 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.

Formatting of manuscripts

All submissions must comply with the requirements set out in the full instructions on the DHM website. Non-compliant manuscripts will be suspended whilst the authors correct their submission. Guidance on the general structure for the different types of articles is given above.

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

- Instructions for authors
- DHM Key words 2018
- DHM Mandatory Submission Form 2018
- Trial design analysis and presentation
- EASE participation and conflict of interest statement
- English as a second language
- Guideline to authorship in DHM 2015
- Helsinki Declaration revised 2013
- Is ethics approval needed?

DIVER EMERGENCY SERVICES PHONE NUMBERS

AUSTRALIA
1800-088200 (in Australia, toll-free)
+61-8-8212-9242 (International)

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

ASIA
+81-3-3812-4999 (Japan)

EUROPE
+39-6-4211-8685 (24-hour hotline)

UNITED KINGDOM
+44-7740-251-635

SOUTHERN AFRICA
0800-020111 (in South Africa, toll-free)
+27-828-106010 (International, call collect)

USA
+1-919-684-9111

The DES numbers (except UK) are generously supported by DAN

DAN ASIA-PACIFIC DIVE ACCIDENT REPORTING PROJECT

This project is an ongoing investigation seeking to document all types and severities of diving-related incidents. All information is treated confidentially with regard to identifying details when utilised in reports on fatal and non-fatal cases. Such reports may be used by interested parties to increase diving safety through better awareness of critical factors. Information may be sent (in confidence unless otherwise agreed) to:

DAN Research
Divers Alert Network Asia Pacific
PO Box 384, Ashburton VIC 3147, Australia
Enquiries to e-mail: research@danasiapacific.org

DAN Asia-Pacific NON-FATAL DIVING INCIDENTS REPORTING (NFDIR)

NFDIR is an ongoing study of diving incidents. An incident is any error or occurrence which could, or did, reduce the safety margin for a diver on a particular dive. Please report anonymously any incident occurring in your dive party. Most incidents cause no harm but reporting them will give valuable information about which incidents are common and which tend to lead to diver injury. Using this information to alter diver behaviour will make diving safer.

The NFDIR reporting form can be accessed on line at the DAN AP website:
www.danasiapacific.org/main/accident/nfdir.php

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Opinions expressed in this publication are given in good faith and in all cases represent the views of the authors and are not necessarily representative of the policies or views of the SPUMS, EUBS or the Editor and Editorial Board.